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THE

DISEASES OF INFANCY AND CHILDHOOD

FOR THE USE OF STUDENTS
AND PRACTITIONERS OF MEDICINE

BY

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WITH TWO HUNDRED AND FORTY-ONE ILLUSTRATIONS
INCLUDING EIGHT COLOURED PLATES

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TO

VIRGIL P. GIBNEY, M. D., LL. D.,

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THIS VOLUME IS INSCRIBED

AS A TRIBUTE TO HIS PERSONAL WORTH AND HIGH PROFESSIONAL ATTAINMENTS,

AND IN GRATEFUL REMEMBRANCE OF MANY ACTS OF KINDNESS,

BY THE AUTHOR.

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PREFACE TO THE FOURTH EDITION.

IN no part of Pædiatrics are new knowledge and experience changing our views more rapidly than in matters concerning nutrition.

It has therefore become necessary to make another general revision of this section of the book. These pages have been largely rewritten and considerable new material introduced. It is hoped that in both simplicity and clearness the chapters upon infant feeding have been improved, and their practical value for the student and practitioner thereby enhanced.

The other sections have been changed but slightly from the Third Edition.

The author desires to acknowledge the assistance rendered in this revision by his associate, Dr. John Howland.

14 WEST FIFTY-FIFTH STREET,
NEW YORK.

PREFACE TO THE THIRD EDITION.

FREQUENT revisions of a text-book in pædiatrics are necessary if it would adequately present to its readers existing knowledge in this department of medicine. The present revision has been made without any important changes in the general arrangement, and at the same time without materially increasing the size of the volume. Certain chapters have been much abridged while others have been much expanded.

The needs of the student and practitioner rather than those of the specialist have been constantly kept in mind. The purpose has been to restrict the book to its own field—now so constantly widening—omitting the discussion of subjects which are fully treated in works upon pathology, general medicine, and surgery.

Convinced of the great value of good pictures, both of clinical and pathological conditions, especial pains have been taken in this revision with the illustrations. Many old ones have been replaced by better ones, and altogether twenty-five new illustrations have been introduced. For much assistance with these, the author desires to express his obligation to his associate, Dr. H. B. Wilcox, by whom many of the original photographs were taken. Credit is due to Dr. F. C. Wood for the color drawing of the blood.

While alterations have been found necessary in almost every chapter, the principal changes have been made in the articles upon the following subjects: Examination of the Sick Child, Hypertrophic Stenosis of the Pylorus, Diarrhœal Diseases and Dysentery, Vaginitis, Cerebro-Spinal Meningitis, Mental Defects, Chondro-Dystrophy, Status Lymphaticus, and Diphtheria. Most of these chapters have been entirely rewritten; some appear for the first time in this edition.

14 West Fifty-fifth Street,
New York.

TABLE OF CONTENTS.

PART I.

CHAPTER PAGE

I.—HYGIENE AND GENERAL CARE OF INFANTS AND YOUNG CHILDREN . . 1

Care of the newly-born child; bathing; clothing; care of the eyes; care of the mouth and teeth; care of the skin; care of the genital organs; vaccination; training to proper control of rectum and bladder; general hygiene of the nervous system; sleep; exercise; airing; the nursery; the nurse; the amount of air space required by infants; the care of premature and delicate infants; incubators; the feeding of the premature infant.

II.—GROWTH AND DEVELOPMENT OF THE BODY 15

Weight; height; growth of extremities as compared with the trunk; the head; the chest; the abdomen; muscular development; development of special senses; speech; dentition.

III.—PECULIARITIES OF DISEASE IN CHILDREN 30

Etiology; symptomatology and diagnosis; pathology; prognosis and infant mortality; prophylaxis; therapeutics.

PART II.

SECTION I.—DISEASES OF THE NEWLY-BORN.

I.—ASPHYXIA 69

II.—CONGENITAL ATELECTASIS 74

III.—ICTERUS 77

IV.—THE ACUTE INFECTIOUS DISEASES OF THE NEWLY-BORN 81

The acute pyogenic diseases; ophthalmia; tetanus; epidemic hæmoglobinuria; fatty degeneration of the newly-born; pemphigus.

V.—HÆMORRHAGES 95

Traumatic or accidental hæmorrhages; spontaneous hæmorrhages.

VI.—BIRTH PARALYSES 107

Cerebral paralysis; facial paralysis; paralysis of the upper extremity.

VII.—TUMOURS OF THE UMBILICUS, ETC. 113

Umbilical hernia; mastitis; intestinal obstruction; diaphragmatic hernia; sclerema; œdema; inanition fever.

SECTION II.—NUTRITION.

I.—INTRODUCTORY 124

The food constituents and the purposes they subserve in nutrition.

CHAPTER PAGE

II.—THE INFANT'S DIETARY 129
Woman's milk; cow's milk; condensed milk; kumyss; matzoon; junket, curds and whey; beef preparations; cereals; infant foods.

III.—INFANT FEEDING 168
Choice of methods; breast feeding; maternal nursing; wet-nursing; weaning; mixed feeding; artificial feeding.

IV.—FEEDING AFTER THE FIRST YEAR 219
Healthy infants during the second year; difficult cases during the second year; feeding from the third to the sixth year; feeding during acute illness.

V.—THE DERANGEMENTS OF NUTRITION 226
Acute inanition; malnutrition; marasmus.

VI.—DISEASES DUE TO FAULTY NUTRITION 244
Scorbutus; rickets.

SECTION III.—DISEASES OF THE DIGESTIVE SYSTEM.

I.—DISEASES OF THE LIPS, TONGUE, AND MOUTH 274
Malformations; diseases of the lips; diseases of the tongue; alveolar abscess; difficult dentition; catarrhal stomatitis; herpetic stomatitis; ulcerative stomatitis; thrush; gonorrhœal stomatitis; syphilitic stomatitis; diphtheritic stomatitis; gangrenous stomatitis.

II.—DISEASES OF THE PHARYNX 293
Acute pharyngitis; uvulitis; elongated uvula; retro-pharyngeal abscess; adenoid vegetations of the vault of the pharynx.

III.—DISEASES OF THE TONSILS 307
Croupous tonsillitis; ulcero-membranous tonsillitis; follicular tonsillitis; phlegmonous tonsillitis; chronic hypertrophy of the tonsils.

IV.—DISEASES OF THE ŒSOPHAGUS 314
Malformations; acute œsophagitis; retro-œsophageal abscess.

V.—DISEASES OF THE STOMACH 318
Digestion in infancy; malformations and malpositions of the stomach; hypertrophic stenosis of the pylorus; vomiting; cyclic vomiting; gastralgia; acute gastric indigestion; acute gastritis; gastro-duodenitis; chronic gastric indigestion; dilatation of the stomach; ulcer of the stomach; tumours of the stomach; hæmorrhage from the stomach.

VI.—DISEASES OF THE INTESTINES 352
Malformations and malpositions; diarrhœa; acute intestinal indigestion.

VII.—DISEASES OF THE INTESTINES (*continued*) 364
Acute gastro-enteric intoxication; cholera infantum.

VIII.—DISEASES OF THE INTESTINES (*continued*) 385
Acute colitis and ileo-colitis; chronic ileo-colitis; amœbic colitis; amyloid degeneration of the intestines; tuberculosis of the intestines and mesenteric lymph nodes.

IX.—DISEASES OF THE INTESTINES (*continued*) 413
Chronic intestinal indigestion; intestinal colic; chronic constipation; intussusception.

X.—DISEASES OF THE INTESTINES (*continued*) 438
Appendicitis; intestinal worms.

XI.—DISEASES OF THE RECTUM 452
Prolapsus ani; fissure of the anus; proctitis; ischio-rectal abscess; hæmorrhoids; incontinence of fæces.

CHAPTER PAGE

XII.—DISEASES OF THE LIVER 458
Icterus; functional disorders; new growths; acute yellow atrophy; congestion of the liver; abscess of the liver; cirrhosis; amyloid degeneration; fatty liver; hydatids; biliary calculi.

XIII.—DISEASES OF THE PERITONÆUM 465
Acute peritonitis; chronic (non-tuberculous) peritonitis; tuberculous peritonitis; ascites; subphrenic abscess.

SECTION IV.—DISEASES OF THE RESPIRATORY SYSTEM.

I.—NASAL CAVITIES 478
Acute nasal catarrh; chronic nasal catarrh; chronic rhinitis; membranous rhinitis; epistaxis.

II.—DISEASES OF THE LARYNX 489
Catarrhal spasm of the larynx; acute catarrhal laryngitis; membranous laryngitis; intubation; submucous laryngitis; chronic laryngitis; new growths; foreign bodies in the larynx.

III.—DISEASES OF THE LUNGS 509
The peculiarities of the lungs in infancy and early childhood; acute catarrhal bronchitis; fibrinous bronchitis; chronic bronchitis; reflex cough; asthma.

IV.—DISEASES OF THE LUNGS (*continued*) 527
Pneumonia; acute broncho-pneumonia.

V.—DISEASES OF THE LUNGS (*continued*) 562
Lobar pneumonia; pleuro-pneumonia; hypostatic pneumonia; chronic broncho-pneumonia; abscess of the lung; gangrene of the lung; acquired atelectasis; emphysema.

VI.—PLEURISY 591
Dry pleurisy; pleurisy with serous effusion; empyema.

SECTION V.—DISEASES OF THE CIRCULATORY SYSTEM.

I.—PECULIARITIES OF THE HEART AND CIRCULATION IN EARLY LIFE . . 606

II.—CONGENITAL ANOMALIES OF THE HEART 610

III.—PERICARDITIS 617
Acute pericarditis; chronic pericarditis with adhesions.

IV.—ENDOCARDITIS AND VALVULAR DISEASE 622
Acute simple endocarditis; malignant endocarditis; chronic valvular disease; myocarditis; anæmic murmurs; functional disorders of the heart; diseases of the blood-vessels.

SECTION VI.—DISEASES OF THE URO-GENITAL SYSTEM.

I.—THE URINE IN INFANCY AND CHILDHOOD 642
Functional or cyclic albuminuria; hæmaturia; hæmoglobinuria; glycosuria; pyuria; lithuria; indicanuria; acetonuria—diacetonuria; anuria; diabetes insipidus.

II.—DISEASES OF THE KIDNEYS 654
Malformations and malpositions; uric-acid infarctions; acute congestion of the kidney; chronic congestion of the kidney; acute degeneration of the kidneys; acute diffuse nephritis; chronic nephritis; tuberculosis of the kidney; malignant tumours of the kidney; pyelitis—pyelo-cystitis; renal calculi; traumatic hydronephrosis; perinephritis; general œdema not dependent on renal disease.

CHAPTER PAGE
III.—DISEASES OF THE GENITAL ORGANS 683
Malformations; diseases of the male genitals; diseases of the female genitals.
IV.—ENURESIS 692
Vesical spasm; vesical calculi.

SECTION VII.—DISEASES OF THE NERVOUS SYSTEM.

I.—INTRODUCTORY 699
II.—GENERAL AND FUNCTIONAL NERVOUS DISEASES 701
Convulsions; epilepsy; tetany; laryngismus stridulus; chorea; other spasmodic affections; hysteria; headaches; disorders of speech; disorders of sleep; injurious habits of infancy and childhood.
III.—DISEASES OF THE BRAIN AND MENINGES 747
Malformations; pachymeningitis; acute meningitis; cerebro-spinal meningitis; simple acute meningitis; tuberculous meningitis; chronic basilar meningitis in infants; thrombosis of the sinuses of the dura mater; cerebral abscess; cerebral tumour; hydrocephalus; infantile cerebral paralysis; mental defects; chondro-dystrophy; sporadic cretinism; insanity; the stigmata of degeneration; deaf-mutism.
IV.—DISEASES OF THE SPINAL CORD 820
Malformations; spinal meningitis; myelitis; compression-myelitis; acute poliomyelitis; tumours of the spinal cord; syringo-myelia; Friedreich's ataxia; Landry's paralysis; the muscular atrophies.
V.—DISEASES OF THE PERIPHERAL NERVES 846
Multiple neuritis; diphtheritic paralysis; facial paralysis.

SECTION VIII.—DISEASES OF THE BLOOD, LYMPH NODES, BONES, ETC.

I.—DISEASES OF THE BLOOD 856
Leucocytosis; simple anæmia; chlorosis; pseudo-leukæmic anæmia of infancy; pernicious anæmia; leukæmia; hæmophilia; purpura.
II.—DISEASES OF THE LYMPH NODES 877
Status lymphaticus; simple acute adenitis; simple chronic adenitis; syphilitic adenitis; tuberculous adenitis; Hodgkin's disease.
III.—DISEASES OF THE SPLEEN 896
IV.—DISEASES OF THE BONES AND JOINTS 899
Acute arthritis of infants; tuberculous diseases of the bones and joints; syphilitic diseases of bone.
V.—DISEASES OF THE SKIN 922
Congenital ichthyosis; milliaria; seborrhœa; eczema; furunculosis; gangrenous dermatitis; impetigo contagiosa; urticaria; scabies; tinea tonsurans.
VI.—ACUTE OTITIS 943

SECTION IX.—THE SPECIFIC INFECTIOUS DISEASES.

I.—SCARLET FEVER 953
II.—MEASLES 977
III.—RUBELLA 993
IV.—VARICELLA 996
V.—VACCINIA—VACCINATION 998
VI.—PERTUSSIS 1004

CHAPTER PAGE
VII.—Mumps 1016
VIII.—Diphtheria and Pseudo-Diphtheria 1019
IX.—Typhoid Fever 1062
X.—Tuberculosis 1070
XI.—Syphilis 1106
XII.—Influenza 1123
XIII.—Malaria 1131

Section X.—Other General Diseases.

I.—Rheumatism 1141
II.—Diabetes Mellitus 1147

LIST OF ILLUSTRATIONS.

PLATES.

FACING PAGE
I. Chart showing by months the mortality of New York city for the different ages for three years 43
II. Meningeal hæmorrhage in the newly born 108
III. Chart showing composition of various infant foods compared with woman's milk 165
IV. Bone in rickets 255
V. Typical rickets 258
VI. Deformity of the chest in severe rickets 261
VII. The stomach at the different periods of infancy 319
VIII. Extensive superficial ulceration of the colon 387
IX. Deep follicular ulcers of the colon 388
X. Membranous inflammation of the ileum 392
XI. Acute broncho-pneumonia 534
XII. Acute pleuro-pneumonia 580
XIII. Chronic broncho-pneumonia 583
XIV. Acute meningitis, complicating pleuro-pneumonia 768
XV. The blood in leukæmia and pernicious anæmia, etc. 857
XVI. Eruption of measles 981
XVII. The pathognomonic sign of measles (Koplik's spots) 989
XVIII. The diphtheritic membrane 1031
XIX. Diphtheria bacilli and their associates 1041
XX. Tuberculosis of the tracheo-bronchial lymph nodes 1082

ILLUSTRATIONS IN THE TEXT.

FIGURE PAGE
1. Incubator 12
2. Breck's feeding tube 13
3. 4. Scales 15
5. Weight curve for the first twenty days 16
6. Weight curve for the first year 18
7. Skull, showing premature ossification 23
8. Apparatus for albolene spray 57
9. Nasal syringe 58
10. Position for nasal syringing 59
11. Croup kettle 60
12. Vapourizer 61
13. Steam atomizer 61

FIGURE PAGE
14. Oiled-silk jacket 61
15. Apparatus for stomach-washing 62
16. Position for stomach-washing 63
17. Kemp's tube 65
18. Colon of a child six months old 66
19. Ribemont's tube 73
20. Pemphigus neonatorum 95
21. Double cephalhæmatoma, infant seven days old 98
22. Erb's paralysis 112
23. Umbilical tumours 114
24. Temperature chart in inanition fever 122
25. Human milk: A, colostrum period; B, later period 130
26. Apparatus for examination of human milk 135
27. A, Babcock tubes; B, Lewi's modification for human milk 136
28. Feser's lactoscope 148
29. Arnold sterilizer 155
30. Freeman Pasteurizer 156
31. Weight curve of nursing and artificial feeding compared 169
32. Weight curve showing effect of bad nursing and good feeding 177
33. Chart showing effect of pregnancy on weight of nursing infant 179
34. Weight curve of infant properly weaned 180
35. Percentage of fat in different layers of milk 192
36. Chapin's dipper for removing upper layers of milk 193
37. Weight curve of bottle-fed infant for first six months 197
38. Weight curve of artificially fed infant, showing effect of beginning with too high percentages 198
39. Weight chart showing the effect of intelligent care 207
40. Weight curve showing the advantage of temporarily stopping milk 216
41. Case of marasmus 240
42. Normal bone 256
43. Rachitic bone 257
44. Rachitic skull, inside view 260
45. Rachitic head 261
46. Rachitic skull, external view 262
47. Rachitic thorax in outline 262
48. Rachitic bow-legs 263
49. Rachitic knock-knees 264
50. Rachitic deformity of legs 265
51. Rachitic bow-legs in outline 272
52. Epithelial desquamation of the tongue 277
53. Thrush 287
54. Cancrum oris 292
55. Adenoid vegetations, natural size 300
56. Chest deformity from adenoid vegetations of the pharynx 302
57, 58. Child with adenoid vegetations, before and after operation 306
59. Dilatation of the stomach 348
60. Malformations of the rectum 352
61. Chart showing mortality from diarrhœal diseases in New York 355
62. Chart showing frequency of diarrhœal diseases 355
63. Weight curve showing effect of acute gastro-enteric intoxication during first year 369

FIGURE PAGE
64. Temperature chart of acute intestinal intoxication with fatal re-infection . 371
65. Acute catarrhal ileo-colitis, superficial type 387
66. Acute catarrhal ileo-colitis, severe form 388
67. Follicular ulceration of the colon, early stage. 390
68. Follicular ulceration of the colon, later stage 391
69. Membranous colitis 393
70. Weight curve showing loss from ileo-colitis 395
71. Temperature chart in ileo-colitis 397
72. Temperature chart in membranous colitis 399
73. Temperature chart in membranous colitis, long case 400
74. Chronic catarrhal inflammation of the ileum 405
75. Chronic intestinal indigestion 417
76. Ileo-cæcal intussusception 429
77. Mechanism of intussusception 430
78. Tænia saginata 446
79. Tænia solium 446
80. Tænia cucumerina 447
81. Bothriocephalus latus 447
82. Ascaris lumbricoides 448
83. Oxyuris vermicularis 450
84. Prolapsus ani 453
85. O'Dwyer's intubation set 499
86. An air vesicle in broncho-pneumonia 528
87. An air vesicle in lobar pneumonia 529
88. Broncho-pneumonia with thickened bronchus. 534
89. Broncho pneumonia, hæmorrhagic form 536
90. Broncho-pneumonia with emphysema 537
91. Broncho-pneumonia, diffuse purulent infiltration 538
92. Persistent broncho-pneumonia. 540
93. Temperature chart in mild uncomplicated broncho-pneumonia 545
94. Temperature chart, prolonged course, broncho-pneumonia 546
95. Temperature chart, relapsing broncho-pneumonia 546
96. Temperature chart, rapidly fatal broncho-pneumonia 546
97–100. Physical signs in broncho-pneumonia 548
101. Temperature chart, persistent broncho-pneumonia 551
102. Temperature chart, broncho-pneumonia following pertussis 552
103. Temperature chart, typical lobar pneumonia 568
104. Temperature chart, remittent type, lobar pneumonia 568
105. Temperature chart, lobar pneumonia, subnormal temperature after crisis . 569
106. Temperature chart, abortive pneumonia 569
107–109. Physical signs, lobar pneumonia 573
110. Section of lung, showing distribution of fluid in chest 598
111, 112. Empyema following pneumonia 599
113. Deformity after old empyema 604
114. Apparatus for inducing lung expansion after empyema 605
115. Showing normal areas of cardiac dulness 609
116. Congenital cardiac disease 611
117. Clubbing of fingers in congenital cardiac disease 614
118. Congenital malformations of the kidney and ureters 657
119, 120. Sarcoma of the kidney before and after operation 673
121. Tetany 718

FIGURE PAGE
122. Spasmodic torticollis . . . 731
123. Meningocele . . . 747
124. Encephalocele . . . 747
125. Hydrencephalocele . . . 747
126. Meningocele . . . 747
127. Frontal meningocele . . . 748
128. Naso-frontal meningocele . . . 748
129. Cerebro-spinal meningitis showing frequency . . . 754
130. Posture in cerebro-spinal meningitis . . . 758
131. Temperature chart, cerebro-spinal meningitis . . . 761
132. Tracing of respiration in tubercular meningitis . . . 773
133. Temperature chart in tubercular meningitis . . . 773
134. Chronic basilar meningitis . . . 776
135. Chronic basilar meningitis . . . 777
136. Section of the brain in internal hydrocephalus . . . 791
137. Brain in external hydrocephalus . . . 792
138. Head in chronic hydrocephalus . . . 793
139. Brain showing atrophy . . . 797
140. Convulsions in infantile cerebral paralysis . . . 798
141. Spastic paraplegia . . . 800
142. Infantile hemiplegia showing contractures . . . 802
143–148. Various types of mental defect . . . 805
149. Brain in idiocy . . . 806
150. Chondro-dystrophy, radiograph of skull . . . 810
151. Chondro-dystrophy, long bones . . . 811
152. Chondro-dystrophy, infantile figure . . . 811
153. Chondro-dystrophy, trident hand . . . 812
154. Chondro-dystrophy, adult figure . . . 812
155. A typical cretin . . . 813
156–159. Cretins, showing effect of thyroid treatment . . . 814
160. Spina bifida, meningocele (partially diagrammatic) . . . 821
161. Spina bifida, meningocele, case of . . . 821
162. Spina bifida, meningo-myelocele (partially diagrammatic) . . . 822
163. Spina bifida, syringo-myelocele . . . 823
164. Spina bifida, sacral . . . 823
165. Spina bifida, section of cord in . . . 824
166. Infantile spinal paralysis of lower extremity . . . 835
167. Infantile spinal paralysis of shoulder . . . 836
168. Muscular pseudo-hypertrophy . . . 845
169. Alcoholic neuritis . . . 848
170. Diphtheritic paralysis . . . 849
171. Facial paralysis . . . 854
172. Enlarged thymus . . . 880
173. Acute suppurative adenitis, cervical . . . 885
174. Acute suppurative adenitis, inguinal . . . 885
175. Chain of tuberculous lymph nodes (posterior cervical) . . . 890
176. Cicatrices following tuberculous adenitis . . . 892
177. Section of the spine in Pott's disease . . . 903
178. Hip-joint disease . . . 909
179. Tuberculous dactylitis . . . 914
180. Syphilitic disease of the radius and ulna . . . 916

FIGURE PAGE
181. Syphilitic disease of the tibia 918
182. Syphilitic disease of both tibiæ 919
183. Syphilitic necrosis of the tibia 920
184. Syphilitic dactylitis 921
185. Congenital ichthyosis 923
186. Temperature chart, acute otitis following influenza 944
187. Temperature chart, acute otitis, early paracentesis 945
188. Mastoid abscess 947
189. Temperature charts in scarlet fever, mild cases 959
190. Temperature chart in scarlet fever, typical curve 960
191. Temperature chart in severe uncomplicated scarlet fever 961
192. Temperature chart in fatal septic scarlet fever 962
193. Temperature chart in scarlet fever with late otitis 966
194. Temperature chart in scarlet fever with late nephritis 967
195, 196. Temperature charts in measles, typical curve 983
197. Temperature chart in measles, occasional course 983
198. Temperature chart in measles, prolonged course 984
199, 200. Temperature charts in measles complicated by pneumonia 985
201. Table showing protective power of vaccination 999
202. Vaccination vesicles 1001
203. Temperature chart in pseudo-diphtheria 1059
204. Temperature chart in typhoid fever, short course 1065
205. Temperature chart in typhoid fever, with relapse 1066
206. Tuberculous broncho-pneumonia, diffuse consolidation 1079
207. Cavity from tuberculous broncho-pneumonia 1079
208. A tuberculous nodule 1080
209. Tuberculous broncho-pneumonia, early stage 1081
210. Tuberculous bronchial lymph nodes 1083
211. Temperature chart of tuberculosis following measles 1092
212. Temperature chart of tuberculous broncho-pneumonia, general tuberculosis 1093
213. Temperature chart of tuberculous broncho-pneumonia with softening . . 1094
214. Syphilitic scaling in an infant 1114
215. Syphilitic notched teeth 1116
216. Syphilitic teeth, variously deformed 1117
217. Temperature chart of severe influenza in an infant 1125
218. Temperature chart of acute broncho-pneumonia complicating influenza . 1127
219. Temperature chart, quotidian intermittent fever 1133
220. Temperature chart, tertian intermittent fever 1134
221. Temperature chart in malaria, irregular type 1135

THE DISEASES OF INFANCY AND CHILDHOOD.

PART I.

CHAPTER I.

HYGIENE AND GENERAL CARE OF INFANTS AND YOUNG CHILDREN.

THE physical development of the child is essentially the product of the three factors—inheritance, surroundings, and food. The first of these it is beyond the physician's power to alter; the second is largely and the third almost entirely within his control, at least in the more intelligent classes of society. These two subjects, infant hygiene and infant feeding, are the most important departments of pediatrics.

The Care of the Newly-Born Child.—After the ligature of the cord the child should be wrapped in a thick blanket and placed in a warm room. In hospital practice the eyes should be cleansed with absorbent cotton and water which has been boiled, and then two or three drops of a two-per-cent solution of nitrate of silver, after Credé's method, instilled into each eye by means of a glass rod or eye-dropper. In private practice a saturated solution of boric acid may be substituted, unless the mother has had a purulent vaginal discharge, in which case the silver solution should always be used. The bath should now be given in a warm room; the body being first oiled thoroughly in order to remove the vernix caseosa and then washed in water at a temperature of 100° F. The mouth should be cleansed with plain tepid water and a soft cloth, and no violence employed. The cord may be covered with salicylic acid one part and starch nineteen parts, or simply with subnitrate of bismuth, and wrapped in sterile gauze or surgeon's lint. The abdomen should now be enveloped in a flannel band, eight or ten inches wide, and pinned rather snugly. Before dressing is completed, the child should be submitted to a thorough examination for injuries received during delivery, congenital deformities, also as to the condition of the respiration, circulation, etc.

After dressing, the child should be placed in its crib and covered with blankets, and if the feet are cold, or the fingers and lips a little blue, it should be surrounded by hot-water bottles covered with flannels, and placed near, but not in contact with, the body. The crib should be placed in a quiet, darkened room. The young infant should not occupy the same bed as the mother, unless it greatly needs the warmth of her body, other means of artificial heat not being at hand.

The cord should be kept dry and disturbed as little as possible until it falls off. Under ordinary circumstances the cord separates from the fourth to the seventh day, the average being the fifth day. The stump should then be covered with the salicylic acid and starch powder, and a pad of sterile gauze about one fourth of an inch thick and two inches square applied and secured in position by means of the abdominal band. The purpose of this is to prevent umbilical hernia. The pad should be continued for the first month. The use of stronger antiseptic dressings than that recommended is somewhat objectionable, since it preserves the cord too long and delays separation. The full bath should not be given until the cord has separated.

The physician should always see to it that the infant cries enough to keep the lungs properly expanded.

The question of food for the newly-born infant is considered in the chapter upon infant feeding.

Bathing.—For the first few months the bath should be given at 98° F. The room should be warm, preferably there should be an open fire. The bath should be short and the body dried quickly, without too vigorous rubbing. The addition of salt to the bath is an advantage where the skin is unusually delicate or excoriations are present. One large handful should be used to a gallon of water. By the sixth month the temperature of the bath for healthy infants may be lowered to 95° F., and by the end of the first year to 90° F. Older children who are healthy should be sponged or douched for a moment at the close of the tepid bath with water at 65° or 70° F. During childhood the warm bath is preferably given at night. In the morning a cold sponge bath is desirable. This should be given in a warm room and while the child stands in a tub partly filled with warm water. This cold sponge should last but half a minute, and be followed by a brisk rubbing of the entire body.

In some young infants and even older children there is no proper reaction after the bath, even when given at the temperatures mentioned; children being pale, slightly blue about the lips and under the eyes. All tub bathing, and especially all cold bathing, should then be stopped, since a continuance can only be a drain upon the child's vitality.

Clothing.—The clothing of infants should be light, warm, non-irritating to the skin, and loose enough to allow free motion of the extremities; nor should bands be pinned so tightly about the trunk as to embarrass the movements either of the chest or of the abdomen. The chest should be covered with a woollen shirt, high in the neck and with long sleeves. All petticoats should be supported from the shoulders and not from waistbands. Canton flannel and stockinet are both superior as absorbents to the more commonly used linen diapers. Stockinet has the advantage of being very soft and pliable. Care should be given that in infants the feet be kept warm. If the circulation is very poor, a bag of hot water should always be in the crib. Cold feet are responsible for many attacks of colic and indigestion.

The abdominal band is usually worn during infancy. It cannot be considered in any sense a necessity after the first few months, excepting in cases of very thin infants whose supply of fat in the abdominal walls is an insufficient protection to the viscera. For the first few weeks a band of plain flannel is to be preferred; later, a knitted band with shoulder-straps. The fashion of low neck and short sleeves for infants and very young children has fortunately passed away—let us hope, never to return.

During the summer the outer clothing should be light and the under clothing of the thinnest flannel or gauze. The changes in the temperature of morning and evening may be met by extra wraps. The custom of allowing young children to go with legs bare has many enthusiastic advocates; while it may not be objectionable during the heat of summer, its advantages at any season are very questionable in a changeable climate like that of New York or the Atlantic coast. Many delicate children are certainly injured by such ill-advised attempts at hardening.

The night clothing of infants should be similar to that worn during the day, but should be loose, the material being of the lightest flannel. The night clothing for older children should consist of a thin woollen shirt and a union suit with waist and trousers, and in some cases with feet, if there is a tendency to get outside the coverings. The common mistake is to overload all children, but especially infants, with covering at night. This is an explanation of much of the restless sleep which is seen particularly in delicate children.

Care of the Eyes.—During the first few days at the daily bath, the eyes should be cleansed with a saturated solution of boric acid. They should be carefully protected from too strong light during early infancy. It is desirable that a child should always sleep in a darkened room.

Care of the Mouth and Teeth.—The mouth of the newly-born infant should be gently cleansed at each morning bath with boiled water and a soft cloth. On the first appearance of thrush the mouth should be washed after every feeding with a solution of bicarbonate of soda or borax (twenty grains to the ounce). Harm is often done by the use of too much force in cleansing the mouth of a young infant.

The primary teeth as well as those of the permanent set should receive daily attention. Too often they are neglected altogether. Dirty teeth are likely sooner or later to become carious; and carious teeth, besides being a cause of bad breath and neuralgia, are a constant menace to the health of the child, since they may harbour infectious germs of all varieties. Such teeth should either be filled or removed.

Care of the Skin.—The skin of a young infant is exceedingly delicate, and excoriations, intertrigo, and eczema are of very common occurrence. These conditions are much easier of prevention than of cure. The first essential in the care of the skin is cleanliness, and this must be secured without the use of strong soaps or too much rubbing. Napkins must be removed as soon as soiled or wet. Some bland absorbent powder, like starch, talcum, or the stearate of zinc, should be used in all the folds of the skin, in the neck, in the axillæ, groins, and about the genitals, and in the folds of the thighs, particularly in very fat infants. If plain water produces an undue amount of irritation, the salt or bran bath should be employed.

Care of the Genital Organs.—The female genitals need but little attention in young children, excepting as to cleanliness. This is more often neglected in older children than in infants. Vulvo-vaginitis is very common among the children of the poorer classes where cleanliness is neglected.

In males the prepuce should receive attention during the first few weeks of life. If the foreskin is long and the preputial orifice small, circumcision should invariably be done. If it is not long, but is only adherent, these adhesions should be broken up, the parts thoroughly cleansed and the foreskin retracted daily until there is no disposition to a recurrence of the adhesions. These operations will be discussed more at length in a subsequent chapter. The only thing to be emphasised in the present connection is that the prepuce should receive proper attention in early infancy, since this can now be done with less pain and discomfort to the child, and at the same time better results are obtained. If this matter is neglected during infancy, it is apt to be overlooked until harm has been produced by local or reflex irritation which phimosis or adherent prepuce may have excited.

Vaccination.—This, although considered elsewhere, should be mentioned in this connection as among the things requiring the physician's attention during the first months of life.

Training to Proper Control of Rectum and Bladder.—It is surprising to see what can be accomplished by intelligent efforts at training in these particulars. An infant can often be trained at three months to have its movements from the bowels when placed upon a small chamber. This not only saves a great amount of washing of napkins, but there is soon formed a habit of having the bowels move at a regular time or times each day. The infant must be put upon the chamber soon after its feeding. The importance of training young children to regular habits regarding evacuations from the bowels can hardly be overestimated. It should be impressed upon every mother and nurse by the physician, and especially the necessity of beginning training during infancy. Much of course will depend upon the food and the digestion; but habit is a very large factor in the case.

The training of the bladder is not quite so important, but the proper education of this organ adds much to the comfort of the child and the ease with which it is cared for. Before the end of the first year most intelligent children can be trained to indicate a desire to empty the bladder. Many mothers and nurses succeed in training children so well that by the tenth or eleventh month napkins are dispensed with during the day. On the other hand, it is very common to see children of two and even two and a half years still wearing napkins because of the lack of proper training. Before it has reached the latter age a healthy child should go from 10 P. M. until morning without emptying the bladder. The annoyance and discomfort from the neglect of early training in this particular are very great. Night feeding is responsible for much of the difficulty experienced in training children to hold the water during the night.

General Hygiene of the Nervous System.—Great injury is done to the nervous system of children by the influences with which they are surrounded during infancy, especially during the first year. The brain grows more during the first two years than in all the rest of life. Normal healthy development of the nervous centres demands quiet, rest, peaceful surroundings, and freedom from everything which causes excitement or undue stimulation.

The steadily increasing frequency of functional nervous diseases among young children is one of the most powerful arguments for greater attention by physicians to the subject of the hygiene of the nervous system during infancy. Most parents err through ignorance. Playing with young children, stimulating to laughter and exciting them by sights, sounds, or movements until they shriek with apparent delight, may be a source of amusement to fond parents and admiring spectators, but it is almost invariably an injury to the child. This is especially harmful when done in the evening. It is the plain duty of the physician to enlighten parents upon this point, and insist that the infant shall be kept quiet, and that all such playing and romping as has been referred to shall, during the first year at least, be absolutely prohibited.

Sleep.—The sleep of the newly-born infant is profound for the first two or three days and under normal conditions almost continuous. In the case of prolonged or tedious labor, or where from any cause undue compression has been exerted upon the head, it may approach the condition of semi-coma for twenty-four or forty-eight hours. This may be so deep as to excite apprehensions of serious brain lesions. If, however, there are associated with it no convulsions and no rigidity, this early stupor usually passes away on the second or third day.

The sleep of early infancy is quiet and peaceful, but not very deep after the first month. After the third year the heavy sleep of childhood is commonly seen. A healthy infant during the first few weeks sleeps from twenty to twenty-two hours out of the twenty-four, waking only from hunger, discomfort, or pain. During the first six months a healthy infant will usually sleep from sixteen to eighteen hours a day, the waking periods being only from half an hour to two hours long. At the age of one year most infants sleep from fourteen to fifteen hours, viz., from eleven to twelve hours at night, and two or three hours during the day, usually in two naps. When two years old usually thirteen to fourteen hours' sleep are taken; eleven or twelve hours at night and one or two hours during the day, generally in a single nap. At the age of four years children require from eleven to twelve hours' sleep. It is always desirable, and in most cases with regularity it is possible, to keep up the daily nap until children are four years old. From six to ten years the amount of sleep required is ten or eleven hours, and from ten to sixteen years nine hours should be the minimum.

Training in proper habits of sleep should be begun at birth. From the outset an infant should be accustomed to being put into its crib while awake and to go to sleep of its own accord. Rocking and all other habits of this sort are useless and may even be harmful. An infant should not be allowed to sleep on the breast of the nurse, nor with the nipple of the bottle in its mouth. Other devices for putting infants to sleep, such as allowing the child to suck a rubber nipple or anything else, are positively injurious. If such means of inducing sleep are resorted to the infant soon acquires the habit of not sleeping without them. I have known of one instance where the habit of rocking during sleep was continued until the child was two years old; the moment the rocking was stopped the infant would wake, and in consequence this practice was continued by the devoted but misguided parents. A quiet, darkened room, a warm and comfortable bed, an appetite satisfied, and dry napkins are all that are needed to induce sleep in a healthy child.

The periods of sleep in young infants are usually from two to three hours long, with the exception of once or twice in the twenty-four hours, when a long sleep of five or six hours occurs. The purpose of training is to have the child take this long sleep at night. The habit of regular sleep is best established by wakening the infant regularly every two or two and a half hours during the day for feeding, and allowing it to sleep as long as possible during the night. This training goes hand-in-hand with regular habits of feeding. Such habits are easily formed if the plan be systematically followed from the outset.

By the fifth month all feeding between 10 P. M. and 7 A. M. should be discontinued. If this is done most infants can be trained by this time to sleep all night. If the room is lighted, and the child taken from the crib or rocked or fed as soon as it wakens at night, there is no such thing as the formation of good habits of sleep. Regularity in sleep and feeding not only make the care of young infants very much easier, but they are of a good deal of importance for the health of the child.

The causes of disturbed or irregular sleep in young infants are mainly two—hunger and indigestion. In nursing infants it is usually the former; in those artificially fed usually the latter. Sleeplessness from hunger is often seen in children who are nursed thirty or forty minutes and then fall asleep, but wake in fifteen or twenty minutes crying and fretful. After being quieted they may fall asleep again for half an hour, but wake at short intervals. The peaceful sleep of two or three hours which should follow a proper feeding is never seen. With this restlessness, in indigestion other signs are usually present, such as bad stools, stationary weight, etc. The disturbed sleep due to overfeeding shows itself by much the same symptoms, excepting that the first sleep after the meal is usually longer.

Exercise.—This is no less important in infancy than in later childhood. An infant gets its exercise in the lusty cry which follows the cool sponge of the bath, in kicking its legs about, waving its arms, etc. By these means pulmonary expansion and muscular development are increased and the general nutrition promoted. An infant's clothing should be such as not to interfere with its exercise. Confinement of the legs should not be permitted. In hospital practice I have often had a chance to observe the bad results which follow when very young infants are allowed to lie in the cribs nearly all the time. Little by little the vital processes flag, the cry becomes feeble, the weight is first stationary, then there is a steady loss. The appetite fails so that food is at first taken without relish, then at times altogether refused; later, vomiting ensues and other symptoms of indigestion. This, in many cases, is the beginning of a steady downward course which goes on until a condition of hopeless marasmus is reached. Such infants must be taken up every few hours and carried about the wards; the position should be frequently changed, and general friction of the entire body employed at least twice a day. Every means must be made use of to stimulate the vital activity. The value of systematic attention to these matters cannot be overestimated in hospitals for infants. Infants who are old enough to creep or stand usually take sufficient exercise unless they are restrained. At this age they should be allowed to do what they are eager to do. Every facility should be afforded for using their muscles. Exercise may be encouraged by placing upon the floor in a warm room a mattress or a thick "comfortable," and allowing the infant to roll and tumble upon it at will. A large bed may answer the same purpose.

In older children every form of out-of-door exercise should be encouraged—ball, tennis, and all running games, horseback riding, the bicycle, tricycle, swimming, coasting, and skating. Up to the eleventh year no difference need be made in the exercise of the two sexes. Companionship is a necessity. Children brought up alone are at a great disadvantage in this respect, and are not likely to get as much exercise as they require. The amount of exercise allowed delicate children should be regulated with some degree of care. It may be carried to the point of moderate muscular fatigue, but never to muscular exhaustion. The latter is particularly likely to be the case in competitive games.

Exercise should have reference to the symmetrical development of the whole body. In prescribing it the specific needs of the individual child should be considered. By carefully regulated exercises very much may be done to check such deformities as round shoulders and slight lateral curvature of the spine, and also to develop narrow chests and feeble thoracic muscles. For purposes like these, gymnastics are exceedingly valuable to supplement out-of-door exercise, but they can never take their place.

There are two important points with reference to exercise indoors: First, the playroom should be cool—from 60° to 65° F.; never above this point. Secondly, during all active exercise the clothing should be loose and light, so as to allow the freest possible motion of the body.

Airing.—In summer there can be no possible objection to a young infant being allowed out of doors at the end of the first week. It should be kept in the open air as much as possible during the day. In the fall and spring this should not be permitted until the child is at least a month old, and then only when the out-of-door temperature is above 60° F. During its outing the head should be protected from the wind and the eyes from the sun. The duration of the outing at first should be only fifteen or twenty minutes, the time being gradually lengthened to two or three hours. The child should be gradually accustomed to changes of temperature in the room by opening wide the windows for a few minutes each day even before it is taken out of doors, the child being dressed meanwhile as for an outing. In the case of children born late in the fall or in the winter this means of giving fresh air may be advantageously begun at one month and followed throughout the first winter. It is only necessary in all such cases that the changes be made very gradually both as to the length of the airing and to the temperature. The great advantage of this plan over that more commonly followed of keeping young infants closely housed for the first six months in case they are born in the fall or early winter, I can positively affirm from quite a wide observation of both methods. It is a matter of very serious importance that every infant be furnished an abundance of pure fresh air in winter as well as in summer. When the plan above outlined is carefully and judiciously followed, the tendency to catarrhal affections instead of being increased is thereby greatly lessened.

When four or five months old, there is no reason why a healthy child should not go out of doors on pleasant days if the temperature is not below 20° F. While there is a prejudice on the part of many mothers and some physicians against a child's sleeping out of doors in cold weather, it is a practice which I have always urged upon mothers, and have never seen followed by any but the most beneficial results. The days of all others when infants and very young children should not be out of doors are when there are high winds, especially those from the northeast, an atmosphere of melting snow, and during severe storms. Delicate infants must of course be more carefully guarded during the cold season. With most of these the plan of house-airing is all that should be attempted.

Nursery.—This should be the sunniest and best-ventilated room in the house. It is the physician's duty to see that proper attention is paid to the hygiene of the room in which the child spends at least four-fifths of its time during the first year, and two-thirds of its time during the first two or three years of life. Sunlight is absolutely indispensable. Sunny rooms always contain less organic matter and less humidity, and hence a room upon the north side of the house should always be avoided, preferably one in the second story should be chosen. Nothing which can in any way contaminate the air of the room should be allowed. There should be no drying of clothes or of napkins, and no plumbing. No food should be allowed to stand about the room. The gas should not be allowed to burn at night; a small wax night-light furnishes all that is needed in the nursery. If possible the heat should be from an open fire; the next best thing is the Franklin radiator. Nothing in the room is worse than steam heat from a radiator unless it be a gas stove which under no circumstances should be allowed, excepting possibly for a few minutes each morning during the bath.

The temperature of the room during the day should be 70° F., but better 68° than 72° F. It is important that every nursery should have a thermometer, and that this and not the sensations of the nurse should be the guide. It is almost invariably true that the nursery is overheated. Often no other explanation can be found for chronic indigestion and falling weight excepting a nursery whose habitual temperature ranges from 75° to 80° F. At night for the first few months the temperature should not be allowed to fall below 65° F. After the first year the night temperature may fall to 60° or even 50° F.

Free ventilation without draughts is an absolute necessity. This is best accomplished by ventilators in the windows, of which there are many excellent devices sold in the shops. While the child is absent from the room the windows should be widely opened and free airing of the nursery accomplished. The room should always be thoroughly aired at night before the child is put to bed. The window may be kept open even in the first year, unless the temperature out of doors is below 35° F. After the first year the window may be open, unless the outside temperature is as low as

20° F. If the window is open the door of the nursery should be closed, that currents of air may be avoided. The ventilation by means of an open fire is the most efficient.

The furniture of the nursery should be as simple as possible, heavy hangings should be positively forbidden, and upholstered furniture used only to a small extent. Floors covered by large rugs are much more cleanly than carpets, and hence are to be preferred.

The child, whenever it is possible, should have a separate bed; and so should the newly-born infant, in order to prevent the danger of overlying by the mother, which among the lower classes is a frequent cause of death, and also to avoid the danger of too frequent night nursing, which is injurious alike to mother and child. Separate beds for older children will prevent the spread of many forms of infection from the diseased child to the healthy. The cradle for infants should be one which does not rock, in order that this unnecessary and vicious practice should not be carried on. The mattress should be of hair and quite firm. The pillow should be small; in the summer, hair pillows are an advantage but not a necessity. The position of the child during sleep should be changed from time to time from one side to the other and then to the back. Attention to all these details should not be beneath the physician's notice, since the violation of these plain rules of hygiene is at the bottom of many of the milder disorders and even of some of the more serious diseases seen in infancy.

The Nurse.—The nurse of a young child should be healthy, young or in middle life, free from tuberculous or syphilitic taint, and from catarrhal affections of the nose and throat. She should be neat in habit, of quiet disposition, and, most of all, she should be a person of intelligence.

The Amount of Air Space required by Infants.—The nursery should always be as large a room as possible. One of the reasons why young infants do so badly in institutions is because of overcrowding. In a well-ventilated ward there should be allowed to each infant at least 1,000 cubic feet for the best results. Children over two years old are not so sensitive to their surroundings, and may thrive in wards where only 700 or 800 cubic feet are allowed to each child.

THE CARE OF PREMATURE AND DELICATE INFANTS.

Infants born before term, and some exceedingly delicate ones which are born at full term, require very special and particular care. The vitality is so feeble in these children that if they are handled in the ordinary way they survive at most but a few weeks. The symptom which indicates that such special care is necessary is most of all the weight of the child. Either congenital feebleness or prematurity may be assumed in most of the children weighing less than four pounds; also if the length of the body is less than nineteen inches. In these children all the organs are likely to be imperfectly developed and they are not ready for their work. Especially is this true of the lungs and of the organs of digestion.

The clinical picture presented by these cases is quite characteristic. The body is limp; the skin very soft and delicate and almost transparent; the cry, a low feeble whine not unlike the mew of a kitten; the respiratory movements, extremely irregular, sometimes scarcely perceptible for several seconds; the movements of the extremities infrequent and never vigorous. The general appearance is one of torpor. The muscles of the mouth and cheek and tongue may lack the requisite force for sucking, so that this is practically impossible, and even deglutition is slow, difficult, and prolonged. It is difficult to maintain the normal body temperature; unless closely watched this may fall far below the normal, and may rise quite as much above it with the use of too much artificial heat. I once saw a fluctuation of 13° F. occur in a few hours from such causes. All the symptoms mentioned vary much according to the degree of prematurity.

In the management of these cases there are two problems to be solved: the first to maintain the animal heat, the second to nourish the infant. Difficult as it always is to rear a premature infant, these difficulties are much increased in cases where proper means are not adopted immediately after birth. The loss which these children sustain during the first few days is in very many cases so great that subsequent measures, however well carried out, are futile. The heat-producing power is so feeble that the body temperature quickly falls below normal unless artificial heat is constantly used. The effect of cold upon these delicate infants is very serious, and not only growth but even life depends upon maintaining the body temperature steadily and uniformly. Their extreme susceptibility is something which it is difficult for one to appreciate who has not had experience in these cases.

One of the simplest means of maintaining the temperature is to oil the skin and then roll the entire body, including extremities, in cotton batting; even the neck and cranium may be covered, leaving only the face exposed. The usual diaper may be replaced by a pad of gauze and absorbent cotton. The body is then wrapped in blankets, placed in a clothes-basket or bassinet with protected sides, and surrounded by bottles or bags containing hot water. A blanket or sheet should partially cover the top of the basket, forming a sort of hood to protect the eyes from light and the face and head from draughts. In using hot-water bags, some caution must be exercised or too much heat may be secured. I have seen the temperature of an infant raised six or seven degrees from this cause. The temperature of the child should at first be taken every few hours to make sure that a proper amount of external heat is supplied, but not too much.

A much better means of furnishing artificial heat is the electric pad known also as the "electrotherm." * These small heaters are attached to an electric fixture like a drop-light. A convenient size is ten by fifteen inches. It is placed between two or three thicknesses of blanket, upon which the infant lies in its basket. Three grades of heat can be obtained, according to the amount of electricity turned on.

This mode of handling premature infants has been given a thorough trial in the Babies' Hospital and has been found to fulfil the indications with children as small as three pounds and as young as seven months quite as well as the incubator, at the same time being free from its dangers. It has not even been necessary, though perhaps desirable, to raise the general temperature of the room. But these patients, when kept in the ward at ordinary temperature, have maintained an even body temperature much more uniformly than I have seen with any other method—the incubator included.

Premature infants should be disturbed as little as possible. The body should be oiled, and fresh cotton applied about once in three days. The feeding may be done without removing the child from its bed.

Incubators.—The essential things in an incubator are means of maintaining a uniform temperature and efficient ventilation; since the dangers of infection are great, absolute cleanliness is indispensable. The temperature for the youngest and most delicate infants should be from 90° to 95° F.; for those somewhat older and stronger, from 85° to 90° F. Ventilation is much more easily secured when the air admitted to the incubator is considerably below these figures, or not above 60° or 65° F. The incubator should therefore stand in a large cool room or communicate with the outside air. A thermostat attachment is a great advantage, as is also filtration of the air through cotton. Metal construction allows greater cleanliness and more complete disinfection. The incubator of Lion (Nice) seems to fulfil all these requirements better than any other yet constructed. A similar one is shown in the illustration. It is necessary to watch not only the temperature of the incubator, as registered by a thermometer beside the baby, but the rectal temperature should be taken every few hours; fluctuations between 97.5° and 100.5° F. are unimportant. If the variations are much wider, the temperature of the apparatus should be modified accordingly. On account of the difficulties and dangers inherent in small incubators, Escherich has devised an "incubator room" in

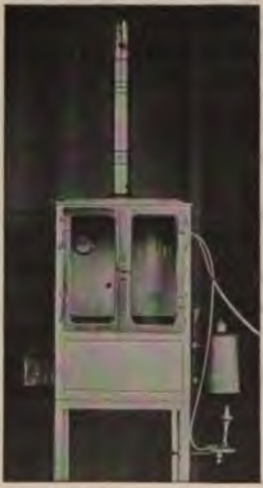

FIG. 1.—Incubator.

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* Obtained of Simplex Electric Heating Co., 39 Cortlandt Street, New York.

which several infants can be accommodated. It is four by eight feet, and six feet high. The nurse can enter this, and thus the removal of the child for feeding or any other purpose is avoided.

Every incubator baby requires close and constant attention, and results depend upon nothing so much as the intelligence and watchfulness of the nurse. In hospitals with nurses skilled in this particular line of work, excellent results are obtained; but outside of such institutions, with the usual obstetric nurse, the chances of failure are many. The incubator requires practically the entire time of one person by night and by day. No matter how carefully constructed, perfect ventilation is difficult to maintain, and with the infant's imperfectly expanded lungs attacks of asphyxia are very likely to occur. A cylinder of oxygen should be at hand for use in such emergencies. Taking everything into consideration, I am not inclined to recommend the use of the incubator except in institutions. Elsewhere the difficulties and dangers are so many and so great that in the majority of cases I believe better results will be obtained with the other means mentioned of maintaining body heat, particularly the electric pad.

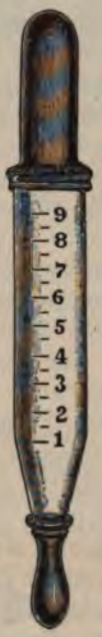

Fig. 2.—Breck's feeding-tube.

Feeding.—The feeding of the premature infant is not less important than the maintenance of heat and proper ventilation. Infants at eight months and those weighing five pounds or thereabouts can usually be made to take the breast after the first few days. Few below this age or weight will do so. Some will suck from a bottle, but the majority must be fed by other means. A medicine dropper may be used, or the Breck feeder (Fig. 2); the smallest and feeblest, however, must be fed by gavage, using a funnel and small rubber catheter. The food should be slowly given; if rapidly, some is liable to be regurgitated, and this may produce attacks of asphyxia or even an aspiration pneumonia. The quantity of food and frequency of feeding will depend upon the size and age of the child. A seven months' baby weighing three and a half pounds should have, for the first twenty-four to thirty-six hours, only water, one to three teaspoonfuls every hour. Then regular food, half an ounce every hour, gradually increased to an ounce every hour and a half at the end of two weeks, and an ounce and a half every two hours at the end of three or four weeks. Artificial feeding I have not found very successful with premature infants. With some of the larger and more vigorous, cow's milk modified according to the directions given in the chapters on Infant Feeding gives good results. I have once succeeded with a child of three pounds two ounces. For most of them under four and a half pounds, breast milk is essential. The mother may furnish milk in a few cases if the child is born near term, and occasionally at eight months, but seldom earlier, so that a wet nurse must usually be depended upon. If the mother's milk is to be used, unless the child is very vigorous, it is better to pump her breasts and feed the baby with the dropper, in order that one may know exactly how much the child is getting; since acute inanition from nursing upon breasts which have little or no milk is not an uncommon experience. In choosing a wet nurse it is not necessary that her child be a very young one. Since the milk must always be diluted at first, that of a woman whose child is between two weeks and two months old may answer. The milk is at first diluted with an equal amount of a 5-per-cent solution of milk sugar. The milk of a wet nurse will usually diminish rapidly in amount, and often change in quality when her breasts are pumped continually; it is therefore better in most cases to have her nurse her own child at the same time, either wholly or in part, for a few weeks, until the premature infant is able to take the breast.

The results with premature babies will depend very much upon how soon after birth they receive proper care. If an incubator is to be used it should be in readiness, so that the child can be put into it as soon as it is breathing properly. If the incubator is not employed until the child is several days old and is losing rapidly, the chances are poor. The age and vigour of the infant are of the greatest importance in estimating the chances of survival. The following table gives Tarnier's statistics, showing the percentage of premature infants saved during a period of five years without the incubator, and during the succeeding five years with the incubator; also the percentage saved at the Sloane Hospital (New York), as published by Voorhees: *

AGE.	Tarnier saved without incubators.	Tarnier saved with incubator.	Voorhees saved with incubators.	Voorhees saved excluding cases dying a few hours after birth.
Born at 6 months..........	0·0	16·0	....	....
" " 6½ "	21·5	36·6	22·0	66·0
" " 7 "	39·0	49·8	41·0	71·0
" " 7½ "	54·0	77·0	75·0	89·0
" " 8 "	78·0	88·8	70·0	91·0
" " 8½ "	88·0	96·0	....	....

Results will improve with the experience of the physician in the feeding and care of these very sensitive patients. Much is yet to be learned about them.

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* Archives of Pædiatrics, May, 1900. An excellent article on the Care of Premature Babies in Incubators.

CHAPTER II.

GROWTH AND DEVELOPMENT OF THE BODY.

Observations upon growth and development are of the utmost importance during infancy and childhood. Only by this means are very many diseases detected in their incipiency. Early recognition carries with it in most cases the possibility of checking such pathological processes as, if allowed to go on, may affect the health not only in infancy but even throughout life.

By familiarity with what is normal, detection of the abnormal soon becomes easy. Investigation in regard to these subjects should be made a part of the physical examination of every child.

WEIGHT.

The weight of the infant is the best means we have to measure its nutrition. It is as valuable a guide to the physician in infant feeding as is the temperature in a case of continued fever. Although the weight is not to be taken as the only guide to the child's condition, it is of such importance that we cannot afford to dispense with it during the first two years. It is a great advantage to keep up regular observations during childhood.

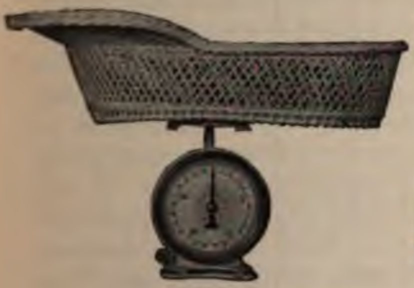

Fig. 3.

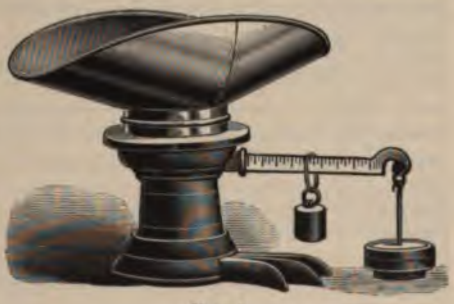

Fig. 4.

Weekly weighings should be made for the first six months, bi-weekly for the rest of the first year, and monthly during the second year. Delicate children should be weighed even more frequently. Satisfactory scales of moderate price for domestic use are sold in most of the shops as "Infants' Scales" (Fig. 3). These weigh up to twenty-four pounds and indicate ounces. For hospital use and for very fine observations more accurate scales are needed. In Fig. 4 are shown the scales I employ; they weigh up to sixty-one pounds and indicate half ounces.*

Weight at Birth.—The following figures are taken consecutively in nearly equal proportion from the records of the Nursery and Child's Hospital, the Sloane Maternity, and the New York Infant Asylum, and include only full-term children:

Average weight of 568 females	7·16 lbs.	(3,260 grammes).
" " 590 males	7·55 "	(3,400 ").
" " 1,158 infants	7·35 "	(3,330 ").

Weight Curve during the First Few Weeks.—The accompanying chart represents the variations in weight for the first twenty days. These observations were made upon one hundred healthy, nursing infants, fifty males and fifty females, at the Nursery and Child's Hospital. The children were weighed daily during the period of observation. The average weight at birth was 7·1 pounds. The curve shows a very marked loss of weight on the first day and a slight loss on the second day, the lowest point being touched at the beginning of the third day; but from this time there was a steady gain. The average initial loss in these cases was ten ounces, being in each sex exactly eleven per cent of the body weight. In eight hundred and thirty-five cases, however, including those above mentioned, the average loss was nine and a half ounces. The loss of the first days is chiefly due to the discharge of the meconium and urine, but is in part from the excess of tissue waste over the nutriment derived from the breasts. After the third day, coincident with an abundant secretion

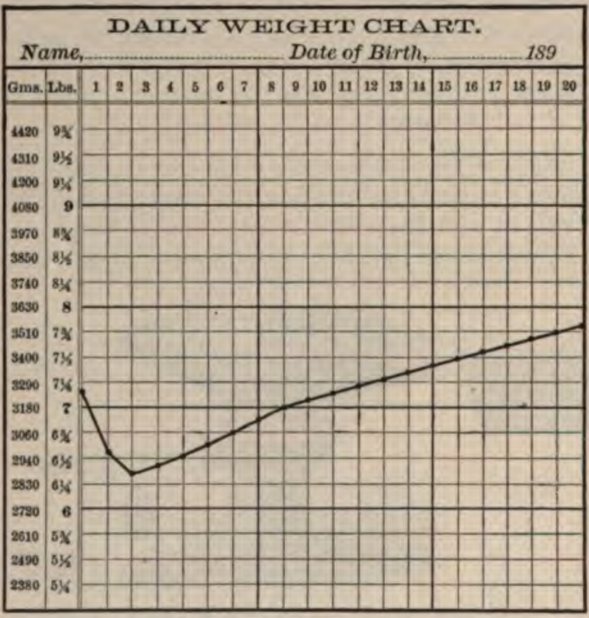

FIG. 5.—Weight curve of the first twenty days.

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* These are made by the Howe Scale Company.

of milk, there is a steady, daily increase in weight. If the milk is very scanty or is wanting altogether, the loss in weight continues.

The birth-weight of nursing children who thrive normally is regained on the average on the tenth day. The most frequent deviation from the normal curve consists in a continued loss or stationary weight after the third day. This may be due to acute illness, such as bronchitis, diarrhœa, pyæmia, or hæmorrhage, but in the majority of cases there is a disturbance of nutrition from improper or insufficient food. This is quite as likely to be the case in nursing infants as in those who are artificially fed. Under these circumstances the loss may continue indefinitely, and it may be slow or rapid according to the character of the nursing or feeding.

The weight curve of infants who are artificially fed, even though they are strong and vigorous and the feeding properly done, rarely follows for the first month the same lines as that of nursing infants. We usually see an initial loss which is about the same as in nursing infants, then a period of nearly stationary weight lasting from one to two weeks. After this the steady regular gain begins, and is quite equal to that of nursing infants. This period of stationary weight is to be expected while the infant is becoming accustomed to his new food. The chief danger at this time is that the physician, because there is no gain, may be led to increase either the strength or the quantity of the food so rapidly as to upset the child's digestion.

There are cases in which an excessive loss of weight during the first three or four days is associated with an elevation of temperature, but without any other evident signs of disease. Both the fever and the rapid loss in weight are to be looked upon as due to the same cause—inanition. This will be more fully considered in the chapter devoted to that subject.

Excessive loss in weight during the first few days from any cause whatsoever, seriously handicaps an infant during the first weeks of its life. The great importance of this has not been sufficiently appreciated. Loss in weight after the third day is an indication for food in addition to that derived from the breast.

Weight Curve of the First Year.—The curve of the accompanying chart is made up from complete weight charts of one hundred healthy nursing infants who were thriving and weighed every week, and the incomplete charts of about three hundred other infants. There are represented in round numbers about ten thousand observations on children under one year. The period of most rapid increase is during the first three months. It is slowest from the sixth to the ninth month. This curve is not to be regarded as a normal line, like the normal line of the temperature chart, but as an average line. An infant who is at birth a pound above the average may keep this distance above the line for the whole year; another weighing one pound less than the average may be as far below it. Girls throughout the year are on the average half a pound lighter than boys. No single child exactly follows the line all the way, but it is surprising how close to it a very large number of the cases come.

In artificially-fed infants—provided the feeding is properly done—the curve does not differ essentially from that of breast-fed infants, excepting in the slower gain of the first month, although this difference is usually made up before the sixth month is reached.

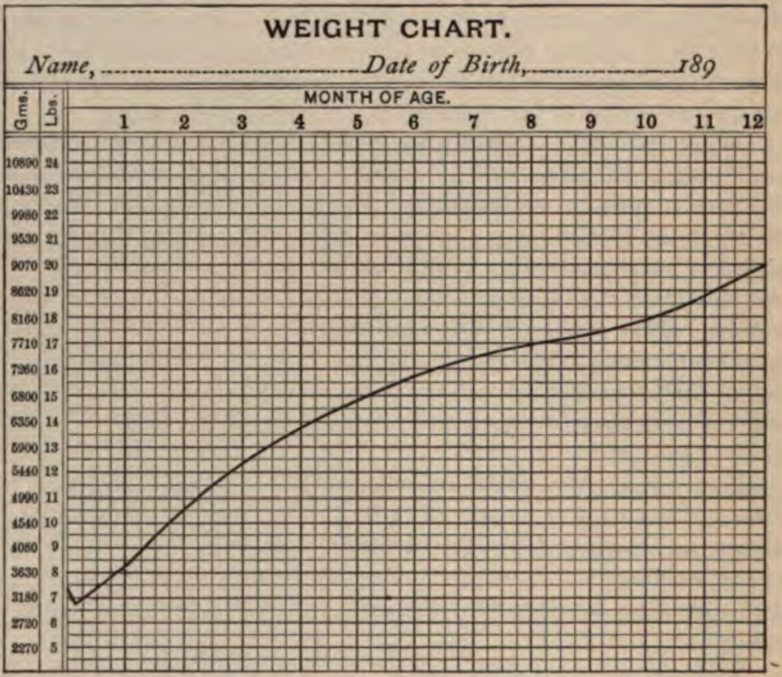

FIG. 6.*—The weight curve of the first year.

At the end of the first year the average child weighs nearly three times as much as at birth. Perfect health during the first year is consistent only with a steady gain in weight. A child may not always gain rapidly, but it should gain steadily, and if it does not, something is wrong. All the conditions surrounding the infant should be investigated, but especially the food. One should not be satisfied unless the average weekly gain during the first six months is at least four ounces. In the second six months it may be slightly less. As a rule a child who gains regularly in weight is thriving; an exception must, however, be made in the case of some infants who are fed chiefly upon carbohydrate foods.

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* Blank weight charts are made by Geo. L. Goodman & Co., Pearl Street, New York.

Weight from the Second to the Fifth Year.—Comparatively few observations have been published upon the weight during this period. From three hundred and seventy-two personal observations it appears that the gain is about six pounds during the second year, about four and a half during the third year, and about four pounds during the fourth year: the actual weights are given in the large table (page 20). During this period the gain is rarely steady even in the second year. With most children it is slowest or the weight is stationary in the summer months, while the most rapid increase is usually seen in autumn. Throughout this period the girls gain in about the same ratio as boys, but remain on the average nearly one pound lighter. During almost every illness, no matter of what character, the gain in weight ceases, and usually there is a loss, the rapidity and extent of which are somewhat proportionate to the severity of the attack; but it is always much more rapid in diseases of the digestive tract than in any other form of illness.

Weight of Older Children.—The weights given in the table of children from five to fourteen years are from Bowditch. Observations were made upon children of American parentage in the public schools of Boston—upon 4,327 boys and 3,681 girls.* It is to be remembered that these weights include the ordinary clothing, while those below five years are without clothing.†

The slowest gain is from the fifth to the eighth year, when it is about four pounds a year. From the eighth to the eleventh year it rises to about six pounds a year. Up to the eleventh year the two sexes gain in about the same ratio. From the eleventh to the thirteenth year the girls gain

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* W. T. Porter has published (1894) observations made upon 14,744 children of American parentage in the public schools of St. Louis. His figures show quite a variation from those of Bowditch, and are as follows:

AGE.	BOYS' WEIGHT.		GIRLS' WEIGHT.	
	Kilos.	Pounds.	Kilos.	Pounds.
6 years	19·66	43·2	18·76	41·3
7 "	21·67	47·7	20·82	45·8
8 "	23·91	52·6	22·71	50·0
9 "	26·08	57·4	25·07	55·1
10 "	28·49	62·7	27·43	60·3
11 "	31·26	68·8	29·93	65·8
12 "	33·45	73·6	33·17	73·0
13 "	35·96	79·1	38·29	84·2
14 "	40·34	88·7	43·12	94·9
15 "	47·25	103·9	46·90	103·2
16 "	52·10	114·6	50·06	110·1

† The average weight of the ordinary house clothing of school children, according to Bowditch, is at five years 2·8 pounds for both sexes; at seven years, 3·5 for both sexes; at ten years, 5·7 pounds for boys and 4·5 pounds for girls; at thirteen years, 7·4 pounds for boys and 5·6 pounds for girls; at sixteen years, 9·7 pounds for boys and 8·1 pounds for girls. This must be deducted from weights given to obtain the net weight.

much more rapidly, passing the boys for the first time and maintaining this lead until the fifteenth year, when again the boys pass them.

*Table showing Weight, Height, and Circumference of the Head and Chest from Birth to the Sixteenth Year.**

AGE.	Sex.	WEIGHT.		HEIGHT.		CHEST.		HEAD.	
		Pounds.	Kilos.	Inches.	Cm.	Inches.	Cm.	Inches.	Cm.
Birth........	**Boys.**	**7·55**	**3·43**	**20·6**	**52·5**	**13.4**	**34.2**	**13·9**	**35·5**
	Girls.	7·16	3 26	20·5	52·2	13·0	33·2	13·5	34·5
6 months	**Boys.**	**16·0**	**7·26**	**25·4**	**64·8**	**16·5**	**42·0**	**17·0**	**43·5**
	Girls.	15·5	7·03	25·0	63·6	16·1	41·0	16·6	42·2
12 months....	**Boys.**	**20·5**	**9·29**	**29·0**	**73·8**	**18·0**	**45·9**	**18·0**	**45·9**
	Girls.	19·8	8·84	28·7	73·2	17·4	44·4	17·6	44·6
18 months....	**Boys.**	**22·8**	**10·35**	**30·0**	**76·3**	**18·5**	**47·1**	**18·5**	**47·1**
	Girls.	22·0	9·98	29·7	75·6	18·0	45·9	18·0	45·9
2 years.......	**Boys.**	**26·5**	**12·02**	**32·5**	**82·8**	**19·0**	**48·4**	**18·9**	**48·2**
	Girls.	25·5	11·56	32·5	82·8	18·5	47·0	18·6	47·2
3 years.......	**Boys.**	**31·2**	**14·14**	**35·0**	**89·1**	**20·1**	**51·1**	**19·3**	**49·0**
	Girls.	30·0	13·60	35·0	89·1	19·8	50·5	19·0	48.4
4 years.......	**Boys.**	**35·0**	**15·87**	**38·0**	**96·7**	**20·7**	**52·8**	**19·7**	**50.3**
	Girls.	34·0	15·41	38·0	96·7	20·5	52·2	19·5	49.6
5 years.......	**Boys.**	**41·2**	**18·71**	**41·7**	**106·0**	**21·5**	**54·8**	**20·5**	**52.2**
	Girls.	39·8	18·06	41·4	105·3	21·0	53·5	20·2	51.3
6 years.......	**Boys.**	**45·1**	**20·48**	**44·1**	**112·0**	**23·2**	**59·1**	....	......
	Girls.	43·8	19·87	43·6	110·9	22·8	58·3	....	......
7 years.......	**Boys.**	**49·5**	**22·44**	**46·2**	**117·4**	**23·7**	**60·6**	....	......
	Girls.	48·0	21·78	45·9	116·7	23·3	59·5	....	......
8 years.......	**Boys.**	**54·5**	**24·70**	**48·2**	**122·3**	**24·4**	**62·2**	....	......
	Girls.	52·9	24·01	48·0	122·1	23·8	60·8	....	......
9 years.......	**Boys.**	**60·0**	**26·58**	**50·1**	**127·2**	**25·1**	**63·9**	....	......
	Girls.	57·5	26·10	49·6	126·0	24·5	62·5	....	......
10 years......	**Boys.**	**66·6**	**30·22**	**52·2**	**132·6**	**25·8**	**65·6**	**21·0**	**53·5**
	Girls.	64·1	29·07	51·8	131·5	24·7	63·0	20·7	52·8
11 years......	**Boys.**	**72·4**	**32·83**	**54·0**	**137·2**	**26·4**	**67·2**	....	......
	Girls.	70·3	31·87	53·8	136·6	25·8	65·8	....	......
12 years......	**Boys.**	**79·8**	**36·21**	**55·8**	**141·7**	**27·0**	**68·8**	....	......
	Girls.	81·4	36·90	57·1	145·2	26·8	68·3	....	......
13 years......	**Boys.**	**88·3**	**40·04**	**58·2**	**147·7**	**27·7**	**70·6**	....	......
	Girls.	91·2	41·36	58·7	149·2	28·0	71·3	....	......
14 years......	**Boys.**	**99·3**	**45·03**	**61·0**	**155·1**	**28·8**	**73·3**	....	......
	Girls.	100·3	45·50	60·3	153·2	29·2	74·1	....	......
15 years......	**Boys.**	**110·8**	**50·26**	**63·0**	**159·9**	**30·0**	**76·6**	**21·8**	**55·5**
	Girls.	108·4	49·17	61·4	155·9	30·3	76·8	21·5	54·8
16 years......	**Boys.**	**123·7**	**56·09**	**65·6**	**166·5**	**31·2**	**79·2**	....	......
	Girls.	113·0	51·24	61·7	156·7	30·8	78·8	....	......

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* The observations of Boas (Science, April 12, 1895) upon 4,319 children over six years old show that first born exceed children born at a later period both in height and weight.

HEIGHT.

The figures showing the height at different ages are given in the foregoing table. The measurements of infants at birth are taken in about equal numbers from the records of the New York Infant Asylum and the Sloane Maternity Hospital. They were made upon full-term infants.

Average length of	231 males	20·61 inches	(52·5 cm.);
" "	211 females	20·47 "	(52·2 ");
" "	442 infants	20·54 "	(52·35 ").

The most rapid gain in length is in the first year. During this period the child grows on an average a little over eight inches (21 cm.). This gain is usually, but not always, proportionate to the increase in weight. During the second year the average increase is three and a half inches (9 cm.). From this time on the rate of increase is quite uniform in both sexes until the eleventh year, it being between two and three inches a year.

After the eleventh year in girls and the twelfth in boys the growth is much more rapid. In height the girls exceed the boys at the twelfth and thirteenth years for the only time in their growth.

In the figures given in the preceding table those of five years and over are taken from Bowditch, the observations being made upon the same children as those whose weights were taken. The observations from six months to four years inclusive are from original sources, and are drawn from about five hundred cases. The height much more than the weight of children is modified by hereditary influences.

Rachitic children during infancy and early childhood are, as a rule, shorter than others. I have frequently measured such children during the third year who were six inches below the average for that age. The effect of malnutrition upon the length of the body is much less than on the weight.

GROWTH OF THE EXTREMITIES AS COMPARED WITH THE TRUNK.

At birth the trunk is relatively long and the extremities short. Subsequently the growth of the extremities is much more rapid than that of the trunk. Thus I have found at birth the length of the lower extremities (measuring from the anterior superior spine of the ilium to the sole of the foot) to be forty-three per cent of the length of the body; at five years, fifty-four per cent, and at sixteen years sixty per cent. The above figures are from one hundred and fifty observations, which, although not numerous enough for exact percentages, are still sufficient to give a very good idea of the general relation of the length of the extremities to that of the body as a whole.

THE HEAD.

Circumference.—The average circumference of the head at birth in four hundred and forty-six full-term infants observed at the Sloane Maternity Hospital and New York Infant Asylum was as follows:

Average circumference of the head,	231 males. .	13.90 inches	(35.5 cm.);
" " " "	215 females.	13.52 "	(34.5 ");
Total........................	446 infants.	13.71 "	(35.0 ").

The occipito-frontal measurement was the one taken.

The growth of the head is most rapid during the first year, the increase being about four inches (10 cm.). During the second year the increase is about one inch (2.5 cm.). From the second to the fifth year the growth is slower, being only about one and a half inches (4 cm.) for the three years. After the fifth year the increase in the circumference of the head is very slow (see table).

Closure of the Sutures.—The main sutures of the cranium are not commonly ossified before the end of the sixth month, and very frequently some mobility may be detected at the end of the ninth month. Distinct separation of the cranial bones after birth is abnormal. It is most frequently seen in premature and in syphilitic infants.

Closure of the Fontanels.—The posterior fontanel is usually obliterated by the end of the second month. The anterior fontanel under normal conditions closes on an average at about the eighteenth month. The usual variations are between the fourteenth and twenty-second months. At the end of the first year the fontanel is generally about one inch in diameter. An open fontanel at the end of the second year may be considered abnormal. The closure of the fontanel is not always early in well-nourished children, nor is it always delayed in those suffering from malnutrition. In very rare cases the anterior fontanel may either be closed at birth or may close during the first few weeks of life. Closure of the fontanel by the middle of the first year is often seen in cases of arrested cerebral development. This indicates a serious condition, usually microcephalus. Closure of the fontanel in the early months of the second year may be due to the slow growth of the brain in a child suffering from general malnutrition but otherwise normal.

In children with very large heads who exhibit no sign of rickets the fontanel is occasionally found open beyond the age of two years. By far the most frequent cause of delayed closure of the fontanel is rickets, in which condition it may be open up to the end of the third year. A large fontanel is one of the striking features of cretinism, and in untreated cases is often seen as late as the eighth year or later. In infancy an open fontanel with a marked enlargement of the head should at once suggest hydrocephalus. There is an hereditary condition in which the fontanel remains open even to adult life. Two such cases in father and son were shown me by Marie in Paris. In both there was also lack of union between the two portions of the clavicle.

Shape of the Head.—The deformity which results from compression during labour usually disappears by the end of the first month. During the first year the head often becomes flattened at the occiput in consequence of the child's lying too much upon the back. This is easily remedied by changing its position. A slight obliquity of the head may result from a habitual position during nursing or sleep. A marked degree of obliquity is sometimes congenital, but usually disappears by the fifth or sixth year.

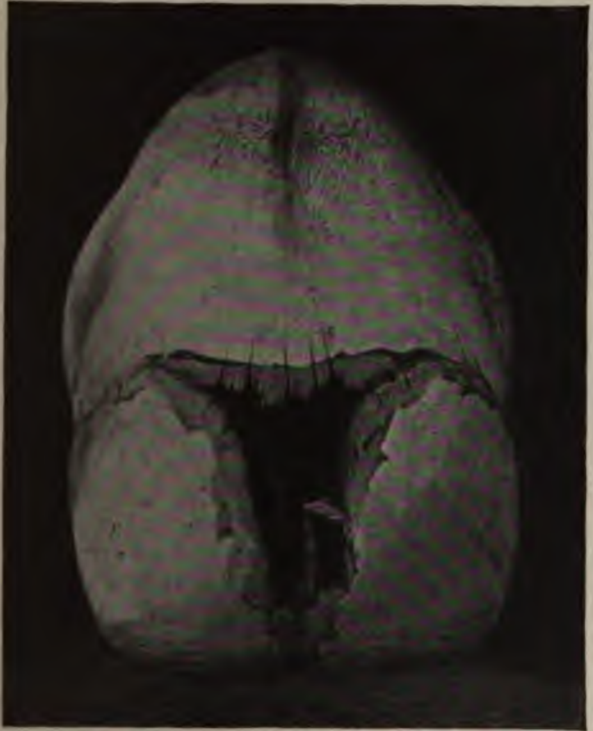

FIG. 7.—Premature ossification of the sagittal suture. Death at six weeks.

The other abnormities in the shape of the head are chiefly due to rickets and hydrocephalus, more rarely to congenital malformations of the brain. They will be considered in the chapter devoted to these topics.

Premature ossification of the sutures of the cranium occasionally gives ise to a very striking deformity of the head. I have seen two cases f such deformity from premature ossification of the sagittal suture. The heads in both cases were very narrow and long in the antero-posterior diameter. The forehead was narrow, prominent, and slightly projecting. The illustration on the previous page shows the skull of one of these cases. There is a complete obliteration of the sagittal suture. In this case there was a wide separation of the sutures at the junction of the parietal and temporal bones.

THE CHEST.

The figures showing the circumference of the chest at the different periods of childhood are given on page 20. The measurements up to and including five years are from personal observations, those from the sixth to the sixteenth are taken from Porter, and are drawn from observations on 31,371 school children. The measurement of the chest is that taken midway between full inspiration and expiration, and at the level of the nipples.

In the newly-born child the antero-posterior and the transverse diameters of the chest are nearly the same. As age advances, the transverse diameter increases very much more rapidly, so that the outline of the chest gradually assumes an elliptical shape, which it maintains during childhood.

At birth, the circumference of the chest is about one half inch less than that of the head, but throughout infancy the two measurements are nearly the same. It is not until the third year that the average circumference of the chest exceeds that of the head. According to Uffelmann, the circumference of the head and the chest are the same until the twenty-first month in a robust child, and until two and a half years in an average child. The chest measurement in infants is always much modified by the amount of fat; but, after making due allowance for this, a large chest always indicates a robust child and a small chest a delicate one. If at any age the circumference of the child's chest is found to be below the average, means should be taken, by gymnastics and otherwise, to develop it.

Deformities of the thorax result chiefly from rickets, sometimes from empyema, emphysema, and cardiac disease; in older children, from lateral curvature of the spine, or from Pott's disease. A peculiar deformity, usually congenital, but sometimes rachitic, is the funnel-shaped chest, the *Trichter-brust* of the Germans. It consists in a deep pit-like central depression at the lower end of the sternum. It is usually permanent.

THE ABDOMEN.

Throughout infancy the circumference of the abdomen is, as a rule, about the same as that of the chest. At the end of the second year the measurements of the head, chest, and abdomen are very often identical; after this time the chest measurement increases much more rapidly than the other two. Marked enlargement of the abdomen is seen in many varieties of chronic intestinal disorders. It is, however, most marked in the tympanites which so constantly accompanies rickets.

MUSCULAR DEVELOPMENT.

The first voluntary movements are usually in the fourth month, when the infant deliberately attempts to grasp some object placed before it. During the fourth month, as a rule, the head can be held erect when the trunk is supported. In many infants this is possible in the early part of the third month. At seven months a healthy child is usually able to sit erect and support the trunk for several minutes.

In the ninth or tenth month are usually seen the first attempts to bear the weight upon the feet. At ten or eleven months a child stands with slight assistance. The first attempts at walking are commonly seen in the twelfth or thirteenth month. The average age at which children walk freely alone has been, in my experience, the fourteenth or fifteenth month. Quite wide variations are seen in healthy children. Very much depends upon the surroundings. I have known infants to walk at ten months and many others not until seventeen or eighteen months, although showing no evidences of disease, and although their development had not been retarded by previous illness. A very marked difference is seen in different families of children with respect to the time of walking.

The physician is often consulted because of backward muscular development, most frequently because the child is late in walking. General malnutrition, or any other severe or prolonged illness, may postpone for several months this or any of the other functions mentioned. When there is no such explanation of the backwardness, a child who does not hold up its head, sit alone, or make efforts to stand or walk at the proper time, should be submitted to a careful examination for a cerebral or spinal paralysis, but especially for rickets which is the most frequent explanation of the symptoms.

Contrivances for teaching infants to walk are unnecessary, and their effect may even be injurious. An infant should be allowed the greatest possible freedom in the use of its limbs. It should not be restrained from walking when inclined to do so, nor continually urged to walk when no voluntary attempts are made. Nothing short of mechanical restraint will prevent a healthy child from walking or standing when it is strong enough to do so.

DEVELOPMENT OF THE SPECIAL SENSES.*

Sight.—The newly-born infant avoids the light. Its pupils contract in a light room, and if a bright light is brought before the eyes they close. During the first few weeks the infant indicates by every sign that excessive light is unpleasant. As early as the sixth day the eyes will sometimes follow a light in the room, and the child may even turn the head for this purpose. The muscles of the eyes of the newly-born infant act irregularly and not in harmony. Co-ordinate action for general purposes is not established until about the end of the third month. Even after this time inco-ordinate action is occasionally seen. The eyelids also move irregularly, and are often partly separated during sleep. The cornea is but slightly sensitive during the first weeks. In Preyer's child it was not until the third month that the lids closed when the water in the bath touched the lashes or the cornea. The recognition of objects seen is usually evident in the sixth month.

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* For many of the facts in this paragraph I am indebted to Preyer's The Senses and the Will, American edition, 1888, D. Appleton & Co.

It is important that the room in which the newly-born child is placed should be darkened, and that for the first few weeks the eyes should be protected against strong light.

Hearing.—For the first twenty-four hours after birth infants are deaf. This deafness sometimes persists for several days. It is believed to be due to absence of air from the middle ear and to swelling of the mucous membrane which lines the tympanum. With the movements of respiration, air gradually finds its way into the middle ear, and the swelling subsides during the first few days. After this the hearing gradually improves, and during the early months of life it is very acute. The child starts at the slamming of a door, and even moderately loud noises will waken it from sleep. By the end of the second month it will sometimes turn its head in the direction from which the sound comes, and by the end of the third month this will usually be done. Demme found, in observations upon one hundred and fifty infants, that the voices of parents were recognised on an average at three and a half months.

Not only are the ears unusually sensitive to sound in infancy, but the impression produced upon the brain is often marked—very loud sounds causing great fright, and sometimes even, it is reported, convulsions.

Touch.—Tactile sensibility is present at birth, but is not highly developed except in the lips and tongue, where it is very acute for the obvious necessity of sucking. After the third month it is fairly acute over the surface of the body generally. Two especially sensitive areas, according to Preyer, are the forehead and external auditory meatus.

Sensibility to painful impressions is present in early infancy, but very dull as compared with later childhood.

Temperature is also distinguished. This recognition is especially acute in the tongue. A young infant is often seen to refuse to take the bottle because the milk is only a few degrees too cold or too warm.

The localization of sensory impressions comes later, probably not much before the middle of the sixth month, and is very imperfect throughout the first year.

Taste.—This is highly developed, even from birth. According to the experiments of Kussmaul, the ability to distinguish sweet, sour and bitter, exists in the newly-born child—sweet exciting sucking movements, and bitter, grimaces. A young infant detects with surprising accuracy the slightest variation in the taste of its food, and the smallest difference is often enough to cause it to refuse its bottle altogether. Sweet substances are always easily administered, and in combination with sirups even very bitter substances can be given; but to aromatic powders and elixirs he usually objects.

Smell.—Observations upon the sense of smell in newly-born infants are few and not altogether conclusive. Kroner's experiments appear to show that smell is present in the newly born. It has been noted to be especially acute in infants born blind. The sense of smell is developed much later than the other senses. Detection of fine differences in odours is not acquired until quite late in childhood.

SPEECH.

There is a very wide variation in children with reference to the time of development of the function of speech. Girls, as a rule, talk from two to four months earlier than boys. Towards the end of the first year the average child begins with the words "papa," "mamma." By the end of the second year it is able to put words together in short sentences of two or three words. Progress in speech from this time is very rapid, each month showing great improvement. Names of persons are commonly first acquired, then the names of objects. Next to this the verbs are learned, and then adverbs and adjectives. Conjunctions, prepositions, and articles follow in order, and last of all the personal pronouns.

If a child of two years makes no attempt to speak, some mental defect may usually be inferred.

DENTITION.

The teeth are enclosed at birth in dental sacs which are situated in the gums. Superficially they are covered by the submucous connective tissue and the mucous membrane; the dental sacs rest in depressions in the alveolar process of the jaw. The tooth grows in length mainly as the result of the calcification of its roots, and being thus fixed below, it pushes upward towards the mucous membrane. This growth undoubtedly goes on steadily from birth until the tooth pierces the gum.

The deciduous or milk teeth are twenty in number. The time at which they appear is subject to considerable variation even under normal conditions. The following is the order and the average time of appearance of the different teeth:

(1) Two lower central incisors	6 to 9 months.
(2) Four upper incisors	8 " 12 "
(3) Two lower lateral incisors and four anterior molars.	12 " 15 "
(4) Four canines.	18 " 24 "
(5) Four posterior molars	24 " 30 "
At 1 year a child should have	6 teeth.
At 1½ " " " "	12 "
At 2 years " " "	16 "
At 2½ " " " "	20 "

Quite wide variations on both sides of the average are common, and are not always easy of explanation. In many cases it seems to be a family idiosyncrasy, since in the different members of a family the teeth are apt to appear at about the same time. I know one family in which no less than three members of three successive generations were born with teeth, and in most of the other members the first teeth appeared in the third or fourth month. The order in which the teeth appear is much more regular than the time of their appearance. Slight variations are exceedingly common, but marked irregularities in the order of the appearance of the teeth are the rule in idiotic children or those suffering from slighter mental defects.

The teeth may pierce the gum without any local manifestations. Very frequently, however, just before a tooth comes through there is noticed a moderate swelling and redness of the mucous membrane of the gum overlying it, and to a slight degree this may affect the general mucous membrane of the mouth. This condition may be accompanied by a little fretfulness and increased salivation, or both of these may be entirely wanting. These symptoms usually disappear when the tooth has pierced the gum. The symptoms of difficult dentition will be discussed in connection with Diseases of the Mouth.

Infants may be born with teeth; this is, however, an exceedingly rare occurrence. It is almost invariably one of the lower central incisors that is present. In case this interferes with nursing, or if it is very loosely attached to the gum, it should be extracted, but under other circumstances it should be allowed to remain, since, if it is removed, a second tooth is not likely to appear in its place in the first set. It is not at all uncommon for the first teeth to appear in the fourth month. Such teeth, in my experience, do not usually differ in character from those appearing later, unless they are in children who are syphilitic. Syphilitic children are rather prone to early dentition, and under such circumstances rapid and early decay is likely to take place. Nursing infants are, as a rule, a little earlier in their dentition than those artificially fed.

Delayed dentition is usually due to rickets. However, in many healthy infants no teeth appear before the tenth month; and I have occasionally seen the first ones at thirteen months in those who seemed perfectly healthy and showed no other evidence of rickets. On the other hand, it is by no means invariable that dentition is late in rachitic children. The latest dentition is seen in cases of cretinism. In such children it is not rare for the first teeth to appear as late as the eighteenth month. I have seen one child two years old with but two teeth. As a rule, dentition and ossification of the bones of the head go on in a corresponding manner; where one is early the other is likely to be rapid, and conversely.

Provided an infant is well nourished and thrives properly for the first six or eight months, the eruption of the teeth is likely to go on steadily after this time, even though the child may later have chronic indigestion or suffer from extreme malnutrition from any cause excepting rickets. If, however, the symptoms of malnutrition date from birth, dentition is almost invariably delayed. It is often a matter of very great surprise to see children who are markedly emaciated as a result of chronic indigestion or ileo-colitis and yet go on cutting their teeth regularly. I once had under my care a delicate infant of sixteen months, whose body length was twenty-eight inches and whose weight was less than nineteen pounds —almost exactly what they had been eight months previously—and yet he had thirteen good teeth.

Eruption of the Permanent Teeth.—The first to appear are the first molars, which usually come in the sixth year, and hence the name six-year-old molars, which is applied to them. These appear posterior to the second molars of the first set. The following table from Forchheimer gives the average time of the appearance of the second teeth:

First molars	6 years.
Incisors	7 to 8 "
Bicuspids	9 " 10 "
Canines	12 " 14 "
Second molars	12 " 15 "
Third molars	17 " 25 "

The incisors and canines replace the corresponding teeth of the first set. The eight bicuspids take the place of the eight molars of the first set. The molars of the permanent set appear back of the bicuspids, room being made for them by the growth of the jaw. As they grow and push upward they cause atrophy of the roots of the first teeth, and gradually cut off their blood supply, so that they loosen and fall out.

The place of dentition as an etiological factor in the diseases of infancy will be considered in the chapter on Difficult Dentition.

CHAPTER III.

PECULIARITIES OF DISEASE IN CHILDREN.

IN many particulars disease in children differs from that of later life. These differences relate to etiology, pathology, symptomatology, diagnosis, and prognosis. The greatest contrast to adult life is presented by infancy and early childhood. After seven years, children in their diseases resemble adults more than they do infants.

ETIOLOGY.

1. **Inheritance** is an important factor. The disease most frequently transmitted directly is syphilis. Occasionally tuberculosis and other infectious diseases have been conveyed directly from the mother to the child. In cases where no distinct disease is transmitted, children may inherit from parents constitutional tendencies, or a diathesis which may manifest itself in infancy, or in some cases not until later childhood. Under this head we may place the influence of rheumatism, gout, the various neuroses, and possibly alcoholism and insanity. In consequence of these conditions in parents, the child may inherit no definite disease, but simply a vitiated constitution.

2. **Malformations** must be considered, particularly in the first two years of life. The most important of these, from a medical standpoint, are those of the heart, brain, and kidney. The various malformations of the mouth, nose, bladder, rectum, and genital organs belong more particularly to the domain of surgery.

3. **The Diseases or Accidents Connected with Birth.**—Some of these are distinctly traumatic, like the meningeal hæmorrhages. A very large class are the infectious processes in the newly born. Infection usually takes place through the umbilical wound, more rarely through the skin or mucous membranes. This class includes pyæmia, with its varied lesions in the brain, lungs, and serous membranes, erysipelas, ophthalmia, and tetanus. In the class of infectious diseases may also be included many of the varieties of pulmonary and intestinal diseases in the newly born, and probably also some of the hæmorrhagic affections.

4. **Conditions Interfering with Proper Growth and Development.**—These are among the largest etiological factors in the diseases of infancy. They are improper food or feeding, unhygienic surroundings, and neglect.

These may cause specific diseases, like rickets or scurvy, or may lead to a condition of general malnutrition or marasmus. In this way they become most important predisposing factors, in infancy, to the acute diseases of the gastro-enteric tract, and later in childhood, to functional nervous diseases.

5. **Infection.**—This has already been mentioned as an important factor in diseases of the newly born. The number of diseases in later life directly traceable to this is very large, and is constantly increasing. Under this head should be included not only the well-known classes of infectious and contagious diseases, but also a very large number of varieties of infection which as yet have not been differentiated, and the nature of which is but imperfectly understood.

SYMPTOMATOLOGY AND DIAGNOSIS.

In older children the symptoms of disease are very much the same as in adults, and similar methods of examination may be employed. What is really peculiar to children belongs especially to the first three years of life, before speech has developed. During this period the chief and almost the sole reliance of the physician must be upon the objective signs of the disease. It is not so much that diseases in early life are peculiar, as that the patients themselves are peculiar.

Two fundamental facts are always to be kept in mind: First, that the common pathological processes are comparatively few, being chiefly of the gastro-enteric tract, the lungs, and the brain, but that the variations in clinical types are almost endless; the second is, that in infants, on account of the susceptibility of the nervous system, functional derangements are often accompanied by very grave symptoms, and may even prove fatal in twelve or twenty-four hours, or there may be speedy and complete recovery after very alarming symptoms. In many of these cases the symptoms are so indefinite that an exact diagnosis is impossible during life, and even the autopsy may throw but little light upon them.

At the bedside it is of great assistance to the physician if he can keep in mind the most frequent forms of acute disease that are likely to be met with. In the first group, including those which are very common, may be placed acute indigestion and ileo-colitis, bronchitis, pneumonia, pharyngitis, and tonsillitis; in the second group, including those which are not quite so common, may be placed otitis and the acute infectious diseases—measles, scarlet fever, diphtheria, influenza, and malaria; in the third group, including the rarer forms of acute disease—meningitis, tuberculosis, rheumatism, and diseases of the kidneys. Under all circumstances, the season, and the nature of the prevailing epidemic, if one exists, are to be considered.

In the examination of a sick infant quite a different method is to be followed from that pursued with adults. Much information is to be gained from a history carefully taken from an intelligent mother or nurse, and much more from a close observation of the child, whether asleep or awake, quiet or crying.

The History.—In view of the fact that but little information can be had from the patient, none at all in most cases, it is important to obtain from the mother or nurse as full and complete information as possible. A good history carefully obtained from an intelligent mother or nurse, puts the physician in possession of a fund of information about the patient which is of the greatest value, not only in arriving at a diagnosis in the illness for which he is consulted, but is exceedingly helpful in the future management of the child. He may thus know the individual peculiarities and special pathological tendencies. The laity attach great importance, and justly so, to advice from the physician who "knows the child's constitution." Such a history should be taken at the first opportunity after the physician is placed in charge of a child, and with note book in hand, or half its value will be lost.

Family History.—This should begin with the parents, going farther back, if possible, in many cases of hereditary disease. One must know regarding tuberculosis, syphilis, rheumatism, or alcoholism, the general vigour of constitution and physical condition of both father and mother. Health during pregnancy and previous miscarriages are important facts in the mother's history. One should know the number of other children living and their general health, the number dead and from what causes. A knowledge of the surroundings in which the child has lived may be necessary to appreciate the chances of exposure to tuberculosis, malaria, and many other forms of infection.

Patient's Previous History.—This should begin with birth. One should inquire whether the child was premature or born at term, regarding the character of the labour, whether natural or instrumental, tedious or complicated, the condition and vigour of the child at birth, primary respirations, early convulsions, and the nutrition during the early days. Next the methods of feeding should be taken up—how long entirely and how long partly breast fed, the date of weaning and the form of artificial feeding then employed. If the patient is an infant, and the problem presented is one of its nutrition, all the reliable data relating to the feeding should be obtained, even to the minutest detail. This may be wearisome and consume time, but in no other way can one appreciate the conditions present. The best idea of the child's growth and development may be obtained from a weight record if one has been kept. If not available, one must depend upon general statements as to how the child thrived at different periods. The date of the appearance of the first teeth and the time and the order in which the teeth came, are significant. The general muscular development may be best determined by learning when the child could first hold the head erect, sit alone upon the floor, bear the weight upon the feet, creep or walk alone; the mental development, by learning as to early recognition of mother or nurse, knowing the bottle, understanding the meaning of words, speaking in words or sentences. The muscular and mental development of a normal child during the first two years is a subject with which the physician should be familiar if he would detect early those differences, often slight at this age, in children whose development is backward owing to cerebral lesions.

All previous attacks of acute illness of whatever character should be noted, particularly the infectious diseases—measles, scarlet fever, diphtheria, pertussis, and influenza—with dates and details as to duration, severity, and complications. One should learn whether the child is especially prone to disorders of digestion or those of the respiratory system. Under the former head are included early difficulties in feeding, acute attacks of indigestion, diarrhœa, or dysentery, also chronic disturbances of the stomach or bowels; under the latter head, frequent catarrhal colds, earache or otitis, catarrhal croup, bronchitis, pneumonia, or pleurisy. Other points to be investigated relate to attacks of tonsillitis, operations for the removal of hypertrophied tonsils or adenoids, and previous disorders of the nervous system. In infants, particularly important are extreme restlessness, insomnia, convulsions, attacks of night terrors; in those who are older, hysterical manifestations, epilepsy, or chorea. Finally, one should know the date of successful vaccination. Inquiry should also be made concerning any recent exposure to infection in the community, school, or home.

Present Illness.—One should first note the chief complaints as stated by mother or nurse. It is important to obtain as definite statements as possible as to the time when the child was quite well, and whether the onset of the illness was abrupt or gradual, and with what particular symptoms. In all digestive disorders one should know exactly concerning the child's food at the time of the onset, its quantity, character, and preparation; also any recent change in diet, the presence or absence of vomiting, and the condition of the bowels, whether loose or constipated, the frequency and character of the stools. General questions as to whether the bowels are regular or the stools normal are of no value, since the informant often is not capable of judging correctly.

Nervous symptoms, like the others, should be elicited in response to direct questions regarding sleep, restlessness, moaning, crying out, or other evidences of pain, excitement, delirium, or convulsions, or unnatural drowsiness. In any acute illness other important symptoms are fever, sweating, dyspnœa, cough, hoarseness, nasal discharge, and the amount and composition of the urine.

The Examination.—With infants, quite a different method should be followed from that pursued with adults. It may well begin with:

General Inspection.—What is learned in this way will depend almost entirely upon the acuteness of observation of the physician, but much that is of value can be ascertained before the clothing is removed for the physical examination by simply watching the patient, whether asleep or awake, for several minutes. In acute disease, the following points should be noted especially:

1. Nutrition and general development: whether the child is well nourished or the features pinched and wasted.

2. The facial expression: whether it is bright and intelligent or dull and stupid, peaceful or anxious, quiet or disturbed, and whether the features are contracted from time to time, as if from pain.

3. The character of the respiration: whether it is rapid or slow, easy or difficult; whether there is nasal obstruction, as indicated by snoring and mouth-breathing, suggesting in infants acute rhinitis, syphilis, or retro-pharyngeal abscess; in older children, diphtheria, scarlet fever, or adenoids. Marked dyspnœa is usually accompanied by active dilatation of the alæ nasi.

4. The posture: whether the child lies upon the back, side, or face; whether the head is drawn back with general flexion of the extremities as in meningitis.

5. The nervous condition: whether the child is restless, excitable, or drowsy and apathetic; if asleep, the nature of the sleep should be observed.

6. The color of the skin of the face: whether pale or cyanotic; and the lips, whether fissured or excoriated.

7. The amount of prostration: a practised eye can usually tell with older children whether the condition is grave or not, but infants not infrequently deceive even the most experienced observer.

8. The cry: in conditions of restlessness or irritability, much information may be obtained from its character. It is important, but not always easy, to determine whether a child cries from fright, as at the approach of a stranger, from nervousness or bad training, from general irritability which may come from any acute disease, or from actual pain. The cry of fright is usually evident, because it comes with the physician's approach and ceases when he goes away. Children of highly neurotic parents and those who have been much indulged and badly trained will often cry when anything out of the usual routine occurs. The cry of pain may be very distinctive; it may be sharp and acute and accompanied by some attempt at localization, as when a child puts his hand to an inflamed part, but in infancy the pain of acute inflammation is often indicated only by general restlessness and irritability. This is frequently true of acute otitis. The cry of pain is usually accompanied by contraction of the features and other evidences of distress.

The cry of some diseases is quite characteristic, as the short, catchy cry of acute pneumonia or bronchitis; the hoarse cry of laryngitis, whether catarrhal, membranous, or syphilitic; the feeble whine of extreme exhaustion or marasmus; the moaning cry of intestinal disease; and the sharp cry of a child with scurvy whenever its bed or body is touched.

Measurements.—These, though of greatest value in chronic diseases, particularly disturbances of nutrition, may be of assistance also in acute conditions. The important measurements are the circumference of the head, chest, and body length. The circumference of the abdomen is at times important, but varies so much with the degree of distention that it is not significant as to the general development. The measurements and weight furnish reliable data which are not only of assistance in the diagnosis of existing disease, but if recorded are useful for future comparison.

In taking the circumference of the head the largest measurement (over the occipital and frontal eminences) is preferable. The measurement of the chest is usually taken over the nipples. The body length of infants is best taken with a tape as the child lies upon his back upon a table or a firm bed. For older children, a special measuring stick is convenient.

To estimate properly the significance of measurements they should be compared with the normal averages and with each other. It should be remembered that the head is normally larger than the chest until near the end of the second year; after this time, with a normal development, the chest should be larger. Any great disproportion between the size of the head and chest is suggestive of disease. The large head and the small chest belong especially to rickets. The measurements form important means of recognizing early such abnormalities as cretinism and achondroplasia, the variations often being marked before the other symptoms are prominent. One who forms the habit of taking regular measurements soon appreciates the variations from the normal, and gains great assistance from these data. Such a record made from year to year in children whose development is in any way abnormal is of great value in indicating what should be done in the way of exercise to correct faulty conditions.

Vital Signs—pulse, respiration, and temperature.—The significance of these signs is not to be measured by adult standards, since the susceptible nervous system of infants and very young children greatly exaggerates their reaction to all forms of acute infection.

The rate, regularity, quality, and tension of the pulse should be noted. In young children, the rate of the pulse is of less importance than its force and quality. A slow, irregular pulse is always significant, and should suggest meningitis or brain tumor; an irregular pulse, when rapid, has no special significance. The pulse rate is much increased from slight disturbances; the approach of the stranger or the examination by the physician may cause it to rise 20 or 30 beats. In acute disease, a pulse rate of 150 is common, and 170 or 180 is often seen where other symptoms are not particularly severe.

The rate, depth, and rhythm of respiration should be noted. The last often cannot be determined except by attentively watching the child for several minutes. In premature and very young infants a rather marked irregularity may be seen, often approaching the Cheyne-Stokes type. It is not to be taken as indicating a cerebral lesion, but seems rather to be due to the fact that the respiratory centre is not yet fully able to control the movements. Respiration of this type is seen only during the first weeks of life. Irregularity of rhythm at other times should suggest cerebral disease, usually meningitis. The respiration rate is proportionately greater in infants than in adults. In acute diseases of the lungs it not infrequently rises to 70 or 80, and occasionally it may be over 100 a minute. The rate is generally in proportion to the extent of the pulmonary lesion.

The temperature of infants and very young children should be taken in the rectum, since groin or axillary temperatures are untrustworthy and those in the mouth difficult to obtain. Immediately after birth the temperature of the child is about the same as that of the mother, or a little higher. It falls from 1° to 3° F. in the course of the first few hours. Soon it again rises to 98.5° or 99° F.

From a large number of personal observations upon healthy infants, I have found that the rectal temperature under normal conditions varies between 98° and 99.5° F.; occasionally the range may be as wide as 97.5° to 100.5° F. in apparently perfect health. The heat-regulating centre in the brain acts only imperfectly in the young infant, and slight causes are enough to disturb the temperature.

The temperature in infants is always higher than from corresponding causes in adults. Moreover, very high temperatures may be met with in cases not serious, and not infrequently when no explanation can be found even after thorough examination. In such cases the temperature seldom remains at a high point for more than a few hours. It is a continuous high temperature rather than a single rise which is significant of disease in infancy. Nothing is more perplexing to the young practitioner than the frequency with which a high temperature is seen in infants in cases of comparatively mild illness.

It is common in chronic wasting diseases, in delicate infants and in those prematurely born, to find the temperature one or two degrees below the normal; 95° and 96° F. are of almost daily occurrence in hospitals, and much lower ones are not rare. Daily observations should be made with the thermometer in such conditions, just as in fever.

Puzzling and apparently alarming temperatures are seen in infants as a result of the application of artificial heat. In one of my patients, an infant two days old, a temperature of 107° F. was caused by the close proximity of two large hot-water bags placed in the baby's basket. The younger and feebler the child the more readily are such temperatures produced.

Muscular and Mental Development.—The general muscular development is determined by seeing how well the child can hold up its head, sit alone, stand, or walk; the mental development in young infants by the intelligence of expression, the manner in which they respond to stimuli, the recognition of objects, fright at strangers, etc.; later in the first year, by the use of their hands, their understanding of speech, and their ability to pronounce words.

Local Examination.—For the purpose of making a complete routine examination of an infant the entire clothing, with the exception of the napkin, should be removed, and the infant placed preferably upon the nurse's lap upon a blanket. With older children the clothing may be removed and the body examined, one part at a time, but with all children it is essential that the examination be complete. A warm room is indispensable, and a table covered with a blanket in many respects better than the nurse's lap, although the latter has usually to be employed. The local examination should be deliberate, the physician should proceed cautiously, winning the child by gradual approaches, and avoiding excitement, force, or anything which may cause pain.

Skin.—The skin should first be inspected for eruptions, and it is important that the entire eruption be examined in order that the distribution as well as the character of the lesion may be seen. It should be noted also whether the skin is dry or moist. Marked wrinkling or loss of elasticity of the skin is one of the best indications of loss in weight. Bedsores are more frequently seen over the occiput than over the sacrum, and any large veins should be noted.

External glands should now be examined, especially the cervical, axillary, inguinal, and epitrochlear. The cause of a marked enlargement of any of these groups should be sought in the skin or mucous membranes with which they are connected. Marked swelling of the cervical glands may indicate early diphtheria, scarlet fever, or a simple acute inflammation dependent upon a rhino-pharyngitis. Enlargement of the epitrochlear glands is especially significant of syphilis. General enlargement of all the glands to a slight degree is seen in most cases of malnutrition and in many acute infectious diseases.

Head.—One should first note whether the sutures are ossified, unnaturally open, or separated; also whether the fontanel is closed or, if open, whether it is depressed or bulging. Prominences of the frontal or parietal regions when symmetrical are indicative of rickets. Irregular prominences of a smaller size, when present, are usually syphilitic. In the newly born, a tumour on the head, if in the median line, may indicate an encephalocele; if limited to either the parietal or occipital bone it is usually a cephalhæmatoma.

Eyes.—The condition of the conjunctiva and lids should be noted, also the presence of ptosis, strabismus, or other paralysis, but particularly the condition of the pupils, whether contracted or dilated, and the nature of their response to light. One should look also for the presence of corneal ulcers or the interstitial keratitis so frequent in late hereditary syphilis.

Ears.—The presence of a discharge may be recognised by sight or by the odour. In any acute febrile condition one should look for tenderness or swelling over the ear or mastoid.

Nose.—The presence of any nasal discharge should be noted and its character determined. An abundant discharge tinged with blood, in young infants, should suggest syphilis; in older children, diphtheria; a chronic discharge, adenoid growths; a purulent discharge of one side, a foreign body.

Mouth.—The appearance of the mucous membrane of the mouth and gums as well as the teeth may often be ascertained by watching the child while it is crying. It should be noted whether the tongue is dry or moist, clean or coated; whether thrush is present or any other form of stomatitis. If the gums are congested, swollen, or hæmorrhagic, they should suggest scurvy. The number, position, and character of the teeth are important. The general colour of the mucous membrane may be significant in cases of cyanosis in congenital cardiac disease, and extreme pallor of the mucous membrane in anæmia. On the mucous membrane of the hard palate may often be found the first local evidence of scarlet fever in the form of a minute punctate eruption, and on that portion of the cheeks opposite the molar teeth should be sought Koplik's sign, the earliest reliable symptom of measles.

Throat.—A careful examination of the pharynx and tonsils should never be omitted in any acute illness, no matter what other symptoms may be present. Not only tonsillitis, but often diphtheria is overlooked from a failure to observe this as an invariable rule. A good light is essential, and one must train himself to take in all the appearances at a single glance. Marked general redness of the pharynx may be associated with scarlet fever, influenza, or simple acute pharyngitis. If other symptoms are present pointing to chronic nasal obstruction or to a catarrhal process of the rhino-pharynx, a digital examination should be made to determine the presence of adenoids. Dyspnœa with mouth-breathing when associated with difficulty in swallowing should, in an infant, always suggest retropharyngeal abscess. The examination of the mouth and throat may wisely be made the last step, since it usually disturbs a child so as to embarrass further investigation.

Neck.—One should consider the position in which the head is held and the amount of rigidity of the cervical muscles. Opisthotonus may be associated with meningitis or old cerebral palsy. A marked rigidity may indicate cervical Pott's disease or, if accompanied by torticollis, may be of rheumatic origin.

Chest.—In young children particular importance should be attached to the shape of the chest. Symmetrical deformities are usually due to rickets. Contraction of one side only is most frequently the result of an old empyema or an extensive interstitial pneumonia. Bulging of the precordial region is frequent in cardiac disease. One should notice also the recession of the soft parts—intercostal spaces, the suprasternal notch, or the epigastrium; the amount of this is usually the best means of judging the severity of obstructive dyspnœa. Details regarding the physical examination of the lungs are discussed in the introductory chapter to pulmonary diseases.

Heart.—It should be remembered that under two years old loud murmurs are almost invariably of congenital origin, that soft murmurs at the base are very frequently due to anæmia, and that acquired cardiac disease is rare until after three years. For further details in the examination the reader is referred to the chapters upon diseases of the heart.

Abdomen.—There should be noted the presence or absence of tympanites or abdominal tenderness, whether general or localized, and the existence of retraction of the abdominal walls as in meningitis. The size and position of the liver and spleen are best determined by palpation. The lower border of the liver is usually slightly below the free border of the ribs. If the spleen can be easily felt below the ribs, it is, as a rule, enlarged. If it can not be felt in a satisfactory examination, it is not sufficiently enlarged to be of any diagnostic importance. In acute disease a large spleen suggests malaria, typhoid, or tuberculosis; in chronic disease, malaria, syphilis, leukæmia, or anæmia.

Spine.—The most frequent spinal curves seen in infancy are those due to muscular weakness. These disappear by placing the child in a prone position. Rachitic curves are of the same general character, but as they have usually lasted a longer time the spine is less flexible and the curve may not entirely disappear by change of posture. An angular deformity or even marked rigidity of the spine should suggest Pott's disease.

Extremities.—The colour of the skin and the character of the peripheral circulation should be noted especially in pneumonia, diphtheria, and other diseases attended by weakened heart. Clubbing of the fingers or toes may be due to congenital heart disease or to chronic disease of the lungs. Desquamation of the palms or soles may indicate hereditary syphilis or scarlet fever, even though no other evidence may be present. The finger-nails may give valuable information in hereditary syphilis. In examining the extremities one should note especially the presence of tenderness, flaccidity, or rigidity of muscles, whether the limbs are wasted or plump, and the degree of muscular power; also any abnormal swelling on the shaft or near the extremities of the bones, and, finally, the function of the joints. A general relaxation of the ligaments is common in rickets and paralytic conditions. Flabbiness of the muscles belongs to malnutrition and rickets; rigidity, if chronic, is usually indicative of cerebral palsy. Weakness of special groups, with atrophy and flaccid muscles, suggests poliomyelitis. Acute tenderness of the legs in infants should suggest scurvy. Rachitic deformities are almost invariably bilateral. Tuberculous bone disease affects the epiphyses, while syphilis usually involves the shafts, the only exception to this being the epiphyseal separation which may occur in the first months of life.

The reflexes may be somewhat difficult to obtain in infants and when exaggerated may indicate cerebral palsy, meningitis, or, as in tetany, only an extreme irritability of the nervous centres without organic disease. The plantar reflex of Babinski has little significance in infants, and in older children it is present in many conditions. Kernig's sign is a form of muscular spasm almost invariably present in cerebro-spinal meningitis, but often seen in other diseases.

Genital Organs.—Male children should be examined to determine the presence of phimosis or of undescended testicles. Hydrocele of the cord is a frequent condition, and may be mistaken for hernia. Both inguinal and umbilical hernia are very common. In female children it should be remembered that preputial adhesions may be considered normal, and are seldom the cause of the nervous symptoms attributed to them. Every vaginal discharge is significant, and if purulent should be examined bacteriologically. The great frequency of gonococcus infections is not appreciated, and they may be found when least expected.

The examination is not complete without the inspection of the *stools,* the chemical and microscopical examination of the *urine,* and an examination of the *blood.* All are more fully considered in special chapters.

PATHOLOGY.

The pathological processes which result from intra-uterine disease and those which are connected with delivery are peculiar to early life. They have already been referred to in the section on etiology. Of the processes of early life which begin after birth, the first in frequency are those of the mucous membranes resulting from the various forms of infection. In summer, it is the stomach and intestines which suffer chiefly; in winter, the respiratory tract.

The serous membranes are rarely the seat of primary inflammation. The pleura is seldom the seat of primary disease, but very often involved secondarily to disease of the lung itself. Affections of the pericardium and peritonæum are quite rare. Meningitis is fairly common both in the simple and the tuberculous form.

Diseases of the lymph nodes (lymphatic glands) play an important part in connection with the acute diseases of the mucous membranes, with many affections of the skin and even of the viscera. Acute infection tends to excite suppurative inflammation, particularly in infants; a less active process leads to chronic hyperplasia in the mesenteric, mediastinal, and cervical glands, in the tonsils, adenoid tissue of the pharynx, etc. The lymph nodes in the neck and thorax are frequently the earliest seat of tuberculous deposits, and in very many cases they are the foci from which secondary infection of the lungs, brain, or joints may occur.

Of the visceral inflammations* those of the lungs are the most com-

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* The following table gives in a general way a very good idea of the relative frequency of diseases of the different organs in infancy. It is based upon seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum, extending over a period of eight years during my connection with that institution. More than one half of the autopsies I made personally. Of these children seventy-two per cent were under one year, twenty-five per cent between one and two years, and only three per cent were over two years. The institution does not receive infants under one month, hence the absence of lesions peculiar to the newly born:

Table showing principal lesions in seven hundred and twenty-six consecutive autopsies in the New York Infant Asylum.

Lungs:		
Pneumonia—	Primary	139
	Complicating other acute infectious diseases	112
	Complicating other conditions	71
	Noted to be present in	322
Pleurisy—	No case uncomplicated with disease of lungs.	
	Empyema	5
	Serous pleurisy	1
	Dry pleurisy in nearly all the severe cases of pneumonia.	
Atelectasis (congenital)		6
Pulmonary abscess (always with pneumonia)		7
Pulmonary gangrene (always with pneumonia)		2
Pulmonary tuberculosis		56
Mouth:		
Noma		1
Peritonæum:		
Acute peritonitis (localized 2, with acute pneumonia and pleurisy 2)		4
Kidneys:		
Acute nephritis (complicating scarlet fever 4, diphtheria 1, pneumonia 4, measles 1, pertussis 1, ileo-colitis 2, pyonephrosis 1, apparently primary 5)		19
Malformations of the kidney		7

mon, it being rare to find the lungs normal at autopsy after any acute infectious disease which has lasted a week. Up to the third or fourth year of life the heart usually escapes. In older children it may be involved, as in adults, in the rheumatic diseases. The liver and spleen are not often the seat of organic disease in early life, nor is serious disease of the kidney likely to be met with excepting in connection with scarlet fever. Organic disease of the brain itself is rare, as is also organic disease of the spinal cord, with the exception of poliomyelitis. Chronic diseases of the different viscera are decidedly rare, except when resulting from acute processes. Diseases of the bones and joints are common, and of extreme importance. They are usually of tuberculous, less frequently of syphilitic, origin. Diseases of the blood are quite common, but as yet but little understood. New growths are rare. The parts most frequently the seat are the kidney and the bones. Disorders of nutrition are extremely common and of great importance, particularly rickets and scurvy.

PROGNOSIS AND INFANT MORTALITY.

The younger the patient the worse the prognosis in all the diseases of childhood. This is in consequence of the feeble resistance of the infantile organism to all diseases, particularly those which are of an acute nature. On the other hand, the rapid metabolism of childhood makes it possible for many conditions of an organic nature to disappear with time, or, as the phrase is, to be "outgrown," provided the patient can be so placed that the general nutrition can be carried to the highest point.

The accompanying chart (Plate I) shows the mortality of New York city by months during the three years from 1890 to 1892, inclusive,

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Stomach and Intestines:	
Acute ileo-colitis, with or without gastritis	116
Acute gastritis (without intestinal lesions)	None
Acute diarrhœal disease (without gross lesions)	72
Intussusception	1
Heart:	
Pericarditis (all with acute pneumonia)	3
Congenital malformations	3
Acute or chronic endocarditis	None
Brain:	
Acute, simple, or purulent meningitis (7 with pneumonia, 2 cerebro-spinal)	14
Tuberculous meningitis	11
Acute encephalitis	1
Chronic pachymeningitis	5
Chronic simple meningitis	1
Chronic hydrocephalus	3

There were twenty-six deaths from marasmus without gross lesions.

PLATE I.

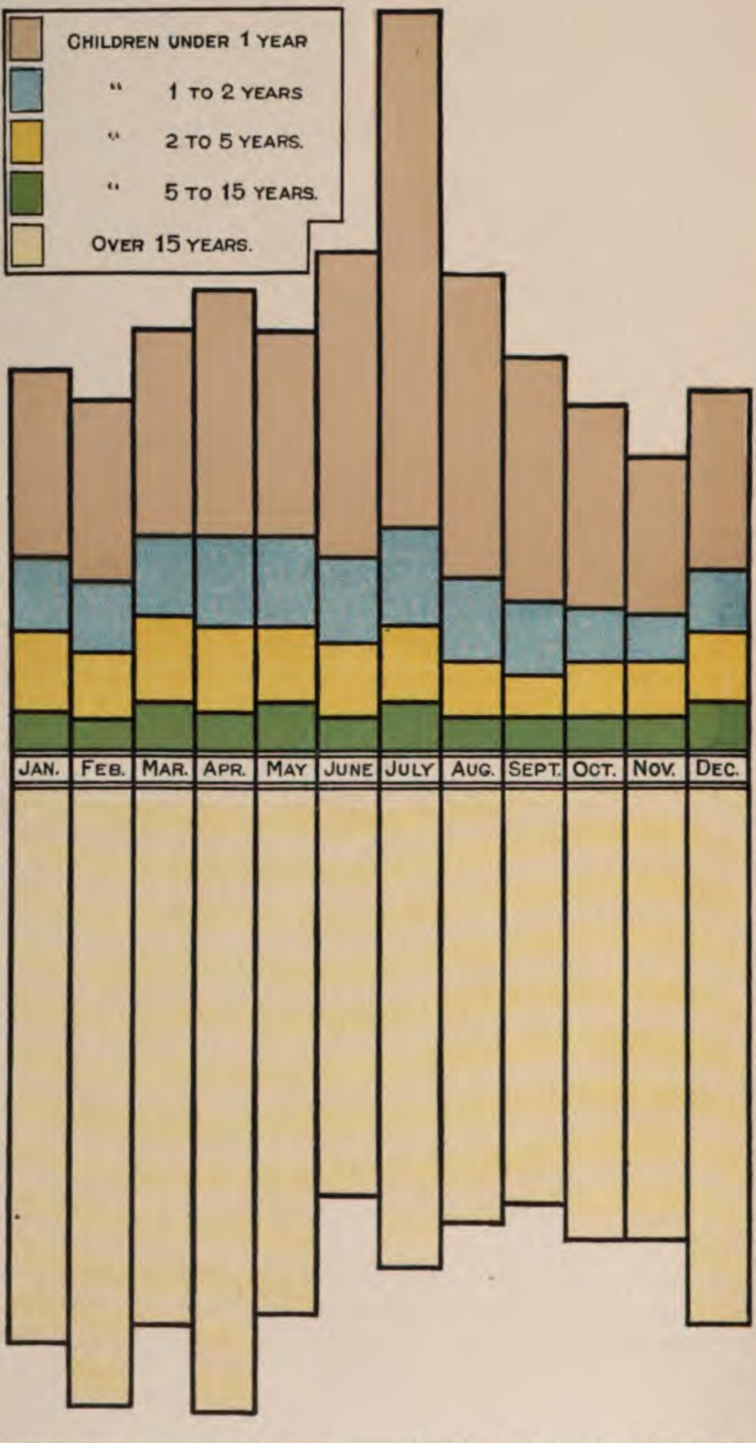

Chart showing by months the mortality of New York city for the different ages for three years. (Scale, 1 in. = 2,200 deaths.)

representing a total mortality of 128,136. The following table gives for comparison similar figures for the years 1898 to 1900:

Deaths—New York City.

1890-1892.		1898-1900.
Under 1 year......	32,916 = 26 per cent.	29,326 = 24 per cent.
1 to 2 years......	10,547 = 8 "	9,012 = 7 "
2 " 5 "	9,794 = 7 "	7,292 = 6 "
5 " 15 "	5,470 = 5 "	6,922 = 5 "
Over 15 years	69,409 = 54 "	71,024 = 58 "
Total.........	128,136	123,576

Thus about one-fourth of all the deaths occur during the first year of life, and nearly one-third in the first two years. The only age in which the mortality is much increased in summer is the first year.

The Most Frequent Causes of Death at Different Periods.—According to the statistics of Eröss from sixteen Continental cities, nearly ten per cent of all infants die during the first month of life. At this time the most important factor is congenital debility; other causes are asphyxia, infection, congenital malformations of the heart, intestine, or genito-urinary tract, hæmorrhages, convulsions, acute diarrhœal diseases, and pneumonia, which occurs both as a primary and a secondary lesion.

Statistics from New York and other large American cities show, for the past ten years, a gratifying reduction in infant mortality, both relative and actual. The following figures for New York are most striking:

Population, Deaths, and Death Rate under Five Years, New York City.

YEAR.	Population under 5 years.	Deaths under 5 years.	Rate per 1,000.	YEAR.	Population under 5 years.	Deaths under 5 years.	Rate per 1,000.
1891....	188,703	18,224	96·6	1896.....	216,728	16,807	77·5
1892 ...	194,214	18,684	96·2	1897.....	220,641	15,395	69·8
1893....	199,886	17,865	89·4	1898.....	224,736	15,591	69·3
1894....	205,723	17,558	85·3	1899....	229,029	14,391	62·8
1895....	212,983	18,221	85·6	1900.....	233,537	15,648	67·0

It will be noted that the actual number of deaths has decreased by 1,500, while the population under five years has increased by 55,000, and the death rate has fallen 30 per 1,000.

Several causes have united to bring about this result, among which may be mentioned: a wider diffusion of knowledge in the matter of infant feeding and hygiene; the fact that a larger number of infants than ever before are now sent into the country in summer; that all infants are looked after with greater care during the summer, many agencies being at work to improve their condition. Not least important of these is a bettering of the milk supply and the furnishing of pure milk, gratis, from different centres, together with a general adoption during hot weather of some form of milk sterilization—a practice well-nigh universal in the tenement districts. Antitoxin has reduced the death rate among older children. We find among rich and poor alike the largest number of deaths in the first year from disease of the gastro-enteric tract and marasmus. In the second rank are acute diseases of the respiratory **tract.** All other causes of mortality fall far below these two. Of the acute infectious diseases pertussis takes the first place, with measles second; while tuberculosis ranks first of the chronic infections. Although rarely the cause of death, rickets is a very important factor in increasing the mortality of other diseases.

During the second year the diseases of the gastro-enteric tract are still a large factor in the death rate, also the acute diseases of the lungs and the acute infectious diseases, especially measles, diphtheria, and pertussis. Deaths from scarlet fever are much less numerous. General tuberculosis and tuberculous meningitis are frequent.

From the second to the fifth year the deaths are mainly from acute infectious diseases—chiefly diphtheria and scarlet fever—much less frequently from measles or pertussis. In the next group come the acute diseases of the lungs, general tuberculosis, and tuberculous meningitis.

From the fifth to the fifteenth year the mortality in childhood is remarkably small, diphtheria and scarlet fever being still in the front rank in point of frequency. Next come the acute diseases of the lungs, simple as well as tuberculous meningitis, diseases of the bones, appendicitis, rheumatism, and cardiac disease.

Sudden Death.—This is not a very uncommon occurrence in infants who are apparently healthy. They are sometimes found dead in bed under circumstances in which grave suspicion may unjustly rest upon the attendants. This usually happens with those who are delicate or suffering from malnutrition, especially in institutions where sudden death is by no means rare. The most frequent causes in infants are the following:

1. *Malformations.*—While in most cases, to be sure, malformations of a serious nature give rise to symptoms, they may be absent, or may be so slight as to be overlooked. Infants may succumb during the first few days of life from malformations of the heart, lungs, kidneys, stomach or intestines, and sometimes from diaphragmatic and umbilical hernia.

2. *Internal hæmorrhage.*—This is chiefly limited to the first two weeks of life. In the cases that have come to my notice the cause has been rupture of some subperitoneal hæmorrhage into the general abdominal cavity. Such cases are reported in the chapter upon Visceral Hæmorrhages in the Newly Born. Under these circumstances no symptoms may exist until the occurrence of collapse, with death in a few hours.

3. *Asphyxia from overlying.*—This is not very common, excepting

among the lower classes, and is most frequently due to intoxication on the part of the mother. Such infants after death present the usual lesions of death from asphyxia, but without any evidence of violence. A recent writer in the British Medical Journal states that one thousand infants die every year from this cause in the city of London alone.

4. *Asphyxia from aspiration of food into the larynx and trachea.*—This may be due to vomiting or to the regurgitation of food during sleep; in a very weak infant it may occur while awake. This is usually seen in infants who are less than a year old, and most of the reported cases have been under six months. Such children are usually delicate. There seems to be vomiting with an attempt at crying, during which the food is drawn into the air passages. In some cases, as that reported by Demme, a single large clot of milk has been found in the larynx. In others, food is found in the larynx, trachea, and large bronchi. Cases have also been reported by Partridge and Parrot, and I have myself met with at least three. The infants have generally been found dead in bed within a few hours after feeding. This accident is more likely to happen when an infant lies upon its back.

5. *Asphyxia associated with enlargement of the thymus.*—Although these cases are very imperfectly understood, they are not rare. I see two or three each year. The condition is most frequent in infancy, but is not confined to this period. When the child is suffering from some minor illness, often bronchitis, severe attacks of asphyxia, sometimes with convulsions, may unexpectedly occur and death soon follow. Or the first attack may not be fatal, and they may recur at intervals of a few hours for two or three days before death. Sometimes sudden death follows the administration of an anæsthetic, particularly chloroform. In most cases there is found a general hyperplasia of the lymphatic tissues throughout the body known as *status lymphaticus*, more fully discussed elsewhere. The fatal asphyxia has been ascribed to the pressure of the enlarged thymus upon the pneumogastric nerve, the trachea, or the heart.

6. *Atelectasis.*—In very young infants there may be no symptoms excepting malnutrition until sudden death occurs, sometimes with convulsions and sometimes without any such symptoms. (See Atelectasis.)

7. *Marasmus.*—In this class of cases sudden death is of very common occurrence. These children are often as well two or three hours before death as for several weeks. Death frequently occurs at night, the children being found dead in bed in the morning. In some of the cases the exciting cause seems to be the lowering of the temperature, while in many no exciting cause can be found; the vital spark simply goes out after burning for some time with a feeble intensity. In some of these cases the autopsy reveals atelectasis, but in many cases nothing abnormal is found, death apparently resulting from heart failure.

8. *Convulsions in children previously showing no signs of disease.*—Most of these cases are seen in children who were previously rachitic. In them the autopsy shows no lesion except those commonly associated with death from convulsions. It is extremely rare for a cerebral lesion such as hæmorrhage to produce death in this way. In some of these rachitic cases death is due to spasm of the glottis.

9. *Asphyxia in older infants and young children.*—This may result from the pressure of a retropharyngeal abscess upon the larynx or trachea, or from the rupture of such an abscess during sleep and the entrance of pus into the air passages. While in most such cases other symptoms have been present, they may be latent. A rare cause of sudden asphyxia in children from eighteen months to five years is pressure upon the pneumogastric by tuberculous bronchial nodes, or by abscesses in the posterior mediastinum connected with caries of the spine. I have seen examples of both the latter. Gibney has reported a case of sudden death from dislocation of the upper cervical vertebræ consequent upon caries.

Sudden asphyxia may follow the ulceration of tuberculous lymph nodes and the escape of cheesy masses into the trachea or primary bronchi. This usually occurs in children from two to five years old, and many cases have been reported.

10. *Death after a few hours' illness, in which the chief symptom is high temperature.*—This is quite a common occurrence. Children who are apparently well may be taken with great prostration and a high temperature, which may rise rapidly to 106° or even 107° F., with death in from six to twelve hours, sometimes preceded by convulsions. In my hospital experience I have met with many such cases. In infants, the most frequent explanation of these symptoms, as shown by autopsy, is acute congestive pneumonia; in older children it may be due to malignant scarlet fever or epidemic meningitis; however, unless these diseases are prevailing epidemically it is somewhat hazardous to make such a diagnosis.

It does not fall within the scope of this chapter to consider cases of sudden death from heart failure after diphtheria, with pleurisy with effusion, or with myocarditis. These will be discussed elsewhere.

PROPHYLAXIS.

There is no more promising field in medicine than the prevention of disease in childhood. The majority of the ailments from which children die, it is within the power of man in great measure to prevent. Prophylaxis should aim at the solution of two distinct problems: (1) The removal of the causes which interfere with the proper growth and development of children; (2) the prevention of infection. The former can come only through the education first of the profession and then the general public, in the fundamental principles of infant feeding and hygiene. This is a department which has received altogether too small a place in medical education. The latter must come through the profession, and through legislation, the purpose of which shall be more rigid quarantine, more thorough disinfection, and improved sanitation in all its departments.

THERAPEUTICS.

Treatment in the diseases of children, and particularly those of infants, is a difficult subject. Therapeutics in infancy consists in something more than a graduated dosage of drugs. Many therapeutic means which are valuable in adults are useless in children, and many others which are of little value in adults are extremely useful in children. There is no doubt of the truth of the statement that children in the past have suffered much from overzealous treatment, particularly from drug-giving. It should be a fundamental principle never to give a dose of medicine without a clear and definite indication. If this rule is followed, it is surprising to find how often medication can be dispensed with, and also, in many cases, how much better children do without drugs than with them. A second rule is equally important: never to give a nauseous dose when one that is palatable will answer the purpose equally well. This is no small matter, and one that is well worth the physician's careful attention, if he would succeed in the management of sick children. The simpler prescriptions are made, the better. As a rule, infants revolt against most of the highly seasoned sirups and elixirs which are used to disguise the taste of unpleasant doses. Bitter medicines when mixed with water, are frequently administered without the slightest difficulty.

It is a common mistake to underestimate the importance of the hygienic surroundings of the patient, the value of good nursing, careful feeding, and judicious stimulation, just as it is to overestimate the beneficial effects of drugs. In the great majority of acute ailments not serious in character, for which a physician is called, the patient recovers quite as promptly without drugs as with them. This does not mean that such children require no treatment, but that the least important part of the treatment is drug-giving, while the most important part is attention to the hygienic matters just referred to. In cases of severe illness, in infants especially, we must avoid all unnecessary medication, in order that the stomach may not be disturbed and vomiting excited. Hence the importance of relying as far as possible upon local measures of treatment. The tendency to recovery from all acute processes, while seen in adults, is even more striking in children, where, if we can but remove that which hampers the bodily functions, Nature will conduct the case to a satisfactory termination. Thus, after an attack of ordinary bronchitis of no great severity, it is often seen that the disturbance of the stomach and intestines, which can be directly traced to the drugs employed, continues long after the original disease has subsided, and is very much more difficult to relieve. In diseases of the stomach and intestines especially there is a great amount of overmedication, very much to the detriment of the patient. In all chronic disturbances of nutrition—chronic indigestion, malnutrition, and anæmia—nothing is of so much value as change of air and surroundings. This is most striking in the case of city children. With them it is a frequent experience that tonics of every description are of little or no avail, and yet immediate and most marked improvement begins when the children are sent to the country.

The tablet triturates have furnished us with a convenient method of administering many drugs to children. Those which are especially useful are: calomel, from one tenth to one half grain; gray powder in the same doses; antimony and ipecac, one one-hundredth of a grain each; phenacetine, one to two grains; arsenious acid, one one-hundredth of a grain; paregoric, ♏v; Dover's powder, one tenth of a grain; atropine, one four-hundredth to one two-hundredth of a grain. This list might be very greatly extended.

As to the method of administration, it is to be remembered that several small doses are more easily given and less likely to disturb the stomach than a few larger ones. This method of administering very many drugs to children will be found extremely satisfactory — e. g., sodium bromide, one half grain every fifteen minutes, is often better than five grains every two hours; phenacetine, one half grain every half hour, is better than two grains every two hours; calomel, one tenth of a grain every hour, is better for constipation than a single dose of two grains.

Antipyretics.—The indications for the employment of antipyretics in children are somewhat different from those in adults. It is to be borne in mind that, where the cause is similar, all temperatures in children are higher than in adults. Thus a simple pharyngitis, which in an adult causes a rise of temperature only to 100° or 101° F., is in a child not infrequently accompanied by a temperature of 104°, or even 105° F. The height of the temperature, as measured by the thermometer, is not to be taken as the only guide for the employment of antipyretics. In many cases the temperature is 104°, or even 105° F., and yet the child exhibits no signs of unusual discomfort. Such a temperature manifestly does not call for interference. Again, a temperature of 103° F. may be accompanied by very marked restlessness and other signs of distress which may be relieved by employing some antipyretic measure. The number of cases seen in practice, of high temperature apparently from trivial causes, is very great. One must not be unduly alarmed even by a very high temperature if it is of short duration. It is the continuously high temperature which indicates serious illness. Whenever the temperature is found to be much above the normal it should be carefully watched, but not interfered with until a diagnosis has been made, unless the symptoms urgently demand it; otherwise the physician may lose one of the most valuable aids to diagnosis, since it is not the height of the temperature but its course which is significant. In many cases it is very important to know whether the temperature uninfluenced by drugs is remittent, intermittent, or steadily high, and hence the advantage of waiting until a diagnosis has been made before disturbing the temperature curve. This is, of course, not admissible when the temperature is itself a source of real danger, which after all is seldom the case. Since the cause of a great many obscure temperatures is found in the stomach and intestines, it very often happens that a purgative, stomach-washing, or intestinal irrigation may be the most efficient antipyretic. In cases of moderate elevation of temperature we need go no further than cold sponging.

The most reliable antipyretic measure for infants is the use of cold. This may be employed—

(1) *As an ice cap to the head.*—In many cases of quite high temperature and restlessness in infants this alone will reduce the temperature one or two degrees and allay the nervous symptoms.

(2) *Cold sponging.*—For this purpose water about 80° to 85° F., equal parts of alcohol and water, or equal parts of vinegar and water may be employed. In the case of infants, all the clothing except the diaper should be removed and the child laid upon a blanket. The body should be sponged for from ten to twenty minutes, and then wrapped in a blanket without further dressing. Cold sponging must be very frequently employed in order to be efficient in reducing high temperature. Its great value in allaying nervous symptoms, even when the temperature is not very high, is not sufficiently appreciated. Its effect is often more satisfactory than an anodyne.

(3) *Cold pack.*—This is one of the simplest and most efficient means of reducing temperature which can be employed. The child should be stripped and laid upon a blanket. The entire trunk should then be enveloped in a small sheet wrung from water at a temperature of 100° F. Upon the outside of this, ice may now be rubbed over the entire trunk, first in front and then behind. By this method there is no shock and no fright, and any ordinary temperature can usually be readily reduced.

The rubbing with ice should be repeated in from five to thirty minutes, according to circumstances, after which the child may be rolled in the blanket upon which he is lying without the removal of the wet pack. The head should be sponged with cold water while this is being carried on, and artificial heat, if necessary, should be applied to the feet. The pack is continued from one to twenty-four hours, according to circumstances.

(4) *The cold bath.*—The child is put into a bath at a temperature of 100° F., the bath being gradually lowered by the addition of ice to 85° or 80° F. The body should be well rubbed while the child is in the bath and water should also be applied to the head. On removal from the bath, the body should be quickly dried and rolled in a warm blanket. The bath is usually continued from five to ten minutes.

(5) *Evaporation baths.*—The trunk is closely enveloped in two layers of surgeon's gauze, or some loosely woven equivalent, which is moistened from time to time with water at a temperature of 115° F., continuous evaporation being kept up by means of a hand, or better electric, fan. The evaporation bath would seem to possess some important advantages in the case of infants and young children, in that it is more efficient than sponging, involves little disturbance of the patient, and causes no shock or fright. Hot applications should constantly be made to the extremities.

(6) *Rectal irrigations.*—These are easily given, disturb the patient very little, and are effective in reducing the temperature. A double tube (see Fig. 17), the in-and-out flow of which can be readily regulated, should be employed. It is best to use at first water at a temperature of 90° F., and gradually reduce this to 70° F. The irrigation should be continued for ten to fifteen minutes, or even longer if the desired fall in temperature is not obtained, and may be repeated as often as every three hours.

Antipyretic Drugs.—Except in cases of malaria, quinine should not be employed for the reduction of temperature in children.

Of the many coal-tar derivatives employed, phenacetine has the advantage for children of being tasteless and causing little depression, but the slight disadvantage of practical insolubility. None of the drugs of this group is, however, to be employed in large doses with the sole purpose of reducing the temperature. Their great value in pædiatrics consists rather in allaying the nervous symptoms which accompany fever, and this purpose can be accomplished by the use of comparatively small doses. To an infant of one year, phenacetine or antipyrine can be given in one-grain doses every hour or two hours until the desired effect is produced. For a child of five years a dose of two grains may be given in the same manner. When used as indicated, these drugs are of very great value in making the patient more comfortable, in promoting sleep, and in allaying headache and general pains. In cases of hyperpyrexia they are, however, much less certain and less safe than the use of cold. In many cases of mild pyrexia the symptoms are relieved by the administration, either separately or in combination, of citrate of potassium, spiritus ætheris nitrosi, and liquor ammonii acetatis, in small frequent doses.

Stimulants.—In spite of the many statements to the contrary, alcoholic stimulants are well tolerated even by very young infants. Proportionately larger doses of alcohol than of many drugs may be administered to infants; still, all stimulants, and alcohol in particular, are very greatly abused in the hands of many practitioners.

The indications for the employment of stimulants are much the same in young children as in adults. They are to be used whenever the pulse is weak, soft, and compressible, and whenever the general powers of the patient are very greatly depressed. In most of the acute fevers they are not to be given early in the disease, and in many cases they are not required at all. They must often be used very sparingly while the temperature is high, but given freely as soon as it falls. In many acute febrile diseases stimulants are not called for at any period. This is especially true of most cases of lobar pneumonia. The time, however, when they are most likely to be needed is at or just after the crisis of the disease, when for twenty-four hours they should be very freely given. In broncho-pneumonia they are more often required, and their use should be begun earlier. This is particularly true of the broncho-pneumonia which develops secondarily to the infectious diseases. In all toxic diseases, such as diphtheria, alcohol should be begun as soon as depressing symptoms show themselves, and continued in doses regulated by the degree of prostration. In the acute gastro-enteric diseases the depletion is often great and there is little absorption of food; the patients may in certain cases be sustained by alcohol for several days.

Alcohol is contra-indicated in all acute febrile processes where there is high temperature, dry skin, flushed face, and a full, strong pulse. In such conditions it is often injurious.

The method of administering alcohol is of no little importance. Brandy and whisky are in most cases to be preferred to the wines, but not always. Champagne may be substituted when spirits are not well borne by the stomach. For infants under one year old, brandy should be diluted with at least eight parts of water. It is commonly given in too concentrated a form. Altogether the best method of administration is to determine the amount to be given in every twenty-four hours, have it diluted sufficiently, and then administer it in small doses at short intervals. In this way vomiting is rarely produced. The addition of brandy to the water required by the thirst makes it less likely to disturb the stomach.

The quantity of alcohol will depend very much upon circumstances. An infant one year old, for whom alcohol is indicated, should not be given to begin with more than half an ounce of brandy or whisky during the twenty-four hours, and even in bad conditions it is rarely advisable to give more than twice this quantity, except for a very short period. In children four years old double the amount may be employed in the corresponding conditions. Too much can not be said against the practice, unfortunately with many practitioners a common one, of the reckless use of alcohol in large doses in young children. I refer to such amounts as six or eight ounces daily of brandy or whisky for children of two or three years in cases of pneumonia or diphtheria. Little good and much harm is likely to follow such therapeutics.

Tonics.—Cod-liver oil stands at the head of the list of tonics for young children. It is particularly in the convalescence after acute diseases of the respiratory tract that we see its most striking benefit. It is also of very great use in anæmia, and in a large number of children who are extremely delicate. In these patients it may be advantageously administered throughout the greater part of nearly every winter season. In convalescence after attacks of gastro-enteric disease it is not nearly so useful, and often must be withheld for a long time. It is a mistake to give cod-liver oil at any time when the tongue is coated, the digestion poor, and the stomach easily disturbed. In the case of infants, as a rule, the pure oil is to be preferred to the emulsions, but this is not always the case. The administration of small doses—i. e., ten or twenty drops of the oil three times a day continued for a long period—is much better than the use of larger doses for a shorter time.

A perfect preparation of iron for use in infancy has not yet been discovered. During the first few years all astringent preparations should be avoided. To be recommended are the various peptonates, the albuminate, bitter wine, sweet wine, saccharated carbonate, pomate, and malate. These are only slightly constipating, and most of them can be given with milk. For older children nothing is better than reduced iron or Blaud's pills.

Arsenic is second only to iron in the treatment of the anæmia of children, and in very many cases it is to be preferred to iron. The tablet triturates of arsenious acid, one one-hundredth of a grain, may be given immediately after meals three times a day, or one or two drops of Fowler's solution largely diluted with water.

Alcohol is of very great value as a tonic in combination with some of the bitters, either small doses of quinine, nux vomica, or the bitter wine of iron. Usually wines, especially sherry, are to be preferred to spirits, although some children take spirits better. When combined with a bitter there is little danger of the formation of the alcoholic habit, even though its use may be long continued.

Of the bitter tonics, quinine and nux vomica are easily superior to all others.

Opiates.—Strong objections have been urged by many against the employment of opium in the diseases of infancy. While opiates have no doubt been abused, the fact remains that opium is almost as valuable a remedy in the treatment of disease during the first five years as at any other period of life. Infants are, however, peculiarly susceptible to the drug, and relatively much smaller doses are required than of most medicines. If the physician will accustom himself to the use of very small doses, he will be surprised to see how satisfactory are the effects produced.

The most useful preparations for young children are paregoric, Dover's powder, the deodorized tincture, morphine, and codeine. The following table gives what may be considered safe initial doses at the different ages:

	1 month.	3 months.	1 year.	5 years.
Paregoric	♏ i	♏ ii	♏ v to x	♏ xxx to xl
Deodorized tincture	♏ $\frac{1}{20}$	♏ $\frac{1}{10}$	♏ $\frac{1}{4}$ to $\frac{1}{2}$	♏ ii to iii
Dover's powder	Gr. $\frac{1}{20}$	Gr. $\frac{1}{10}$	Gr. $\frac{1}{4}$ to $\frac{1}{2}$	Gr. ii to iii
Morphine	Gr. $\frac{1}{1000}$	Gr. $\frac{1}{600}$	Gr. $\frac{1}{200}$	Gr. $\frac{1}{30}$ to $\frac{1}{20}$
Codeine	Gr. $\frac{1}{300}$	Gr. $\frac{1}{200}$	Gr. $\frac{1}{60}$	Gr. $\frac{1}{10}$ to $\frac{1}{8}$

Ordinarily doses like the above should not be repeated oftener than every two hours. In exceptional circumstances, as when very great pain is present, the dose may be given more frequently. In the hypodermic use of morphine it should be remembered that its effects are always more uniform and striking than when the drug is administered by the mouth, and the dose should therefore be smaller. In every instance where a full dose of opium has been given the physician should wait until the effects have subsided before the dose is repeated.

Anodynes.—Chloral is usually well borne even by quite young infants. In them it should never be administered by the mouth, but, on account of its irritant properties, always by the rectum. After rectal administration its effects are usually manifest in half an hour, and sometimes sooner. The rectal dose for an infant of one month is one grain; three months, two grains; one year, three to five grains. It may be repeated every two to four hours, according to indications. Other drugs may replace this in most diseases, but in the case of infantile convulsions nothing is so reliable as chloral.

Belladonna is well borne by children, and in larger doses than most drugs. A tolerance is quite readily established. The eruption is more readily produced than the other physiological effects, and even quite small doses may be sufficient to bring out a very abundant blush. The parents should be advised of this fact, lest undue alarm be felt.

The drugs classed as antipyretics—phenacetine, antipyrine, and antifebrine—are exceedingly valuable in the treatment of many diseases of infancy where irritative nervous symptoms are prominent. In many cases they may advantageously take the place of opium, except where pain is the principal symptom, as in otitis or pleurisy. In all conditions where spasm is a prominent symptom, whether of the larynx or bronchi, or local or general convulsions, antipyrine is especially valuable.

Drugs well borne by Children.—In this list might be mentioned belladonna, the bromides, the iodides, chloral, quinine, calomel—in fact, all mercurials—and alcohol.

The drugs not well borne include particularly cocaine and all preparations of opium. In the case of many others, while the constitutional effects are well tolerated, they must be given carefully to young infants, since they are irritants to the stomach. In this class may be mentioned the salicylates, salol, the astringent preparations of iron, and the acids.

Counter-irritants.—These are of great value in a large variety of diseases. *Blisters* should never be employed in the case of infants, and very rarely, and never needlessly, in the case of older children. In the latter they may be required in inflammations of the ear, of the joints, or of the spine; they should never be applied to the chest.

The *mustard paste* is probably the most satisfactory means of producing quick counter-irritation over a large surface. To make a mustard paste: Take one part powdered mustard and six parts of wheat flour, mix with lukewarm water, and spread between two layers of muslin. This should be removed as soon as a thorough redness of the skin has been produced—in most cases from five to eight minutes, according to the strength of the mustard employed. This may be repeated as often as every three hours, and continued for a week if necessary, without producing excoriations of the skin. For older children the paste may be made one part mustard to four parts flour. In pulmonary diseases it should be large enough to surround the chest. When it is used to produce general reaction in heart failure it should cover the entire trunk.

The mustard pack.—The child is stripped and laid upon a blanket, and the trunk is surrounded by a large towel or sheet saturated with mustard water. This is made as follows: One tablespoonful of mustard to one quart of tepid water. In this a towel is dipped, and while dripping wound around the entire body. The patient should then be rolled in the blanket. This pack may be continued for ten or fifteen minutes, at the end of which time there will usually be a very decided redness of the whole body. It may be repeated according to indications. Where it is desired to produce a general counter-irritation, the mustard pack is not quite as efficient as the mustard bath, but it has the advantage in causing much less disturbance to the patient. The mustard pack is useful in the condition of collapse or of great prostration from any cause whatever, in convulsions, and in cerebral or pulmonary congestion.

The *turpentine stupe* is made by wringing a piece of flannel out of water as hot as can be borne by the hand. Upon this is sprinkled ten or fifteen drops of the spirits of turpentine. The stupe is then applied to the body and covered with oiled silk or dry flannel. It is useful chiefly in abdominal pains or inflammations, but in infancy must be carefully watched or vesication will be produced. For continuous use it is not so valuable as the mustard paste.

Stimulating liniments containing turpentine and other irritants are useful in inflammations of the chest, although less reliable than the mustard paste. One of the mildest and most useful preparations is camphorated oil. Another is olive oil four parts and turpentine one part. These may either be rubbed upon the surface, or a piece of flannel may be saturated with them and then applied to the skin. The old-fashioned spice bag is useful in many cases where a very mild counter-irritant is desired over the abdomen.

Local blood-letting.—Leeches are often useful in arresting acute inflammations of the mastoid or middle ear. They may also be applied to the præcordium in acute pneumonia with signs of failure of the right heart, viz., great dyspnœa and cyanosis. In robust children even venesection may be employed with advantage for the above indications.

Dry cups are useful even in young infants, to relieve acute pulmonary congestion. From four to six cups may be applied, and the effect may be continued by the application of the mustard paste. Wet cups should never be used for young children.

Poultices are useful in local inflammations about the glands of the neck, the joints, and in cellulitis in various parts of the body. They are indicated in pulmonary diseases in which there is great pain, as in pleurisy or in pleuro-pneumonia. In bronchitis and in broncho-pneumonia their prolonged use is objectionable on account of their weight. Better effects can generally be produced by hot fomentations and counter-irritation. Ground flaxseed is the best material for poultices. This should be mixed with boiling water until the proper consistency is reached, when the poultice should be put into a bag of muslin. The poultice should be covered with oiled silk or flannel, so that it will retain its heat as long as possible. To be of value, poultices must be applied hot and changed frequently.

Hot fomentations are more cleanly than poultices and much more easily changed. One of the best means of applying them is by a piece of spongio-piline wrung from water as hot as the hand can bear. Where this can not be obtained, a large piece of flannel may be used in the same way, covered with cotton batting, and then with oiled silk. This method of using hot fomentations is exceedingly satisfactory for applications to the extremities.

Cold.—Cold is useful in all forms of inflammation of the eyes and brain. In inflammation of the cervical lymph glands and of the joints it is of undoubted value, but its advantage over heat is questionable. The efficiency of both cold and heat in these cases depends largely upon the method of application. Sometimes in pleurisy much greater relief is obtained from the use of an ice bag to the chest than from hot applications, but this is not the general experience. The treat the application of the ice bag to the chest has ma in my own hands it has not yielded the results admissible only in lobar pneumonia, and here chiefl children. The application of cold in young or should be made with caution in all inflammations

Cold is best applied to the head by an ice cap ordinary rubber or flannel bag filled with ice m The rubber coil filled with ice water is also an inflamed glands or joints the ice bag should be compresses changed every minute.

The Hot Pack.—All clothing is to be remov covered with towels wrung from water at a temp 110° F., after which the body should be rolled in hot applications may be changed every twenty or perspiration is produced, which may be continu This is mainly useful in uræmia.

The Hot Bath, like the mustard pack or th used to promote reaction in cases of shock or coll be put into the bath at a temperature of 100° F ally raised to 105°, or even to 110°, but rarely ab should be well rubbed while the patient is in t should be kept in the water to see that the tem high. During the bath, in most cases, cold shou

The Hot-Air or Vapour Bath.—All the clot and the patient laid upon the bed with the bed body ten or twelve inches, and sustained by m The bedclothing should be pinned tightly abo the head is outside. Beneath the bed clothing from a croup kettle or a vapourizer. This wi spiration in fifteen or twenty minutes. It may to thirty minutes at a time. Instead of vap duced in the same way. The air space about The vapour bath is applicable chiefly to cases of

The Mustard Bath.—Four or five tablespoon should be mixed for a few minutes with one g this should be added four or five gallons of pl of 100° F. The temperature of the bath may hot water to 105° or 110° F. if desired. Not the hot mustard bath for a general derivative to the surface in cases of shock, collapse, hear in sudden congestion of the lungs or brain. be continued for more than ten minutes. If

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mouth between the back teeth.
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d be employed.

but this is not the general experience. The treatment of pneumonia by the application of the ice bag to the chest has many advocates, although in my own hands it has not yielded the results claimed for it. It is admissible only in lobar pneumonia, and here chiefly in older and stronger children. The application of cold in young or very delicate children should be made with caution in all inflammations of the respiratory tract.

Cold is best applied to the head by an ice cap made like a helmet; an ordinary rubber or flannel bag filled with ice may answer the purpose. The rubber coil filled with ice water is also an excellent method. For inflamed glands or joints the ice bag should be used; for the eyes cold compresses changed every minute.

The Hot Pack.—All clothing is to be removed and the child's body covered with towels wrung from water at a temperature of from 100° to 110° F., after which the body should be rolled in a thick blanket. These hot applications may be changed every twenty or thirty minutes until free perspiration is produced, which may be continued as long as necessary. This is mainly useful in uræmia.

The Hot Bath, like the mustard pack or the mustard bath, may be used to promote reaction in cases of shock or collapse. The patient should be put into the bath at a temperature of 100° F., the water being gradually raised to 105°, or even to 110°, but rarely above this point. The body should be well rubbed while the patient is in the bath. A thermometer should be kept in the water to see that the temperature does not go too high. During the bath, in most cases, cold should be applied to the head.

The Hot-Air or Vapour Bath.—All the clothing should be removed and the patient laid upon the bed with the bedclothing raised above the body ten or twelve inches, and sustained by means of a wicker support. The bedclothing should be pinned tightly about the neck, so that only the head is outside. Beneath the bed clothing hot vapour is introduced from a croup kettle or a vapourizer. This will usually induce free perspiration in fifteen or twenty minutes. It may be continued from twenty to thirty minutes at a time. Instead of vapour, hot air may be introduced in the same way. The air space about the body is indispensable. The vapour bath is applicable chiefly to cases of uræmia.

The Mustard Bath.—Four or five tablespoonfuls of powdered mustard should be mixed for a few minutes with one gallon of tepid water. To this should be added four or five gallons of plain water at a temperature of 100° F. The temperature of the bath may be raised by the addition of hot water to 105° or 110° F. if desired. Nothing is more efficient than the hot mustard bath for a general derivative effect in bringing the blood to the surface in cases of shock, collapse, heart failure from any cause, or in sudden congestion of the lungs or brain. The bath should not usually be continued for more than ten minutes. If necessary, it may be repeated in an hour.

The Bran Bath.—Put one quart of ordinary wheat bran in a bag made of coarse muslin or cheese cloth and place this in four or five gallons of water. The bran bag should be frequently squeezed and moved about until the bath water resembles a thin porridge. It may be of any temperature desired, but usually about 90° to 95° F. is best. A bran bath is of great value in cases of eczema, excoriations about the buttocks, or in other cases where the skin is very delicate, and plain water seems to irritate it.

The Tepid Bath may be given at a temperature of 95° to 100° F. It is very useful in many conditions of excitement or extreme nervous irritability. To induce sleep it is often more efficient than drugs.

The Cold Sponge or Shower Bath should be given in the morning before breakfast, and in a warm room. The child should stand in a foot tub containing warm water enough to cover the feet, then a large sponge holding about a pint of water at a temperature of from 40° to 60° F. should be squeezed three or four times over the chest, shoulders, and spine of the child, the skin being rubbed meanwhile. The bath should not last more than half a minute. It should be followed by a brisk rubbing until a thorough reaction is established. This is very useful at all ages, but a particularly valuable tonic in delicate children. It may be used in those only eighteen months old. Not the least of the beneficial results is the full expansion of the lungs from the strong cry which the bath usually excites. In younger infants a cold plunge may be substituted. This should be merely a single dip of the entire body in water at a temperature of 50° to 60° F. In order that beneficial effects shall follow the cold plunge or cold sponging, a good reaction must be established. If children lack sufficient vitality to secure this, and if they remain pale, pinched, and blue for some time after the bath, it must be discontinued altogether, or water of a higher temperature used.

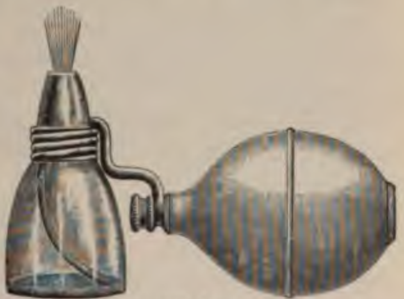

Fig. 8.—Albolene atomizer.

Nasal Spray.—This may be either of an aqueous or oily solution. For the oil spray an atomizer similar to that shown in the accompanying cut should be employed. It is valuable in cases of dry catarrh, where there is a formation of crusts in the nose. A variety of oils may be used in the spray, albolene being perhaps as satisfactory as any. Fig. 8 shows an efficient atomizer for albolene.

There are a good many forms of hand atomizers to be found in the market for the production of an aqueous spray. For a cleansing nasal spray, Dobell's solution, Seiler's solution, Listerine ten-per-cent solution, or a two-per-cent solution of boric acid may be used.

Nasal Syringing.—In cases of considerable nasal obstruction and in the more serious affections of the rhino-pharynx, only the syringe can be considered an efficient means of cleansing the cavity.

The fountain syringe has the advantage of being easily regulated as to the force employed, this being determined by the height at which the bag is suspended above the bed. For ordinary purposes an elevation of one or two feet is sufficient, and rarely is a greater pressure than three feet advisable. The last is desirable when a thorough flushing of the rhino-pharynx is required. The position of the patient is the same as that shown in Fig. 10. The danger of forcing fluid into the middle ear is greatly lessened if the patient keeps the mouth wide open.

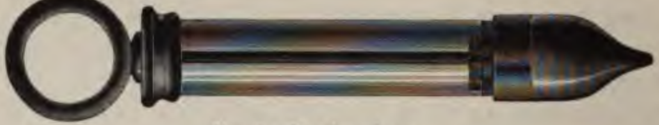

FIG. 9.—Nasal syringe.

Where a smaller amount of fluid is sufficient a piston syringe may be employed. This should be small enough to be easily worked with one hand. It should have a soft rubber tip, to prevent injuring the nasal mucous membrane, and the tip should be large enough to fill the nostril. The best piston syringe for nasal use is shown in Fig. 9. This is made either of glass or hard rubber, and fulfils all the conditions mentioned. It is easy of action, can be readily cleansed, and holds about half an ounce. The same syringe should not be used for more than one patient, unless it has been very thoroughly disinfected. In hospitals, and even in private practice, nasal syringes are frequent carriers of infection.

Either of two positions may be used in nasal syringing. In diphtheria, scarlet fever, or any constitutional disease attended by great depression, the child should not be removed from the bed. The syringing may be done by a single nurse, who stands at the head of the bed, alternately syringing the right and left nostril, turning the head from side to side (see Fig. 10). The other method is to hold the child erect on the lap, with the head inclined somewhat forward, the syringing being done by a second person standing behind. In either position the childs arms and hands should be securely pinioned to the sides by a sheet. To make sure that the rhino-pharynx has been reached the water should return through the opposite nostril or the mouth. When properly done, no prostration and very little irritation are caused. The bulb (Davison) syringe should not be employed for nasal irrigation; as the pressure can not be satisfactorily regulated, fluids are likely to be forced into the Eustachian tubes.

Syringing the mouth and pharynx is useful in many pathological conditions of these parts, particularly in children too young to gargle. Either the fountain, piston, or bulb (Davison) syringe may be used. If the pharynx is to be reached, the nozzle is used as a tongue depressor. This should be placed at the angle of the mouth between the back teeth. The child should be held in the sitting posture, with the head inclined forward. Only bland solutions should be employed.

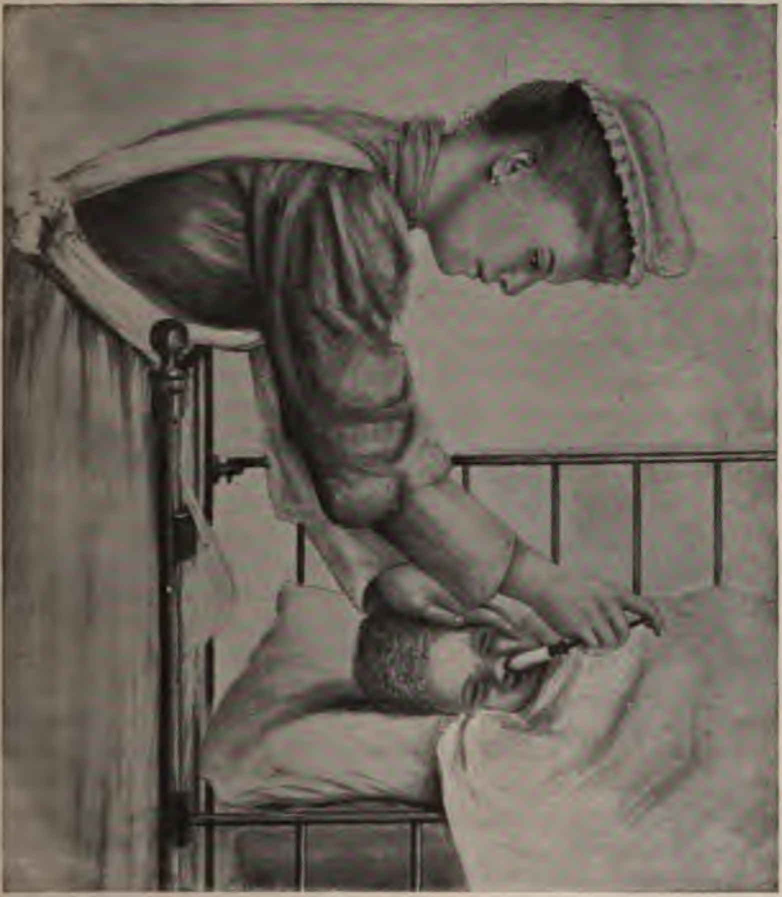

FIG. 10.—Method of syringing the nose.

Inhalations.—These are of very great utility in all affections of the respiratory tract. To be efficient, the patient should be put under a tent. A satisfactory tent may be made by erecting a T-shaped piece of wood at the head and foot of the crib and throwing over this a large sheet folded and pinned at the corners. Another method is, to stretch a cord around the top of each of the four posts of the crib, or simply from the centre of the head piece to the centre of the foot piece; the sheet should be used as in the first instance. A very good tent may be improvised by throwing a large sheet over an open umbrella. Instead of an ordinary cotton sheet one of rubber cloth may be used. For hospital use I have found it convenient to have a rubber cover made to fit closely over the top of the crib to be used for inhalations. The better the tent the more satisfactory are the results from inhalations.

Inhalations may be in the form of vapour or spray. The apparatus employed may be the croup kettle, the vapourizer, or the steam atomizer. As all of these are used with alcohol lamps, innumerable accidents from fire have occurred with them. Patients and nurses should always be cautioned regarding this. The ordinary croup kettle is a clumsy affair and especially likely to be the cause of accidents. In Fig. 11 is shown one of an improved pattern,* which possesses the advantages both of the ordinary croup kettle and of the vapourizer. The base has been weighted, to prevent the apparatus being easily upset. The pail is low, which fact also contributes to its stability. It is provided with a safety alcohol lamp, the flame of which can be regulated by a screw. The lamp holds enough alcohol to burn from five to six hours. This kettle may be used to produce simple vapour, or vapour from lime water, or a medicated vapour may be employed. If the latter is desired, the substance to be vapourized is placed on a sponge held in the expansion of the spout. The kettle should be filled with hot water before using. It should be placed upon the floor or a low box beside the crib, so that the end of the spout is just inside the tent at a level with the surface of the bed.

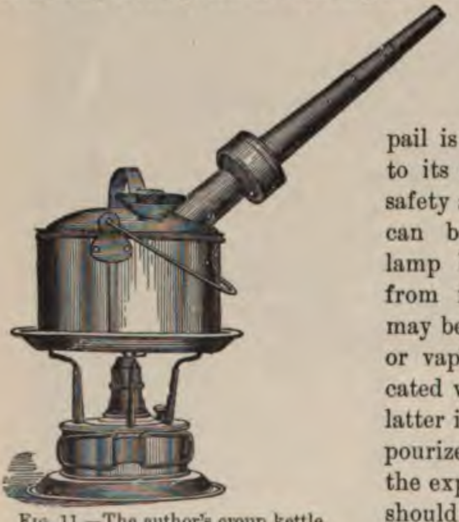
FIG. 11.—The author's croup kettle.

The vapourizer † (Fig. 12) is one of the most satisfactory means of obtaining medicated inhalations. The boiler is half filled with water, and the substance to be vapourized is placed upon a sponge which lies on a perforated diaphragm placed at the top of the boiler, so that all the steam generated in the boiler passes through it.

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* Made by Lewis & Conger, 130 W. 42d St., New York.

† Made by Whitall & Tatum, New York and Philadelphia.

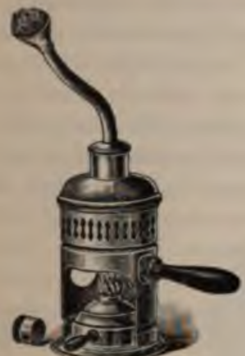

Fig. 12.—Vapourizer.

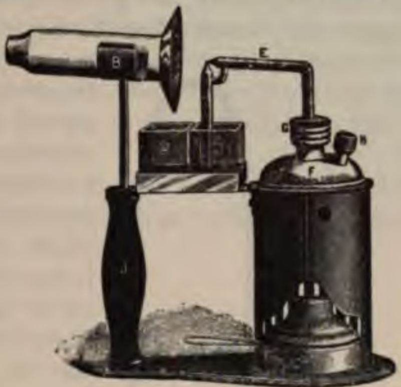

Fig. 13.—Steam atomizer.

The steam atomizer is shown in Fig. 13. For this no tent is required. It should be placed about one and a half or two feet from the patient's face, and the clothing protected by a rubber sheet. This is very efficient where steam or vapour of lime water are used, but is not to be advised for carbolic acid, creosote, etc.

Oiled-silk Jacket.—In all forms of acute pulmonary inflammation this form of local application has largely supplanted the time-honoured poultice, both in hospital and in private practice. It keeps the skin at a uniform temperature, maintains a moderate degree of counter-irritation, and gives the patient a great deal of comfort. The jacket consists of three layers—an outer one of oiled silk, an inner one of cheese cloth or light flannel, and a middle one of cotton batting or wool. The middle layer should be half an inch in thickness. The purpose of the lining is to keep the cotton in position. Fig. 14 shows the pattern of the jacket. It is generally made in two pieces, each of which should be about twelve inches wide and twelve inches long for a child of one year. These are sewed together along one border and lapped at the other, where it is secured by safety pins. A properly made jacket will last two weeks.

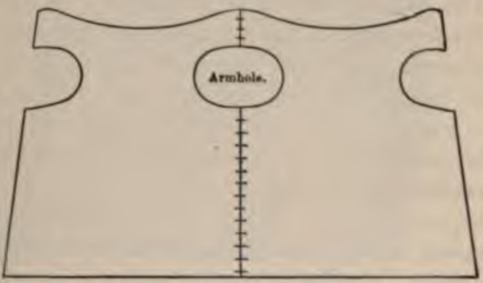

Fig. 14.—Pattern for oiled-silk jacket.

Stomach-Washing consists in the introduction of water into the stomach through a flexible catheter or stomach tube and then siphoning it out. It was introduced into general practice among infants by Epstein, of Prague. To Seibert (New York) is due the credit of bringing the subject prominently before the minds of the medical profession in America. It is one of the most valuable therapeutic measures we possess. Stomach-washing has been employed almost daily for the past twelve years in the hospitals with which I am connected, during which period the stomach has been washed many thousand times. No accident whatever has occurred, and the operation may be considered entirely free from danger; in fact, it is difficult to pass the tube anywhere else than into the œsophagus. The amount of prostration may be compared to that of an ordinary attack of vomiting.

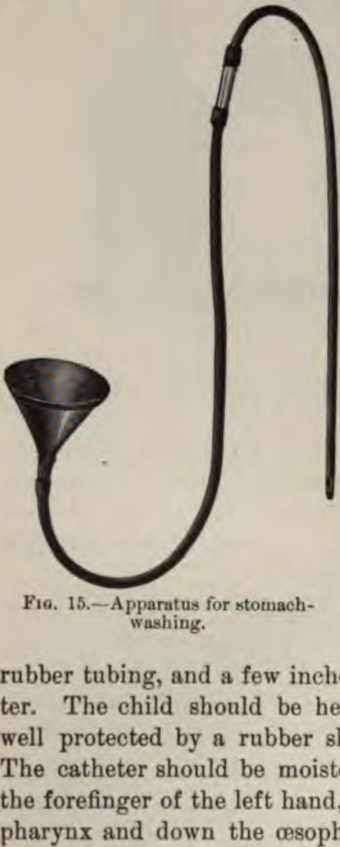

FIG. 15.—Apparatus for stomach-washing.

The apparatus for stomach-washing is very simple (Fig. 15). There is required a soft-rubber catheter, size 16, American scale (24 French)—one with a large eye is preferred; a glass funnel, holding four to six ounces; two feet of rubber tubing, and a few inches of glass tubing to join this to the catheter. The child should be held in a sitting posture (Fig. 16), the body well protected by a rubber sheet, with a large basin conveniently near. The catheter should be moistened. While the tongue is depressed with the forefinger of the left hand, the catheter is passed rapidly back into the pharynx and down the œsophagus. It is important that the first part of the introduction should be as rapid as possible, for if the child begins to gag from the pharyngeal irritation the introduction of the tube may be quite difficult. No resistance is ordinarily encountered after the tube reaches the œsophagus. About ten inches of the catheter should be passed beyond the lips. When it has reached the stomach the funnel should be raised as high as possible, to allow the escape of gases almost invariably present. It should then be lowered, in order to siphon out the fluid contents. If nothing escapes, the funnel is then to be raised and from two to six ounces of water poured into it from a pitcher; the funnel is then lowered and the water siphoned out. This procedure is repeated from four to ten times, or until the fluid comes back perfectly clear. About a quart of water is ordinarily used. Various solutions have been advised for stomach-washing, but nothing is better than boiled water, used at the temperature of from 100° to 110° F.—the higher temperature being employed when the gastric irritation is very great. Through the tube are easily discharged mucus and small curds; larger ones are gradually broken down by repeated washing. Vomiting may be induced by overdistending the stomach with water. If there is great thirst there is often an advantage in leaving one or two ounces of water in the stomach. To this water it is at times beneficial to add lime water.

Stomach-washing in its application is practically limited to children under two and a half years. It is easiest in those under eighteen months. Children of three years and over are usually so much alarmed and struggle so violently as to make it difficult and undesirable.

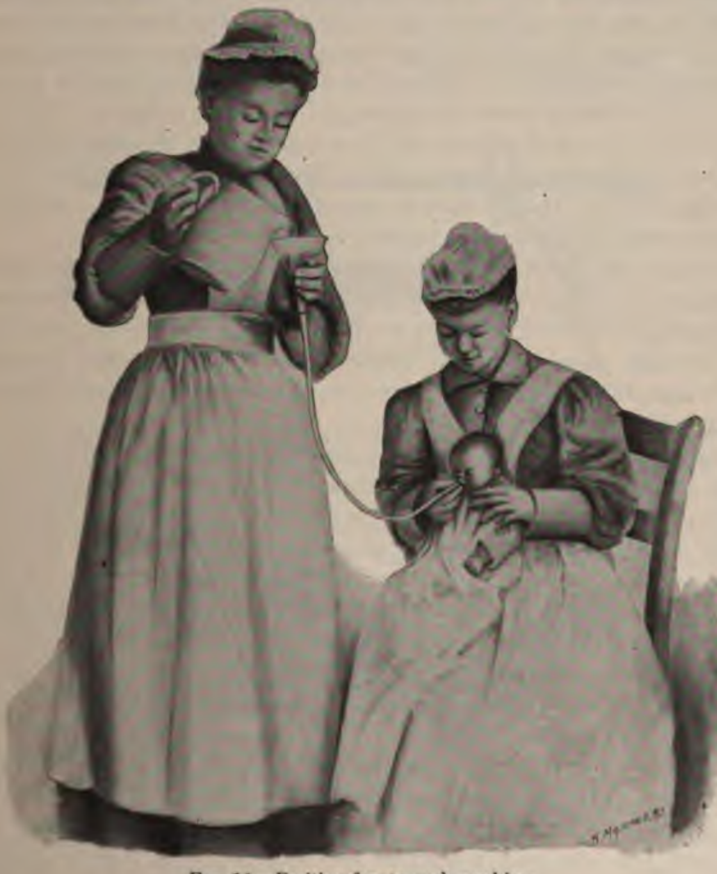

Fig. 16.—Position for stomach-washing.

The indications for stomach-washing are: (1) Acute gastric indigestion, either with or without persistent vomiting. Here the purpose is simply to clear the stomach of its irritating contents, and a single washing may be sufficient. (2) Chronic indigestion attended with great production of gastric mucus, and sometimes, though rarely, by dilatation of the stomach. In these cases daily washing is required for a considerable period. (3) Poisoning.

Gavage.—Gavage consists in the forcible introduction of food into the stomach by a tube passed through the mouth. The same apparatus is employed as in stomach-washing, and the method is similar, with the exception that for gavage the child should be placed flat upon the back, the head being steadied by an assistant. In older children a mouth-gag is often necessary. After the tube has entered the stomach the funnel should be raised to allow the gas to escape. The food is then poured into the funnel; as soon as it has disappeared the tube is tightly pinched and quickly withdrawn, to prevent food from trickling into the pharynx, since this is often a cause of vomiting. In young infants, after removing the tube, it is well to keep the jaws separated by the fingers for a few moments to prevent gagging. If the food is regurgitated this usually happens at once. It may then be introduced a second time. After feeding, the child should be kept absolutely quiet upon the back.

In cases where all the food is given by gavage the interval between feedings must be considerably longer than under other circumstances. The food given should be either wholly or partly predigested, since digestion in these cases is usually feeble. The stomach should be washed before each feeding, in order to remove mucus and to be sure that it is empty before the meal is given.

Gavage is valuable, as already indicated in connection with the incubator, in the management of premature infants and after certain operations upon the mouth and neck. It is also useful, first, in the case of very young infants, who, suffering from severe malnutrition, can not be induced to take food enough to sustain life; secondly, in many acute diseases, particularly in septic cases where the child will not readily take the necessary food, as in diphtheria, scarlet fever, typhoid, pneumonia, etc.; thirdly, in many cases of cerebral disease where food is refused on account of delirium or coma; and, fourthly, in uncontrollable vomiting, as first suggested by Kerley.

Gavage is a very simple procedure and one which a nurse can easily be taught. It is free from danger, and in a great majority of cases the food given is not regurgitated. In acute septic cases not only may food be given, but also such medicines and stimulants as may be required, with little or no trouble. The advantage of gavage over the continued coaxing or holding the nose and forcing the patient to swallow, will be at once apparent to one using it.

Nasal Feeding.—The method is similar to gavage, the only difference being that the tube is passed through the nose, and consequently a much smaller one is used. No. 10 American or No. 16 French scale is a proper size. Nasal feeding is applicable to children over two years old, in whom the tube can seldom be passed through the mouth without the use of a gag, and then only after much struggling. It is of value after intubation, tracheotomy, and other operations about the throat, also in some cases of throat paralysis, especially after diphtheria.

Irrigation of the Colon.—By irrigation of the colon is meant the flushing of the entire large intestine by fluids injected high up through a catheter or rectal tube. Very rarely indeed do the injected fluids pass beyond the ileo-cæcal valve.

The apparatus required for irrigating the colon is a fountain syringe, five or six feet of rubber tubing, and a flexible rectal tube or soft-rubber catheter—No. 26 or 27, French scale, being preferred. Kemp's double-current tube of hard or flexible rubber (Fig. 17) is of great advantage. The child is placed upon the back, with the thighs flexed and the buttocks brought to the edge of the bed or table. He should lie upon a Kelly pad or a rubber sheet so arranged as to form a trough emptying into a large basin or tub. The bag containing the water is hung four or five feet above the bed. If a catheter is used it is inserted just within the sphincter before the water is turned on. As it flows the catheter is gradually pushed upward to a distance of twelve or fourteen inches. The water distending the intestine in advance of the catheter usually makes its introduction quite easy. If, however, the attempt is made to introduce the catheter before turning on the water, it almost invariably doubles upon itself. In Fig. 18 is shown the colon of an infant of six months in position. It is the peculiar curve and the great length of the sigmoid flexure that make the introduction of water difficult, unless the tube is passed quite to the descending colon. When this is done the remainder of the colon fills with ease; but if the tube is introduced only three or four inches the irrigation is not likely to extend beyond the sigmoid flexure.

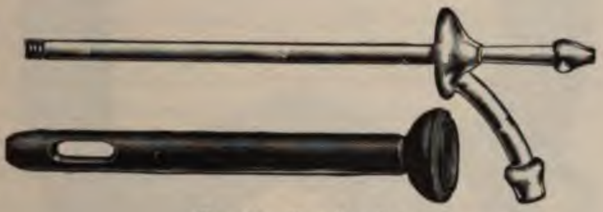

FIG. 17.—Kemp's tube.

Usually a pint, and often a quart, will be introduced before any water returns. This is an advantage, since one can then be reasonably sure that the upper part of the colon has been reached. The water is passed from time to time alongside the catheter, often with considerable force. At least a gallon of water should be used for a single irrigation. The washing should be continued until the water returns quite clean. Gentle kneading of the abdomen should be continued during the irrigation, particularly the early part of it, to facilitate the passage of the water into the upper part of the colon. At the end of the irrigation the rubber tube is detached and the water allowed to escape through the catheter, which remains *in situ*. Sometimes as much as a pint of water remains in the intestine. This is usually passed within half an hour. As the irrigation of the colon almost invariably excites active peristalsis of the lower ileum, this part of the intestine is emptied as well. It is to be remembered that the colon of an infant six months old will hold one pint without distention, and at the age of two years from two to three pints.

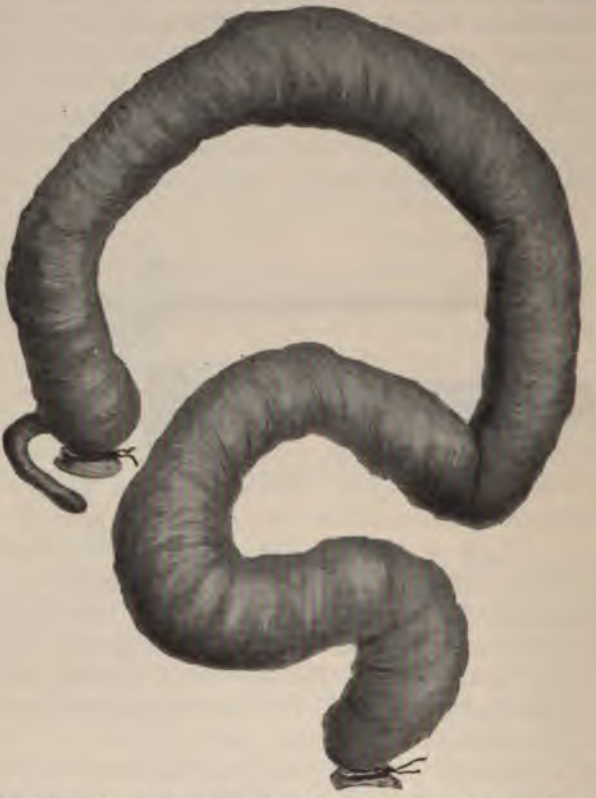

FIG. 18.—Colon of a child six months old, in position. (From a photograph.)

Irrigation of the colon is useful to clear this part of the intestine of mucus, fæcal matter, undigested food, and the products of decomposition.

It may also be employed as a means of local medication in ileo-colitis. Where the object is simply to cleanse the intestine, a saline solution—a teaspoonful of common salt to a pint of water—is preferred.

The temperature of the water used for irrigation may be varied according to the special indications. For ordinary purposes, where cleansing only is aimed at, a temperature of from 95° to 100° F. seems to be best. When the body temperature is high, or when there is much pain, tenesmus and straining, cold water has important advantages. In cases of collapse or great prostration hot injections may be employed; these should not be hotter than 110° F., but at this temperature they may be used with safety.

Irrigation under most circumstances is required only once in twenty-four hours. When it is employed it is important to use a large quantity of water. It must be done thoroughly to be of value, and either by the physician himself or an experienced nurse.

Enemata.—Simple enemata are useful in infants and older children for constipation. Where an immediate effect is desired the most efficient is one containing glycerine—e. g., for an infant, one teaspoonful to one ounce of water. Oil enemata are useful where the fæcal mass is hard and dry and expelled with difficulty. Enemata should always be given with care, and preferably a rubber catheter should be attached to the nozzle of the syringe.

Nutrient enemata have a limited application in infancy. The rectum soon becomes intolerant, and rarely can more than three or four injections be given before they cease to be retained. The quantity injected should be small, rarely more than one or two ounces, and the interval between injections should be at least four hours. In older children they may be used as in adults. For this purpose either completely peptonized milk or some of the forms of beef peptones, like Mosquera's beef jelly, may be employed. In giving stimulants in enemata care should always be taken that they be well diluted.

The administration of drugs *per rectum* is useful in certain cases where, on account of the unpleasant taste or vomiting, the administration by mouth is difficult—e. g., quinine and chloral. As a diluent, gruel is preferable to water. If quinine is used, the bisulphate is the best preparation, but this must be well diluted. The temperature of enemata which are to be retained should be about 100° F. It is necessary in infancy to press the buttocks together for half an hour afterwards to prevent the expulsion of the injection.

Hypodermic Medication.—This is not often used in childhood, but it must not be forgotten that it is at times of the greatest service even in infancy. The use of morphine hypodermically in convulsions, of morphine and atropine in cholera infantum, of strychnine in heart failure, as in pneumonia, may be cited as examples.

Massage.—In older children massage is useful for the same conditions as those for which it is employed in adults; the most important are anæmia, general malnutrition, chorea, and chronic constipation. It is necessary that in the beginning only the mildest movements of massage should be employed, and these but for a short time.

In infancy massage has a limited application, and it is doubtful whether it really does more than can be accomplished by the general friction of the body. This rubbing, either with the bare hand or with cocoa butter, or with some form of fat, is useful in malnutrition, in rickets, and in wasting diseases where the circulation is feeble and the muscular tone low. Cocoa butter is cleanly and has a pleasant odour, and is, I think, quite as valuable as the more commonly employed cod-liver oil, which is exceedingly disagreeable. The inunctions should be given daily after the morning bath, before an open fire. The rubbing should be continued for fifteen to twenty minutes.

Anæsthetics.—As a general anæsthetic for routine use, ether is to be recommended for children. Its disadvantages can largely be overcome by proper administration; in point of safety it is immeasurably superior to chloroform for the very young. The administration of ether to young children may be advantageously preceded by a few whiffs of nitrous oxide or ethyl chloride; both, however, are to be used with caution in infants. Ether should be given slowly, well diluted with air, and if used in this way its unpleasant features may be obviated. This can best be accomplished by the use of some special form of inhaler. Ether should not be selected as the anæsthetic for patients suffering from nephritis, bronchitis, pneumonia, pleurisy, or any other disease attended by obstructed respiration. For all these conditions chloroform is much safer.

The dangers from chloroform are greatest when it is given too rapidly or in too concentrated a form. Both are exceedingly likely to occur where it is administered to a struggling child. The greatest care and judgment should be exercised at such times, or disastrous consequences may follow. To produce and maintain the effect desired with the minimum amount of chloroform should always be the aim. All anæsthetics, but especially chloroform, are dangerous in children with the so-called lymphatic diathesis. For the removal of tonsils or adenoids, so often required in such children, chloroform should not be employed.

Nitrous oxide, while very useful in older children, as in adults, for momentary operations, is not well borne by infants. It produces so early and so deep asphyxia that its prolonged use may be fraught with serious danger.

Ethyl chloride is coming into use as a rapidly acting anæsthetic for momentary operations, or preliminary to the use of ether. It is powerful, and acts so quickly that it must be used with great caution in young children. Only a small amount is required.

PART II.

SECTION I.

DISEASES OF THE NEWLY BORN.

CHAPTER I.

ASPHYXIA.

THE lungs in the full-term fœtus are of a uniform dark red colour, and show very distinctly upon their surface the lobular divisions. They are firm and solid and readily sink in water. The connective tissue is very abundant, and forms distinct fibrous septa, which stretch through the lungs in every direction.

Inflation of the lungs begins with the first cry uttered by the infant as it is born into the world. The parts first expanded are the anterior borders of the lungs, then the upper lobes, and finally the lower lobes posteriorly. The superficial lobules are nearly always expanded before those in the interior of the lung. The inflation is sometimes irregular, because of the accumulation of mucus in some of the bronchial tubes. The right lung is frequently stated to be expanded earlier than the left. Although this is often the case, there is no uniformity in this respect. The important point to be remembered is, that the parts last inflated are the posterior portions of the lower lobes. The expansion of the lungs is a gradual process, and in healthy infants it is probably not complete much before the end of the second day. In delicate children it may be postponed for several days, or even weeks. The above statements are based upon post-mortem observations upon infants dying from various causes during the first weeks. It has often been a matter of great surprise to find at autopsy on an infant two or three days old, that less than one half of the lung tissue was expanded, although the child had breathed well and shown no signs of atelectasis. Under normal conditions at full term inflation of the lung takes place very readily, but not so readily in premature or delicate infants, on account of the feebleness of the respiratory muscles. The longer it is postponed after birth the more difficult does it become, on account of the changes which occur in the collapsed air vesicles. The condition of the child *in utero* may be described as one of fœtal apnœa, its oxygen being received and its carbon dioxide discharged through the placenta, which is essentially the organ of respiration at this period. This condition is interrupted by cutting off the supply of oxygen and the accumulation of carbon dioxide in the blood. Which of these is the important factor in inducing pulmonary respiration has been much debated; but the best experimental evidence seems to show that it is the want of oxygen which stimulates the respiratory centres.

Under the term "asphyxia" may be included all cases in which primary respiration is not spontaneously established with sufficient force to maintain life. Usually there is no attempt at pulmonary respiration until after the birth of the child, but it may occur *in utero* or at any stage of parturition. Asphyxia may be of intra-uterine or extra-uterine origin.

Etiology.—1. *Intra-uterine asphyxia.* The maternal causes include any disturbance of the placental circulation during labour—anything which prolongs the second stage of labour, convulsions, hæmorrhage, the use of ergot in the second stage, or, finally, the death of the mother. The causes relating to the child are pressure upon the cord, multiple winding of the cord about the neck, early separation of the placenta, and pressure upon the brain. If the respiratory stimulus comes before the birth of the child, the effort at respiration may cause the entrance into the mouth and air passages of amniotic fluid, mucus, blood, meconium, etc.

2. *Extra-uterine asphyxia.* This condition is a much less common one. It arises from causes quite apart from those above mentioned, and depends upon malformations or intra-uterine disease of the organs of respiration, circulation, or of the brain. It may be secondary to an injury of any of these organs received during parturition. It is also seen in premature infants, where it depends upon the feeble development of the nerve centres and respiratory muscles and upon the soft, yielding chest walls.

Lesions.—In infants dying of intra-uterine asphyxia there are seen the usual changes found in death from suffocation, together with the effects of attempts at breathing *in utero.* There is general congestion of all the viscera, particularly of the brain and its meninges, the liver, and the lungs. They may show small, punctate hæmorrhages, and occasionally large extravasations. Blood or bloody serum may be found in any of the serous cavities. The right heart is overdistended with dark, soft clots, and the blood generally is more fluid than normal. The lungs may contain no air, but more frequently there are small, scattered areas in which lobular inflation has taken place. If the child has lived several hours there are larger areas of expanded lung, especially in the upper lobes, and these may even be emphysematous, if artificial inflation has been employed. In the mouth, nose, larnyx, and even as far as the finest bronchi, there may be found aspirated materials—amniotic fluid, blood, mucus, or meconium. In extra-uterine asphyxia there are organic changes in the viscera—malformations of the lungs or the heart, intra-uterine pneumonia or pleuritic effusion, malformation of the diaphragm and sometimes of the brain.

Symptoms.—Under normal conditions the newly-born infant begins at once to scream and to use its limbs, the purplish colour of the skin giving place in a few moments to a rosy pink. In the first degree of asphyxia—*asphyxia livida*—the child is deeply cyanosed. Either no attempt whatever is made at respiration, or it is superficial and repeated only at long intervals. The pulse is slow, full, and strong. The vessels of the cord are distended. Muscular tone is preserved, and also cutaneous irritability, so that with the application of almost any kind of external stimulus, respiration is excited and the symptoms disappear.

In the second degree—*asphyxia pallida*—the picture is quite a different one. The face is pale and death-like, though the lips may still be blue. The heart's action is weak, and by palpation can rarely be felt at all. By auscultation the sounds are feeble, irregular, and usually slow. The cord is soft, pale, and flaccid, and its vessels nearly empty. The sphincters are relaxed, and meconium oozes from the anus. There is entire loss of tone in the voluntary muscles, so that the extremities and entire body seem perfectly limp. Cutaneous sensibility is abolished. The extremities are often cold. There may occur a few short, convulsive contractions of the respiratory muscles, but these are without effect and soon cease. Unless such cases receive the most prompt and efficient treatment, the heart's action becomes more and more feeble until it ceases and death occurs. Other cases are partly resuscitated and may survive for a few hours or days, when they gradually sink, respiration becoming more and more feeble in spite of all efforts to maintain it. Between these two extremes all degrees of severity are seen.

In extra-uterine asphyxia there may be some attempts at voluntary respiration continuing for several hours, sometimes for a day or two, but this may be inadequate to sustain life.

Diagnosis.—Almost the only condition with which asphyxia is likely to be confounded is cerebral compression from a meningeal hæmorrhage. The difficulties in the case are much increased by the fact that the two conditions are not infrequently associated. It may then be impossible to tell that in addition to asphyxia, intracranial hæmorrhage is present. If the hæmorrhage is extensive and the asphyxia only moderate, a diagnosis is possible in most of the cases. In hæmorrhage there is often a history of undue compression during delivery—sometimes the use of forceps. The fontanel is bulging; there is coma, and there may be paralysis. The respiratory murmur may be quite strong for several hours, but it gradually fails as the child becomes completely comatose. Anæmia resulting from a large hæmorrhage, like that due to rupture of the cord, may simulate the severe form of asphyxia.

Prognosis.—This depends upon the grade of asphyxia and the treatment employed. There is but little tendency to spontaneous recovery in any form. In the milder cases recovery is almost invariable with any intelligent treatment. In the severest cases the outcome is always doubtful, although by persistent effort many that are apparently hopeless may be saved. In a prognosis as to the ultimate result, the frequent complication of asphyxia with meningeal hæmorrhage should always be kept in mind. Apart from this complication it is doubtful whether asphyxia has anything to do with the production of idiocy.

Treatment.—In every case the first step is to clear the mouth and pharynx of mucus by means of the finger covered with absorbent cotton. In the milder forms respiration is usually excited either by spanking the child or the alternate use of hot and cold baths. If the hot bath is employed, the water should be from 105° to 110° F. and always tested by a thermometer. After a few moments the child may be dipped into ice-water, or the body may be douched with it. In the livid cases relief is often afforded by allowing the cord to bleed for a few moments before ligation. The loss of half an ounce of blood is ordinarily sufficient. Simply swinging the child in the air is a powerful stimulus to respiration. The above means will suffice in the great majority of cases. In the more severe forms, however, these are inadequate. There is no response whatever to external stimulation, either by heat or mechanical irritation. In these cases two methods of resuscitation may be employed: artificial respiration and direct inflation of the lungs.

One of the most widely employed methods of inducing artificial respiration is that of Schultze. The infant is grasped by both axillæ in such a way that the thumbs of the physician rest upon the anterior surface of the chest, the index fingers in the axillæ, and the remaining fingers extending across the back. The child is thus suspended at arm's length between the knees of the physician, the feet downward and the face anterior. The body is now swung forward and upward, until the physician's arms are nearly horizontal. This produces the inspiratory effort. When this point is reached, an arrest in the swinging causes flexion of the trunk, the head now being directed downward, the lower extremities fall toward the physician until the whole weight of the body rests upon the thumbs. In this way expiration is produced. Lusk cautions against the employment of this method if the heart's action is very feeble, as it may cause the heart to stop altogether.

A method introduced by Dew has been extensively employed in New York. The infant is grasped in such a way that the neck rests between the thumb and forefinger of the left hand, the head being allowed to fall far backward, the upper portion of the back resting upon the palm of the hand; with the right hand the knees are grasped between the thumb and fingers, the thighs resting against the palm of the hand. Inspiration is produced by depressing the pelvis and lower extremities thus causing the abdominal organs to drag upon the diaphragm, and at the same time gently bending the dorsal region of the spine backward. In expiration the movement is reversed, the head being brought forward and flexed upon the thorax, while at the same time the thighs are flexed so as to bring them against the abdomen. The body is thus alternately folded upon itself and unfolded as the movements are carried on. If there is much mucus in the mouth, the movement of expiration should first be made with the body completely inverted. This method is simple, efficient, and much less fatiguing than that of Schultze when it is to be maintained for a long time. It is also of great advantage in that it can be carried on while the child is in the hot bath, one of the greatest objections to the method of Schultze being the loss of animal heat incident to its use.

In all cases where artificial respiration is used the first movement should be that of expiration, to expel, so far as possible, foreign substances from the air passages. The movements should be made from eight to twelve times a minute, and not too forcibly, the child being kept in the hot bath between the movements, and as much as possible during them. As long as the heart beats resuscitation is possible, and the case should not be abandoned.

Inflation of the lungs is not usually of so much general value, although it is sometimes successful when all other means have failed. It may be done by the mouth-to-mouth method, or by the introduction of a catheter into the larnyx. The former is much easier, but is much less certain, since the air is liable to pass into the stomach. If, however, the head be carried pretty well backward, compression made over the epigastrium, and the nose closed, this is less likely to occur. The introduction of a flexible catheter into the larynx is by no means an easy matter even with considerable practice. The use of a stiff catheter is not so difficult, but it is capable of doing harm. A much better instrument is the laryngeal tube of Ribemont (Fig. 19). This is inserted like an intubation tube. By means of the rubber bag attached, air may be forced into the lung, or mucus aspirated from the trachea and bronchi as may be desired. In all these methods, but especially when the catheter is used, care is necessary not to employ too much force. It should always be remembered that the ca-

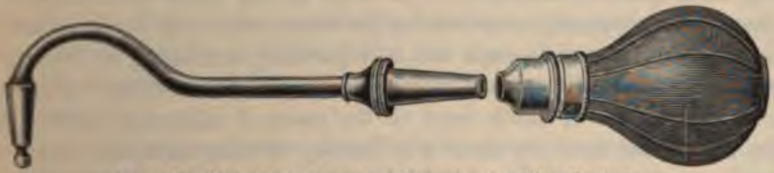

FIG. 19.—Ribemont's laryngeal tube for inflating the lungs.

pacity of the lungs of the child is much less than that of those of the physician. Like artificial respiration, inflation is to be used in connection with the external application of heat, preferably the continuous hot bath.

The method introduced by Laborde, of making rhythmical traction upon the tongue ten or twelve times a minute as a means of exciting respiration, is one of the most efficient within our reach. It may be resorted to in conjunction with other methods, or used alternately with them.

In cases of asphyxia it is not enough to make the child cry. The deep respirations must be made to continue, for very often it happens that resuscitation is only partial, and that the child after six or eight hours lapses into its previous condition. All severe cases require careful watching for the first twenty-four or thirty-six hours, as a repetition of the treatment is often required.

CHAPTER II.

CONGENITAL ATELECTASIS.

This condition is one in which there is a persistence of the fœtal state in the whole or in any part of the lung.

Atelectasis is the pathological condition with which asphyxia of the newly born is usually associated. In most of the cases the condition of atelectasis is completely overcome by the means employed in resuscitation; in some, however, these means are only partially successful, so that a portion of lung of variable extent remains in the fœtal condition. These are the circumstances in which most of the cases of atelectasis arise. But there are others in which there is no history of early asphyxia, where the primary respirations, although taking place spontaneously, have not been of sufficient force and depth to produce full pulmonary expansion. This usually occurs in feeble infants, or in those who are premature. The causes of congenital atelectasis are therefore, in the main, those mentioned as producing asphyxia.

Lesions.—In cases where the child dies during the first few days the amount of expanded lung is often very small, frequently not more than one fourth of the pulmonary area. The expanded portion is usually the anterior borders of the upper lobes. This is often the seat of acute emphysema. The rest of the lung is still in the fœtal state; it is of a brownish-red colour, very vascular, does not crepitate, and shows the lobular outlines both on the surface and on section. With a little force the atelectatic lung may be completely inflated.

If children have lived several months, nearly the whole of the upper lobes and the anterior portion of the lower lobes are usually well inflated. These portions are either normal or slightly emphysematous. The posterior portion of the upper lobes and the lower lobes are almost invariably the seat of the atelectasis. On the surface even these portions may present quite a large area of expanded vesicles, but the lobe is solid to the touch, and crepitates but slightly. On section it is seen that only the most superficial part of the lung is inflated, often only to the depth of a line, while the interior of the lobe is unexpanded. Small hæmorrhages are frequently seen beneath the pleura.

It is usual for both lungs to be affected, and often, but by no means uniformly, to about the same degree. It is frequently a great surprise to discover that a child has lived two or three months without presenting any signs of cyanosis, using not more than one third of its pulmonary area. This variety of atelectasis closely resembles the hypostatic pneumonia of delicate infants, and very often the two conditions are associated. It may require the microscope to decide between them. If congenital atelectasis has existed for some months, there are usually found evidences of pneumonia. Inflation is not so easy as in recent cases, but with force the greater part of the lung can usually be expanded. The heart commonly shows the right auricle and ventricle to be distended with dark clots, and there is occasionally found a patent foramen ovale or some other form of congenital lesion. The liver and spleen are in most cases congested, and the spleen may be considerably enlarged. The mucous membrane of the stomach and intestines is sometimes deeply congested.

Symptoms.—In one group of cases the children are asphyxiated at birth, but the attempts at resuscitation have been only partially successful. Although the patients may live for a few days, there is cyanosis, which gradually deepens, and death takes place from asphyxia, exhaustion, or convulsions.

In a second group of cases the infants have been asphyxiated at birth, and resuscitated perhaps with difficulty, but to all appearance completely. They do not thrive, however, remaining small and delicate, gaining very little or not at all in weight, and showing poor circulation, cold extremities, and occasionally subnormal temperature. It is characteristic of these cases that the cry is never loud, strong, and lusty. Some of them will not cry at all. Such children may live several weeks, or even months. There may develop at any time, often quite suddenly and without assignable cause, attacks of cyanosis with prostration. Children may have several such attacks, which do not excite suspicion since they pass away spontaneously. In other cases the symptoms are so severe that they may result fatally in a few hours, death being frequently preceded by convulsions. If energetically treated the symptoms may pass away but, reappearing in a few hours, or again after a week or more, they gradually deepen in intensity until death occurs.

Two cases coming under my observation in the New York Infant Asylum in 1890, illustrate this point. The infants were twins, ten weeks old and delicate. Suddenly at night one child was taken with convulsions, became deeply cyanosed, and died in two and a half hours. It had been suffering from a slight attack of indigestion and diarrhœa for a week previous, but apparently was not seriously ill. The other twin had been on the previous day as well as for several weeks. Two hours after the death of the first child the second was taken with similar symptoms, dying in a few hours. At autopsy I found very extensive atelectasis involving the posterior part of the upper and the greater part of both lower lobes. The lesions were almost identical in the two cases. In both, the stomach was greatly distended with food and gas. I have repeatedly seen the effect of overdistention of the stomach in producing cyanosis in young children, and in this instance I believe it to have been the exciting cause of the final symptoms. It was subsequently learned that during the six weeks of observation the nurse had witnessed several slight attacks of cyanosis in one of the infants.

I have seen a number of such cases, in which there was nothing whatever to attract attention to the lungs until the final attack of cyanosis occurred, the children showing only the signs of malnutrition. In not all of these cases is there a history of asphyxia at birth. Some are only puny, delicate or premature, exhibiting during the early weeks of life all the signs of feeble vitality. The subsequent course is the same as in those in which there is early asphyxia. The duration of life in these cases depends chiefly upon the extent of the atelectasis.

It is not to be supposed that all cases of congenital atelectasis terminate fatally. Infants in whom there is every reason to believe that atelectasis exists, from the occasional attacks during the first few weeks of cyanosis, feeble cry, poor circulation, etc., may under favourable conditions recover completely, even though no special treatment is directed to the lungs.

Diagnosis.—For this the physical signs are of much less value than the symptoms. It should be remembered that the principal seat of the disease is the lower lobes posteriorly. Percussion usually gives resonance over the entire chest, although this may be somewhat diminished posteriorly. There is not, however, so much change as one would expect to find, for the collapsed areas are surrounded by others which are overdistended, and there are in the midst of the collapsed parts, especially upon the surface, lobules which are inflated. If the two sides are involved to about the same degree, as is often the case, we can get no difference in the percussion note over the two lungs, and the change from the normal may be so slight as not to be appreciable. Where only one lung is affected a difference can usually be made out. The respiratory murmur is rarely bronchial, but generally only feeble in its intensity, and rather ruder in quality than normal. As in the case of percussion, if only one lung is affected this is of some value in diagnosis, but it is not sufficiently marked to be readily recognized when both sides are involved. Occasionally râles are present.

Treatment.—In the newly-born child, whether asphyxiated or not, the physician should see to it that the infant not only cries, but does so loudly and strongly, and that this cry is repeated every day. If children do not cry naturally they must be made to do so by the alternate use of the hot and cold bath, as in cases of asphyxia, or by mechanical means, like spanking. This should be repeated at least twice a day, and continued for from fifteen to thirty minutes. It may seem cruel, but it is often the only means of saving life. Expansion of the lungs is much more easily induced during the first few days of life, becoming more and more difficult the longer it is delayed. Provided the condition is recognized, treatment is fairly successful. In institutions where delicate infants spend most of the time in their cribs, atelectasis is likely to be found. An infant needs exercise, and this is often only to be obtained by taking the child from its crib several times a day, by general friction, massage, the stimulus of fresh air, etc. Nothing is more certain to perpetuate atelectasis than to allow the infant a life of feeble vegetative existence. Food and feeding must be carefully attended to, but even these are of less importance than the maintenance of the animal heat. The temperature is often subnormal, and should be closely watched. If there is difficulty in keeping the child warm it should be rolled in cotton and surrounded by hot bottles, or kept in an incubator during the first few weeks. (See page 10.) During attacks of cyanosis the same means are to be employed as in cases of asphyxia of the newly born—cutaneous stimulation and artificial respiration—the administration of drugs being of little or no value.

CHAPTER III.

ICTERUS.

Several varieties of icterus are met with in the newly born.

1. It is often seen in the various forms of pyogenic infection. In such cases the icterus is usually mild.

2. It may depend upon syphilitic hepatitis—a rare cause.

3. It may be due to congenital malformations of the bile-ducts.

4. The most frequent of all varieties is the so-called idiopathic icterus, sometimes spoken of as "physiological" icterus.

In the cases included under the first and second heads icterus is a minor symptom. The other varieties are sufficiently important to require separate consideration.

MALFORMATIONS OF THE BILE-DUCTS.

The common bile-duct is the most frequently affected. There may be atresia at the point where it opens into the intestine, the duct may be represented by a fibrous cord, or it may be absent altogether. In many cases this is the only lesion; in others it is associated with an impervious hepatic or cystic duct; in still others the common duct is normal, but the cystic or hepatic ducts are impervious.

At autopsy all the organs are usually found intensely jaundiced, particularly the liver. In recent cases this is very much swollen, but presents no marked organic changes. In cases which have lasted several months there is commonly found chronic intestinal hepatitis, sometimes to a very marked degree. This was present in nine of the fifty cases collected by Thomson.* The gall-bladder is usually small, and often rudimentary. In cases of atresia of the common duct it may be greatly distended.

The condition of the bile-ducts is ascribed to an error in development and subsequent catarrhal inflammation. There does not seem to be sufficient evidence to prove that hereditary syphilis is an etiological factor of much importance. This was present in but five of Thomson's cases.

Symptoms.—The most striking symptom is jaundice, which is usually noticed a day or two after birth, and steadily increases until it becomes intense. The urine is colored a dark brown or bronze by bile pigment, and even the meconium stools may be white, except in cases where malformation is limited to the cystic duct. The liver as a rule is much enlarged. The spleen is often swollen. Hæmorrhages beneath the skin or from any of the mucous membranes are quite common. Vomiting is usually absent. In most cases there is progressive wasting, and death within the first few weeks. Of Thomson's fifty cases, nine lived less than a month, and only eighteen over four months. Lotze has reported a case of a child living eight months with an impervious hepatic duct. A frequent cause of death in the rapid cases is convulsions.

These malformations cannot be influenced by any treatment.

PHYSIOLOGICAL OR IDIOPATHIC ICTERUS.

In 900 consecutive births at the Sloane Maternity Hospital icterus was noted in 300 cases. In 88 it was intense, in 212 it was mild. According to the statistics of various lying-in hospitals of Germany, it was found in from 40 to 80 per cent. of all infants. In the 300 cases just referred to, icterus was noticed on the first day in 4, on the second day in 19, on the third day in 72, on the fourth day in 86, on the fifth day in 67, and on or after the sixth day in 44. From the second to the fifth day is therefore the usual period for its appearance.

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* Edinburgh Medical Journal, 1892.

It usually increases in severity for one or two days and then slowly disappears. The average duration in the mild cases is three or four days; in those of moderate severity about a week; in the most severe cases it may last for two weeks. The icterus is first noticed in the skin of the face and chest, then in the conjunctivæ, then in the extremities. The skin varies in colour from a pale to an intense yellow. The urine in most cases is normal. It sometimes is of a light brown colour, and only in the most severe cases does it contain bile pigment. According to Runge, both urea and uric acid are produced in larger amounts than in children not icteric. The stools are unchanged, the normal yellow evacuations occurring in the icteric as early as in those not affected.

According to some observers, in infants who are icteric the initial loss in weight is greater and the subsequent gain slower than in other children. This is not borne out by the Sloane statistics. Of the 300 icteric children, 155 made satisfactory progress in every respect and gained rapidly. The progress in 106 cases was said to be "fair"—i. e., at the time of discharge, usually on the tenth day, a slight gain in weight was noted. The remaining 39 did badly, not gaining in weight and showing other symptoms of malnutrition. The proportion of icteric infants who did well, moderately, and badly, was practically the same as of the other children in the institution not suffering from icterus. Icterus occurs with equal frequency in both sexes. According to Kehrer, it is more frequent in first children than in later ones, and considerably more frequent in premature children than in those born at term. The presentation, the duration of labour and its character—whether natural or artificial—have no influence upon the production of icterus. As a rule icteric children appear in other respects healthy, but in those below the average size the icterus is apt to be more intense.

Few subjects have given rise to wider speculation than this form of icterus. Its exact pathology is at present unknown. Of the many theories advanced, that of Silbermann is perhaps the most satisfactory—viz., that the icterus is due to resorption, and is hepatogenous in its origin. With this view Frerichs and Schultze agree. Silbermann explains the resorption by the existence of stasis in the capillary bile-ducts which are compressed by the dilated branches of the portal vein and the blood capillaries. The change in the circulation of the liver is one of the results of the change in the blood which occurs soon after birth. This results from an extensive destruction of the red blood cells—a kind of blood fermentation. The more feeble the child the more intense the icterus, because the blood changes are more intense. In consequence of this destruction of red blood cells abundant material for the formation of bile pigment exists and accumulates in the hepatic vessels.

In jaundiced infants who have died from accident or other causes the skin and almost all the internal organs are found icteric. There is also staining of the internal coat of the arteries, the endocardium, the pericardium, and the pericardial fluid. Sometimes the subcutaneous connective tissue is yellow, also the brain and cord; the spleen and kidneys only in the most severe cases. In the kidneys uric-acid infarctions are often found, and sometimes bile pigment. The liver is rarely discoloured. The bile-ducts are normal. In certain cases Birch-Hirschfeld has discovered bile pigment in the liver cells.

This jaundice is never fatal, and is not serious. Other conditions, such as atelectasis, may coexist, which may make the case grave. The chief point in diagnosis is not to confound physiological icterus with that depending upon other more serious conditions, such as sepsis or congenital malformation of the bile-ducts. In sepsis other symptoms are present, usually an abnormal condition of the umbilicus, and the symptoms appear at a later date. In malformation of the bile-ducts the jaundice is very intense, and is frequently accompanied by marked hepatic enlargement.

Physiological icterus requires no treatment.

CHAPTER IV.

THE ACUTE INFECTIONS OF THE NEWLY BORN.

It is possible for the newly-born infant to suffer from almost all of the common infectious diseases. Smallpox probably has been most frequently observed. In rare instances measles, influenza, typhoid fever, malaria, and pneumonia have occurred in the first days of life. As the mothers in many instances were suffering from the diseases during or just prior to delivery, the infants appear to have been infected before birth through the circulation of the mother. In other cases, especially in pneumonia, influenza, and gastro-enteritis, infection may take place soon after birth. The symptoms of these diseases in the newly born differ very little from those occurring in any other young infant. In addition to the diseases mentioned, there are other forms of infection which belong especially—some of them exclusively—to the newly born.

THE ACUTE PYOGENIC DISEASES.

Under this head are grouped various infections of the newly born, due to the entrance of the common pyogenic bacteria. They have been designated as *puerperal fever of the child,* also as *pyæmia* or *septicæmia*, or simply as *sepsis of the newly born.* A variety of pathological and clinical conditions are met with. In some cases there is only a localized external inflammation, often terminating in abscess formation; sometimes one or more of the internal organs is affected; occasionally a general blood infection—a true septicæmia—is seen without any noteworthy local lesion; finally, there are the cases attended by the production of multiple abscesses in the viscera, joints, or cellular tissue—a true pyæmia. Formerly infections of this class were very common, especially in large lying-in hospitals; but, owing to the general adoption of the methods of aseptic midwifery, they have steadily diminished.

Etiology.—The source of infection of the child may be the vaginal secretion of the mother or, in rare cases, the mother's milk. Although it has been shown that in a great proportion of the cases the milk of a woman suffering from mastitis or from septicæmia contains pyogenic germs, still the taking of these into the stomach is not likely to infect the infant. More frequently the child is infected by the nurse in the process of dressing the cord, bathing, or cleansing the mouth or eyes, possibly after having attended to the needs of a septic mother or another child. Infection may be carried by the physician, by instruments, or by the dressings of the cord. Infection through the atmospheric air, while possible, is not a frequent cause.

Infection through the umbilicus may occur either before or after the separation of the cord. The poison may enter through the umbilicus, although this may give no external evidence of disease. This was true in a case studied by Van Gieson, in which the infant died of meningitis when eight days old. The cord had healed properly, and at the autopsy the navel appeared normal. But the umbilical vessels inside the body contained pus. From this the meningitis evidently arose, as the same bacteria were found by culture both there and in the brain. Entering through the mouth, bacteria may lead to infectious processes in the throat, they may involve the stomach and intestines, rapidly producing death; or the alimentary tract may be the focus from which infection of distant parts may arise.

The micro-organisms chiefly concerned in these infections are the common pyogenic bacteria, staphylococcus pyogenes aureus and the streptococcus. The next in importance is the gonococcus, the rôle of which, especially in cases accompanied by joint suppuration, has only recently been appreciated. In one case of meningitis of my own only the colon bacillus was found. Pneumococcus infections occasionally complicate the others mentioned. While streptococcus infections are in general more serious than those due to the staphylococcus, some of the most severe ones met with belong to the latter class.

Clinical Varieties.—*Omphalitis.*—In this variety there is inflammation of the umbilicus, and cellulitis of the abdominal wall in the immediate neighbourhood. This results in the formation of an umbilical phlegmon. It may terminate in resolution, in abscess, or in gangrene. The usual termination is in abscess. These abscesses may be small and superficial, or they may be more deeply seated between the abdominal muscles and the peritonæum. Omphalitis usually begins in the second or third week of life, before the umbilicus has cicatrized. Locally there are redness, swelling, and induration. The process may result in abscess, there may be diffuse inflammation of the abdominal walls of an erysipelatous character with extensive sloughing, or the infection may spread to the peritonæum.

Inflammation of the umbilical vessels.—This is one of the most frequent primary processes in pyæmic infection. The umbilical arteries are more frequently involved than the vein. According to Runge, inflammation of the vessels is always preceded by inflammation of the connective tissue which surrounds them, as the poison is taken up by the lymphatics and not by the blood-vessels. Omphalitis is frequently present, but in some cases the umbilicus shows nothing abnormal.

In arteritis the vessels may be involved to any degree: sometimes only a short distance from the abdominal wall, sometimes quite to the bladder. They contain pus, and often septic thrombi. Saccular dilatation is frequently present at several points. Pus sometimes exudes from the umbilical stump on pressure. The other lesions accompanying arteritis are those of pyæmic infection, more or less widely distributed. There are frequently peritonitis, suppuration of the joints, erysipelas, multiple abscesses of the cellular tissue, sometimes suppurative parotitis. Atelectasis is common. Pneumonia was found in twenty-two of Runge's fifty-five cases.

In cases of phlebitis, the umbilical vein is usually involved for its entire length from the abdominal wall to the liver. This may lead to an acute interstitial hepatitis going on to suppuration, or to phlebitis of the portal vein and some of its branches. In either case there is more or less parenchymatous hepatitis, and often multiple abscesses of the liver, most of the patients being jaundiced. Peritonitis also is a frequent complication.

Peritonitis.—This is one of the most frequent pathological processes in pyæmic infection, and is very often the cause of death. It is generally associated with umbilical arteritis, and often with erysipelas. In a considerable number of cases it is the most important lesion found. It may be localized or general. Localized peritonitis is generally in the neighbourhood of the umbilicus or of the liver. It may result in adhesions, or in the formation of peritoneal abscesses. More frequently the peritonitis is general, and resembles the septic peritonitis of adults. There is a great outpouring of lymph coating the intestines and other viscera and the inner surface of the abdominal wall, causing adhesions between the abdominal contents. Collections of sero-pus are found in the pelvis and in various pockets formed by the adhesions. Sometimes blood is present in the exudation.

The special symptoms which indicate peritonitis are vomiting, abdominal tenderness and distention, and protrusion of the umbilicus. The abdominal enlargement is chiefly from gas, but may be partly from fluid. There are present thoracic respiration, dorsal decubitus, and flexion of the thighs as in all varieties of acute peritonitis. The temperature is usually but not necessarily high.

Pneumonia.—The most common form seen is pleuro-pneumonia. There is an abundant exudate of grayish-yellow lymph covering the lung. Occasionally collections of pus are found in the sacs formed by the adhesions. Serous effusions are rare. The pulmonary lesion consists usually in a broncho-pneumonia, with consolidation of larger or smaller areas in the lungs—more often in the upper than in the lower lobes. It is not uncommon for minute abscesses to be found in the lung at various points. There is a purulent bronchitis of the larger and smaller tubes.

The symptoms are obscure and often indefinite. The only characteristic ones are cyanosis and rapid respiration, with recession of the chest walls on inspiration. The physical signs are inconstant and uncertain. Pneumonia cannot usually be diagnosticated during life. In most of the fatal cases of pyogenic infection, whatever its type, there is found some involvement of the lungs. The changes are most extensive in cases in which the serous membranes are involved.

Pericarditis is rare and usually associated with pleurisy. Endocarditis is very rare. Hirst has, however, reported a case.

Meningitis.—The pia mater is the least liable to be affected of all the serous membranes, with the possible exception of the pericardium. When meningitis is present it is usually associated with peritonitis or with pleurisy. The lesions are those of acute purulent meningitis with a copious exudation, sometimes associated with meningeal hæmorrhages, or with acute encephalitis and the production of multiple minute abscesses in the cortex. The local symptoms are often not marked, and are sometimes very obscure. The most characteristic are stupor, dilated pupils, opisthotonus, bulging fontanel, general rigidity, convulsions, and occasionally localized paralyses. The temperature is generally high.

Gastro-enteritis.—Diarrhœa is a frequent symptom in all septic cases, constipation being rarely present. In many instances vomiting is a prominent symptom. In a small proportion of cases the most important local lesions are in the intestines, generally in the nature of a superficial catarrhal inflammation.

Pseudo-membranous inflammations of the throat.—These are rarely seen in the newly born. J. Lewis Smith has made a report on a group of five cases occurring as a small epidemic in the New York Infant Asylum. They were associated with other lesions, and all were fatal. In several cases there was omphalitis. One of these was studied bacteriologically by Prudden, who found no Loeffler's bacilli, but streptococci both in the exudation in the throat and in the umbilical abscess. Such inflammations are to be regarded as one manifestation of a general streptococcus infection.

Osteomyelitis.—Allard has reported a series of cases in which, after the general and local symptoms of pyogenic infection had existed for some time, suppuration occurred over various bones, especially the humerus, tibia, metatarsal bones, sacrum, etc. Trephining revealed the lesions of osteomyelitis. The abscesses usually made their appearance between the fourth and the sixth week. The most rapid case died on the fourteenth day, and none lasted more than two and a half months.

Joint suppuration.—In certain pyæmic cases, and in some in which there are no other symptoms, acute suppuration in the joints occurs. This may come on very acutely in the first or second week, or more slowly as late as the second or third month. In the acute cases it is exceptional to have but one joint involved; often there are four or five. The small joints are rather oftener affected than the large ones, but almost any articulation in the body may be involved. With multiple joint suppuration there are present the general symptoms of pyæmia—high temperature, marked prostration, wasting, and often secondary visceral inflammations develop. In those which occur late, fewer joints are involved, often but a single one, the febrile symptoms are less marked, and the duration may be much longer. In my own experience, the organism most frequently found in these cases is the gonococcus; next to this in importance is the streptococcus. The joint lesion is usually a superficial one, the bones often escaping. The gonococcus cases probably occur most frequently as a complication of ophthalmia; but I have seen several in which ophthalmia was not present and where the point of entry could not be determined.

Abscesses in the cellular tissue.—These are quite frequent, and may occur with suppuration in the joints or internal organs, or they may exist as the only lesion. They are nearly always multiple and may be found in almost any location. They vary in size from that of a small pea to one containing half an ounce of pus. They are due to the introduction of pyogenic germs, usually staphylococci. Their course is benign, and they require no treatment except incision and cleanliness. Where there is a disposition to their continued formation, the skin should be washed with an antiseptic solution.

Erysipelas.—This is seen especially during the first two weeks of life, and usually starts from the umbilicus or some abrasion of the skin, most frequently about the genitals, or the scalp. When originating at the umbilicus it is generally complicated by other lesions, such as peritonitis and umbilical phlebitis. If it starts from any other part of the body it may be uncomplicated. Erysipelas beginning at the umbilicus gives rise to an area of induration and a circumscribed blush. At first it may resemble a simple cellulitis; but the steadily increasing area of elevated induration and redness soon indicates the nature of the inflammation. From whatever point starting, the erysipelatous inflammation, owing to the feeble resistance of the tissues, in most cases spreads widely. The entire abdomen, chest, and back may be involved, and it may even spread to the extremities. It may extend so that nearly the whole trunk is affected in four or five days. It usually involves only the skin and superficial cellular tissue; but it may involve the deeper areolar planes and terminate in diffuse suppuration, or even in gangrene.

The constitutional symptoms are severe: great prostration, continuously high temperature—102° to 105° F.—rapid wasting, and often vomiting, diarrhœa, or convulsions are present. The disease is always serious, and usually fatal. It is often complicated by broncho-pneumonia.

Distribution of the Lesions.—The frequency of the different visceral lesions in eighty-seven autopsies published by Bednar was as follows: Peritonitis in twenty-nine, pneumonia in fifteen, pleurisy in ten, meningitis in nine, meningeal hæmorrhage in eight, encephalitis in eight, cerebral hæmorrhage in four, entero-colitis in five, pericarditis in four. In thirty-one cases there was umbilical arteritis, and in nine cases umbilical phlebitis. There was one case each of pulmonary hæmorrhage, pleural hæmorrhage, acute hydrocephalus, acute bronchitis, and suppuration in the cellular tissue. Runge's later observations of thirty-six cases showed umbilical arteritis in thirty, umbilical phlebitis in three, and normal umbilicus in three. He found pneumonia in twenty-two of fifty-five cases. Other lesions frequently associated are atelectasis, swelling and softening of the spleen, cloudy swelling of the liver and kidneys, occasionally with foci of suppuration in these organs. The blood is dark, and coagulates imperfectly.

General Symptoms.—These may begin at any time during the first ten days—very rarely after the twelfth day. Fever is an exceedingly variable symptom—it may be very high; it may be almost absent; occasionally there is subnormal temperature. The course of the temperature is very irregular. Wasting is constant and quite rapid. It depends upon the inability to take and digest food, upon the intestinal complications, and upon infection. In quite a number of cases wasting is almost the only symptom. Icterus is exceedingly common; in many of the worst cases it is intense. It is met with where the liver is the seat of an acute parenchymatous or acute suppurative inflammation, and in many other cases where it depends apparently upon the blood changes. Hæmorrhages are common, and may be the direct cause of death. They may come from the umbilicus, the intestine, or almost any mucous membrane. They are sometimes subcutaneous, causing a general hæmorrhagic eruption. Nervous symptoms are generally present, and are sometimes marked. They are restlessness, rolling of the head, a constant whining cry, twitchings of the muscles of the extremities or face, stiffening of the body, more rarely general convulsions. Late in the disease, dulness and stupor are present. The pulse is rapid and weak and the respirations are often irregular, even when there is no cerebral complication. Diarrhœa is frequent; the stools are green, brown, sometimes black from the presence of blood, and are often very foul. Vomiting is less common.

In addition to these there are symptoms due to the various forms of local inflammation—peritonitis, meningitis, pneumonia, subcutaneous suppuration and gangrene, these all being found in varying degrees and in various combinations.

Prophylaxis.—Pyogenic infection of the child, like puerperal fever in the mother, may be considered a preventable disease. Its occurrence is usually due to a failure to carry out proper rules regarding cleanliness and asepsis in connection with delivery. The statistics of the Moscow Lying-in Asylum, published by Miller in 1888, show that previous to the general introduction of antiseptic methods, from six to eight per cent of all infants born in the institution died from some variety of infection. In twenty-three hundred successive labours at the Sloane Maternity Hospital, covering about eight years, not a single marked case occurred.

From these figures it will be evident that in the vast majority of cases the occurrence of a case of infection of a serious nature is the fault of the physician or nurse in attendance.

The umbilicus should be cleansed and treated like any other fresh wound. Dry dressing should invariably be employed, and sterilized gauze or salicylated cotton in preference to household linen. If suppuration occurs at the time the cord separates, the parts should be cleansed daily with a bichloride solution, and a wet dressing of the same applied. The ligatures and everything which comes in contact with the umbilical wound should be sterilized. Careful attention should be given to the mouth, genitals, and all the muco-cutaneous surfaces, to prevent excoriations and intertrigo. Finally, every septic case occurring in an institution should be immediately isolated. A nurse in charge of a septic mother should not have the care of the infant.

Prognosis.—Pyogenic infections in the newly born, even in their mildest forms, are serious, and in their most severe forms almost always fatal. Very few cases recover in which erysipelas or any important visceral inflammation is present. The resistance of these little patients is so feeble that the tendency of every inflammation is to spread, until the child dies of exhaustion. Only patients with localized inflammations, such as those of joints, skin, etc., are likely to get well.

Treatment.—This practically resolves itself into the treatment of individual symptoms as they arise. Wherever suppuration occurs, external abscesses should be evacuated and treated antiseptically. For the local inflammations of the lungs, peritonæum, and brain, little or nothing can be done in the way of direct treatment. Such inflammations are to be prevented, but can seldom be cured. The general indications are to look closely to the child's general nutrition by careful attention to all details of nursing and feeding, using stimulants whenever required by the condition of the pulse. For a local application in erysipelas, nothing in my experience has proven better than ichthyol ointment, ten to twenty-five per cent strength. It should be applied daily, spread upon muslin, which is then covered by gutta-percha tissue to prevent drying.

OPHTHALMIA.

Ophthalmia of the newly born is to be classed among the pyogenic diseases. It usually consists in a purulent conjunctivitis. In the more severe cases there may be ulceration of the cornea, and even perforation into the anterior chamber of the eye.

The highly infectious nature of this ophthalmia is established. In the most severe cases the micro-organism generally found has been the gonococcus; but in the milder forms the gonococcus may be absent, and any of the common pyogenic germs may be found. In the gonococcus cases the infection occurs during labour, from the secretions of the mother, from the examining fingers of the physician, or from instruments; or after birth from infected cloths and other materials which come in contact with the eye. Healthy lochia produce only a catarrhal inflammation. The infection occurring after birth may take place at any time. That due to gonorrhœal infection from the mother is generally manifested on the third day, and is often violent from the outset.

The symptoms are swelling of the lids, chemosis, copious purulent discharge, sometimes hæmorrhages from the lids, ulceration and there may even be sloughing of the cornea. The course of the disease depends upon the cause and upon the treatment employed. In the cases not due to the gonococcus the course is generally benign, and with ordinary cleanliness usually results in recovery without any permanent damage to the sight. The gonorrhœal cases, unless energetically treated from the outset, are very frequently followed by permanent loss of vision. The best statistics upon the causes of blindness in adults show that from twenty-six to thirty per cent of such cases are due to ophthalmia in the newly born. This disease is occasionally complicated by other symptoms of gonococcus infection of a pyæmic nature. Many cases followed by acute articular symptoms have been observed.

Prophylaxis is of the utmost importance. Credé's statistics show that in 1874 the frequency of ophthalmia in his lying-in hospital was 13·6 per cent. In the three years ending 1883, among 1,160 newly-born children only one or two cases occurred. The method of prophylaxis which he adopted consists in dropping into the eyes of every child, immediately after birth, one or two drops of a two-per-cent solution of nitrate of silver. The general adoption of Credé's method, or of some similar means of disinfection, has resulted in a very great diminution in the frequency of ophthalmia throughout the world. These prophylactic means should be obligatory in all institutions, and should be used in all cases in private practice wherever there is any possible suspicion of the existence of gonorrhœa. In all other cases the eyes should be carefully cleansed with a saturated solution of boric acid. The use before delivery of an antiseptic vaginal douche is theoretically indicated, but practically it has been found to be inadequate to the prevention of the disease.

Treatment.—Everything which comes in contact with the eyes should be carefully disinfected. All cloths, cotton, etc., used for cleansing should be immediately burned. The strictest antiseptic precautions should be insisted on to prevent the spread of the infection by nurses. In institutions containing infants, severe cases of ophthalmia should always be isolated. The most important thing is to keep the eyes clean. In severe cases they must be cleansed every twenty minutes, night and day. It may be done by irrigation, or by using an eye-dropper with a bulbous tip, inserted alternately at the inner and the outer angle of the eye, and the fluid injected with force sufficient to empty thoroughly the conjunctival sac.

Either a saturated solution of boric acid, or a 1-to-5,000 solution of bichloride, may be used in this way. Once or twice in twenty-four hours two or three drops of a one-per-cent solution of protargol should be used in each eye after cleansing with sterile water; this preparation is altogether more efficient than the commonly employed silver nitrate. Next to these measures is the use of cold. It may be applied as ice compresses which are changed every minute or two from a block of ice to the eye. These may be continued one-fourth of the time in the milder cases; in the severe ones almost constantly. When the cornea is involved the pupil should be dilated by atropine. If only one eye is affected the sound one should be protected by covering it with a compress kept wet with an antiseptic solution.

TETANUS.

Tetanus is an acute infectious disease characterized by tonic muscular spasm, which increases in severity by paroxysms occurring at longer or shorter intervals. It may be limited to the muscles of the jaw (trismus), or may affect all the muscles of the trunk, extremities, and neck.

Though many writers have sought to maintain a difference between tetanus of the newly born and tetanus of later life, whether traumatic or not, their identity has been admitted for at least a dozen years. The discovery of the exact cause of tetanus is due to the work of Nicolaïer, who in 1884 found a bacillus in the soil, with which he produced the disease in animals. He demonstrated the presence of this bacillus in the wounds of tetanus patients. Nicolaïer did not, however, obtain the germ in pure culture; but this was done by Kitasato in 1889. The bacillus is generally known as Nicolaïer's bacillus. Since that time the germ has been found in the wounds of numerous patients with tetanus, including newly-born infants.

The rapidity with which the infection spreads from the point of inoculation is very remarkable, as shown by Kitasato's experiments. Thus, if one hour elapsed after infection before cauterizing the inoculated wound, the animal succumbed to the disease. The bacilli are not found in the blood or internal organs. The symptoms of the disease have been shown to depend upon the absorption of a toxic product of the tetanus bacillus called *tetano-toxine.*

The germ of tetanus usually gains access to the body of the infant through the umbilical wound. It exists in the soil, and the disease prevails endemically in certain localities. It is common in certain parts of Long Island and New Jersey. Among the negroes in some parts of the South it has for many years occurred with great frequency. It is stated that on one of the islands of the Hebrides every fourth or fifth child dies of tetanus. In a single house in Copenhagen eighteen cases were observed. Tetanus is rare except where dirt and filth prevail; but these alone are not sufficient to produce the disease. It is a very rare disease in the tenements of New York.

Lesions.—There are no essential lesions of tetanus. Those which have been found have been partly accidental and partly a result of the disease rather than its cause. In most of the cases intense hyperæmia of the spinal cord and its membranes is found, and not infrequently small extravasations of blood. Such small hæmorrhages are occasionally found in the meninges of the brain—more frequently at the base than at the convexity. In rare instances hæmorrhages of considerable size have occurred into the brain itself. The lungs are generally congested, and the right side of the heart overdistended. In most of the cases the umbilicus has not healed, and it may present evidences of septic infection in varying degrees.

Symptoms.—These, as a rule, begin on the fifth or sixth day, or at the time of the separation of the cord. The first symptoms may not appear until the tenth or twelfth day, but rarely later than this. Generally the first thing noticed is difficulty in nursing, which, on examination, is found to be due to rigidity of the jaws (trismus). Nursing may be impossible on this account. The muscles of the jaw feel hard, the lips pout and all the muscles of the face seem firm. Soon a slight stiffening of the body occurs, the child straightening the back as it lies upon the lap and continuing rigid for a moment or two. In the interval it is at first completely relaxed. These paroxysms soon increase in frequency until they may come on every few minutes, being excited by any movement of the body. The relaxation is then only partial, and the neck and extremities, sometimes nearly the whole body, become rigid and stiff as a piece of wood. The arms are extended, the thumbs adducted, and the hands clenched. The thighs and legs are extended, and no motion is possible at the hip or knee. The jaws can be separated slightly or not at all. The firm contractions of the facial muscles give a peculiar expression to the features. There is a low, whining cry. Swallowing is difficult, sometimes impossible. The pulse is rapid and soon becomes weak. The temperature at first is normal, but in the most acute cases rises rapidly to 104° or even 106°; in the milder cases it does not go above 101° F.

Death is due to exhaustion, to fixation of the respiratory muscles, or to spasm of the larynx. In the less severe cases all the symptoms are milder, and there may be intervals in which the rigidity is scarcely noticeable, so that respiration and deglutition may be carried on for some time. In cases which terminate in recovery the temperature is but slightly elevated. The tonic contractions gradually become less severe, and the paroxysms less frequent. The children usually suffer for several weeks from the general symptoms of malnutrition, which are proportionate to the severity of the attack. Of eighty-eight fatal cases which are reported by Stadtfeldt all but five died between the ages of six and ten days. The duration of the disease in the fatal cases is seldom more than forty-eight hours, often less than twenty-four hours; in those terminating in recovery, between one and three weeks.

Prognosis.—No disease of infancy is more fatal than tetanus. Where it prevails endemically it is regarded by the laity as so uniformly fatal that usually no physician is called. Scattered through medical literature are quite a large number of isolated cases in which recovery has occurred. At the present time the proportion of fatal cases is probably between ninety and ninety-five per cent. Sporadic cases more frequently recover than those occurring in districts where the disease is endemic. The later the development of the symptoms, the slower their course, and the lower the temperature the more likely is the case to recover.

Prophylaxis.—A proper understanding of the nature of the disease has brought with it the means of rational prevention. The first essential is obstetrical cleanliness, which must include scissors, hands, dressings, ligatures—in short, everything which comes in contact with the umbilical wound. In districts where tetanus is endemic, thorough antiseptic treatment of the umbilicus should be insisted upon, both at the first dressing and later, particularly at the time of the separation of the cord.

Treatment.—All drugs whose physiological action is that of motor depressants of the spinal cord have a certain amount of value in tetanus. The most important ones are chloral, the bromides, and calabar bean. Nearly all the reported cures have been by one of these drugs or a combination of them. The mistake usually made is in using too small doses to be of any efficacy. Enough to produce the physiological effects of the drug must be given. The initial dose should not be large, but it should be repeated until the full effects are obtained. Of those mentioned, chloral has been the one most generally relied upon. An hourly dose of one or two grains is usually required. If no effect is visible in ten or twelve hours the dose may be further increased, as the patient is in much greater danger from the disease than he can possibly be from the drug. Chloral may be given by the mouth or by the rectum, but must always be well diluted. The single case of recovery which I have witnessed was one treated by the bromide of potassium. This infant took eight grains every two hours for three days, afterwards smaller doses. Calabar bean has the advantage in that its extract may be given hypodermically; one tenth of a grain may be administered from three to ten times daily, according to the severity of the symptoms. Monti has reported two cases cured by its use. The child must at all times be kept as quiet as possible, without unnecessary handling or bathing. If nursing or feeding by the mouth is impossible, because the jaws cannot be separated, the child may be fed by a tube passed through the nose. This is greatly to be preferred to rectal alimentation. Drugs may be administered in the same way.

The antitoxine treatment.—Behring and Kitasato, after a series of experiments upon animals, were the first to produce an *antitoxine* which has the power of neutralizing the tetanus poison. In animals immunity is produced by its injection. It is also curative in those cases where tetanus has been produced experimentally. Its value has now been demonstrated in quite a large number of cases of traumatic tetanus in adults. The practical obstacle to the success of the antitoxine treatment is the rapid absorption of the tetanus poison from the wound. To be efficient it must be used early.

Cases of tetanus neonatorum successfully treated by antitoxine have been reported by Papiewski, Escherich, McCaw, and others; but the number of cases in which it has been used is as yet too small to admit of positive deductions. It should by all means be tried wherever practicable. The best method of administration is still under discussion. Roux's experiments appear to show that the antitoxine is more efficient when injected directly into the brain than when used subcutaneously. Fortunately in the newly-born child this adds no difficulty, since the needle can readily be introduced through the open sutures. It is hardly necessary to add that the strictest antiseptic precautions must be observed. Reliable tetanus antitoxine is now prepared by Behring, the New York Health Department, and Parke, Davis & Co. The question of dosage is still unsettled.

EPIDEMIC HÆMOGLOBINURIA (WINCKEL'S DISEASE).

The essential features of this disease are hæmoglobinuria with icterus and cyanosis, this combination giving the skin a deeply bronzed hue (*maladie bronzée*). It is a rare disease, but has generally occurred epidemically in institutions. It is usually fatal. All the symptoms point to an acute, rapid disintegration of the red blood-cells—a sort of blood fermentation. It is, without doubt, infectious, but its cause has not been discovered. Although generally called by the name of Winckel,* who in 1879 made a report upon an epidemic of twenty-three cases, the disease was quite well described by Charrin in 1873, with a report of fourteen cases, and observed by Bigelow, in Boston, in 1875. All the cases included in Winckel's report occurred in one institution, affecting one fourth of the children born during the period.

There is cyanosis, with a more or less intense icterus of the skin and internal organs. The umbilical vessels are usually normal. The kidneys are swollen, show small hæmorrhages into their substance, and under the microscope the straight tubes are seen to be filled with crystals of hæmoglobin, but contain no blood-cells. The bladder frequently contains brownish, smoky urine. The spleen is swollen and filled with blood pigment, which is diffused throughout the cells of the pulp, and free in the blood-vessels. Punctate hæmorrhages are seen in most of the other viscera.

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* Winckel, Veröffentlich. der pädiatrischen Section der Gesellsch. f. Heilk., Berlin, April, 1879.

This disease most frequently attacks those who have been previously healthy. The symptoms usually begin from the fourth to the eighth day after birth. They are intense and fulminating in character, seldom lasting more than two days, and often only one. The early symptoms are general restlessness, rapid pulse and respiration, prostration, cyanosis of the face, and general icterus, which is at first slight, but steadily increases until it becomes intense, the skin resembling that of a mulatto. The temperature is normal or slightly elevated. There is rapid asthenia, often terminating in coma or convulsions. The most characteristic symptoms are those connected with the urine. It is passed frequently, in small quantities, with pain and straining. It is of a brown, smoky colour, and under the microscope shows hæmoglobin in considerable quantity, renal epithelium, and sometimes granular casts and blood-cells, but does not contain bile pigment. Albumin is sometimes present, but not in large quantity. Examination of the blood shows an increase of the white cells and many free granules.

Treatment is of little avail, since all severe cases die.

FATTY DEGENERATION OF THE NEWLY BORN (BUHL'S DISEASE).

A disease has been described by the author whose name it bears, the essential nature and causation of which are unknown. It occurs as isolated cases and not in groups, and is characterized by inflammatory changes leading to fatty degeneration in the viscera, especially the heart, liver, and kidneys; it seldom lasts more than two weeks, and is almost invariably fatal. There may be hæmorrhages in any of the viscera, into the serous cavities, or from any mucous membrane. In the lungs are found large or small hæmorrhagic infarctions, and the bronchi contain blood and bloody mucus. The liver in recent cases is large and soft; in those of longer standing it is pale and jaundiced, and shows marked fatty degeneration. The spleen is large and soft. The epithelium of the tubules of the kidney is acutely degenerated. The heart muscle is pale, soft, and fatty. Many of the lesions are similar to the ordinary post-mortem changes, and when found they should not be interpreted as pathological unless the autopsy is made within at least twelve hours after death.

The clinical features of this disease, as described, resemble those of pyogenic infection; and since the observations were made before modern methods of bacteriological study, it is highly probable that Buhl's disease is merely a form of pyogenic infection in the newly born.

PEMPHIGUS NEONATORUM—BULLOUS IMPETIGO.

Pemphigus is a term which designates a lesion rather than a disease. By it is meant an eruption of bullæ occurring usually upon a red base, the contents being in most cases clear serum. A condition somewhat resembling pemphigus sometimes follows the use in the newly born of too hot baths. Again, bullæ are seen as one of the lesions of congenital syphilis; they are then usually present at birth or appear soon after. They are most frequently seen upon the palms and soles. Infants so affected are generally in wretched condition, and soon die.

The only condition to which the term *pemphigus neonatorum* should be applied is quite different from both the preceding, and it has nothing in common with the pemphigus of later life. The disease is of infectious origin; it is somewhat contagious, and occasionally occurs in small epidemics in institutions. It differs from the common impetigo contagiosa seen in older children, chiefly in severity and its association with visceral infections. Most patients in whom the disease occurs are delicate, but not always. I have seen it even in robust infants.

The greater number of cases studied thus far have shown the presence in the blebs of the staphylococcus pyogenes aureus. This was true of three typical cases occurring in my own hospital service. In one of these which came to autopsy, a general staphylococcus septicæmia was present. It is, however, not impossible that the staphylococcus infection is a secondary condition, the primary one being as yet undetermined.

The clinical picture presented by pemphigus neonatorum is so striking that it can scarcely be mistaken. The symptoms begin in most cases between the fourth and tenth day of life. The bullæ first appearing are scattered and often not larger than one fourth or one half inch in diameter. They may be seen upon any part of the body, but are especially frequent about the face, hands, and other exposed parts. They rupture or dry to form crusts without suppuration. The small bullæ may gradually increase in size or several may coalesce until they cover an area two or three inches in diameter. As the disease progresses, new bullæ come out over almost any part of the body. The skin at first appears slightly reddened, then an exudation of serum occurs beneath the epidermis which loosens and slides upon the true skin. After rupture of the large bullæ, the epidermis at the margin forms a thin filmy border or hangs in shreds easily detached. The base of the large vesicles is a moist bright red surface. When many have formed, the appearance closely resembles that seen after an extensive burn. (Fig. 20.)

The course of the local symptoms is at first slow; then the bullæ may spread with great rapidity and death occur in from twenty-four to forty-eight hours. In less severe cases the course is more prolonged, the blebs are smaller, and recovery may take place.

The constitutional symptoms are at first wanting, but increase with the number and extent of the bullæ. There may be a slight rise of temperature or it may be subnormal. There is progressive weakness and great depression, much like that occurring after a burn, and death occurs from exhaustion or from some visceral inflammation such as pneumonia or meningitis.

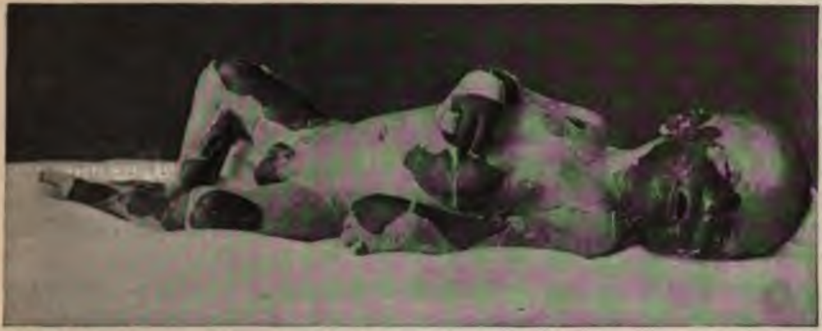

FIG. 20.—Pemphigus neonatorum. Symptoms began on 13th day; death on 16th day of asthenia; temperature subnormal. The dark areas in the picture are entirely denuded of epidermis; they were formed by the coalescence of large bullæ.

It is important to distinguish pemphigus neonatorum from congenital syphilis. In syphilitic cases, the liver and spleen are usually markedly enlarged, and other characteristic changes may be present in the nails, mucous membranes, or elsewhere.

No treatment is of any avail in the most severe cases, when the bullæ cover a considerable part of the surface of the body. In all cases the indications are absolute cleanliness and the use of absorbent powders, such as equal parts of boric acid and starch, to dry up the eruption, or wet dressings of 1–10,000 bichloride or one-per-cent solution of ichthyol. On account of the contagious nature of the disease cases occurring in institutions should be isolated.

CHAPTER V.

HÆMORRHAGES.

HÆMORRHAGES are quite frequent during the first days of life, and are important not only from the fact that they are often the cause of death, but, when the brain is the seat, from their remote effects. There are several conditions in the newly born which predispose to bleeding—the extreme delicacy of the blood-vessels, and the great changes taking place in the blood itself and in the circulation in the transition from intra-uterine to extra-uterine life. Hæmorrhages may complicate many of the diseases of the early days of life, such as syphilis or sepsis, or they may exist alone.

The cases may be divided into two groups: (1) Traumatic or Accidental Hæmorrhages, which depend upon causes connected with delivery; (2) Spontaneous Hæmorrhages, or The Hæmorrhagic Disease of the Newly Born.

TRAUMATIC OR ACCIDENTAL HÆMORRHAGES.

These are mainly due to pressure in natural labour, or to means employed in artificial delivery, but some of them may possibly result from injuries received before birth. They are more frequent in large children, in difficult labours, and where from any cause the body of the child has been subjected to undue pressure.

Hæmatoma of the Sterno-Mastoid.—Hæmatoma, or, as it is sometimes called, induration of the sterno-mastoid muscle, leads to the formation of a tumour in the belly of the muscle. It is a rare condition, usually noticed in the second or third week of life, and it disappears spontaneously, without causing any permanent deformity. The tumour varies from three quarters of an inch to one inch and a half in length, being about the size and shape of a pigeon's egg. It is movable, almost cartilaginous to the touch, and sometimes slightly tender. The situation of the tumour is usually about the centre of the muscle. There is no discoloration of the skin.

In about two-thirds of the cases it occurs after breech presentations. It is much more frequent upon the right than upon the left side. In twenty-seven cases collected by Henoch the right side was involved in twenty-one and the left in only six cases. The explanation of this difference is to be found in the obstetrical position. Rarely, both sides may be involved. The head is usually inclined towards the shoulder of the affected side and rotated towards the opposite side. The swelling slowly diminishes in size, and in most cases by the end of the third month has entirely disappeared. Occasionally a slight torticollis remains for a longer time, but in the majority of cases the recovery is perfect. Hæmatoma of the sterno-mastoid is due to the twisting of the head during parturition. It is not an evidence of the employment of any improper force in delivery. The twisting of the head produces laceration of some of the blood-vessels of the muscle, and in some cases there is doubtless rupture of some of the fibres of the muscle itself. Following this there occurs a certain amount of inflammation of the muscle and its sheath. The tumour is due partly to blood-extravasation and partly to inflammatory products. In one or two recent cases in which the sheath of the muscle has been opened it has been found filled with blood.

The condition requires no treatment. Operative interference is positively contra-indicated.

Cephalhæmatoma.—This is a tumour containing blood, situated upon the head, usually over one parietal bone, and tending to spontaneous disappearance by absorption. The source of the blood is the rupture of the small vessels of the pericranium.

Etiology.—Cephalhæmatoma is sometimes due to a distinct traumatism like the application of forceps or to some other injury during labour. In the majority of cases, however, there is no evidence of such injury. Besides the conditions predisposing to all hæmorrhages, there is the increased pressure in the blood-vessels of the head during delivery, especially when labour is prolonged or difficult; there may be changes in the bone, such as an imperfect development of the external table, which has been found in a few instances, and in consequence of which the periosteum readily separates when the head is subjected to the pressure of the pelvis; and, finally, there may be changes in the blood itself. Cephalhæmatoma is a comparatively rare condition, being present, according to the statistics of the Sloane Maternity Hospital, in 20 of 1,300 consecutive births, or 1·6 per cent. The condition is more common after first, or difficult labours, and in vertex presentations; occurring twice as often in males as in females, probably from the greater size of the head.

Lesions.—In the 20 Sloane cases, the situation was over the right parietal bone in 12; over the left in 2; over both parietals in 4; over the occipital in 2. The location of the tumour seems to have a very close relation to the position of the head in the pelvis. In 8 of the right-sided cases the head was in the left occipito-anterior position; in 3 it was in the right occipito-anterior; in 1 case the position was unknown. Of the cases with occipital tumours, both were breech presentations. Of the 16 cases with a single tumour the labour was natural in 10, tedious in 4, and in 2 forceps were used. Of the 4 double cases, 2 were forceps deliveries, 1 a tedious labour, and but 1 was natural.

In rare cases triple tumours are met with, one over each parietal and one over the occipital bone. The attachment of the periosteum along the sutures, usually limits the tumour to the surface of one bone. It never extends across the sutures or over the fontanel. In cases where there is a more definite injury, such as from forceps, the tumour may be present over any one of the cranial bones, but more frequently over the parietal. The seat of the hæmorrhage is between the periosteum and the cranium. The scalp shows punctate hæmorrhages and sometimes infiltration with blood. In recent cases the blood is fluid; later it is coagulated. The amount of extravasated blood is usually from half an ounce to an ounce. In extreme cases it may be from four to six ounces. The cases following natural delivery are generally uncomplicated. The traumatic cases may be complicated by extravasations between the bone and the dura (internal cephalhæmatoma), or by meningeal or cerebral hæmorrhages. If there is a wound, infection may be followed by purulent meningitis and even by cerebral abscess.

Symptoms.—The tumour is usually noticed from the first to the fourth day after birth, appearing as a slight prominence in one of the positions mentioned (Fig. 21). Gradually increasing in size, it attains its maximum at the end of a week or ten days, and then slowly diminishes. In the average case the tumour is about the size of a hen's egg, and is oval in form. In marked cases it may be one-third the size of the child's head. To the touch it is soft, elastic, fluctuating, and irreducible. It does not increase with the cry or cough. There is no extra heat and no signs of inflammation. Usually the tumour does not pulsate, although in rare instances pulsating cephalhæmatomata have been seen. Very soon the tumour is surrounded by a marginal ridge. At first this is apparently from coagulation of blood, but later it may be bony. The prominent ridge with the soft centre gives a sensation somewhat like that of a depressed fracture. Sometimes on pressure there is obtained a sort of parchment-crackling. This is generally found as the swelling is subsiding, and is sometimes clearly due to the formation of minute bony plates upon the inner surface of the periosteum. It may be found when there is nothing but thin coagula to explain it. In certain cases following severe traumatism, cephalhæmatoma may be complicated with wounds of the scalp, fracture of the skull, and even lacerations of the dura mater or the brain. In such cases the tumour may become inflamed, but in the spontaneous cases this is extremely rare. The usual signs of abscess develop, which may open externally or burrow. Fortunately this termination is seldom seen.

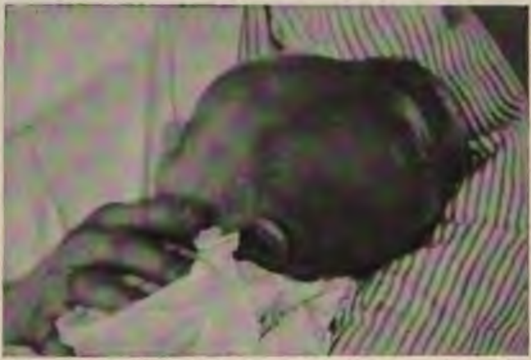
FIG. 21.—Double cephalhæmatoma, infant seven days old.

As a rule, without any interference, the uncomplicated cases go on to recovery. The complete disappearance of the tumour may be expected in from six weeks to three months, depending on its size; but a hard, uneven elevation may remain at its site for a longer time. The cases due to severe traumatism are more serious, the gravity depending not upon the cephalhæmatoma but upon the complicating lesions.

Diagnosis.—Cephalhæmatoma may be confounded with encephalocele. This, however, occurs along the line of the sutures or at the fontanels, is partly reducible, pressure causes cerebral symptoms, and frequently the tumour increases with respiratory movements. Hydrocephalus is distinguished by the symmetrical enlargement of the head, the large frontanels, and the widely separated sutures. Caput succedaneum often appears in the same place as a cephalhæmatoma and at the same time, but is an œdematous, not a fluctuating tumour, is not circumscribed, lacks the hard, marginal border, and begins to disappear by the second or third day. From a depressed fracture of the skull, it is differentiated by the fact that in cephalhæmatoma there is a tumour and not a depression; the prominent margin which is raised above the contour of the skull, is not osseous and the skull can be felt at the bottom of the centre of the tumour.

The *treatment* in the uncomplicated cases is simply protective, all such cases tending to spontaneous recovery. No local or general treatment to promote absorption is required. The child should be so placed and so handled that no injury may be done to the affected part. Compresses are unnecessary. If complications exist, such as injury to the bones, dura, or brain, they are to be treated in accordance with general surgical principles. Operative interference is called for only when suppuration has occurred, or when there are brain symptoms which point to the existence of internal as well as external cephalhæmatoma.

Visceral Hæmorrhages.—While these are most frequent in large children and following difficult labours, they may occur in small children and where the labour has been easy and normal—their occurrence here being due to the feeble resistance of the blood-vessels. From one hundred and thirty autopsies upon still-born children or those dying soon after birth, Spencer concludes that intracranial hæmorrhages are more frequent in head-forceps than in breech cases, and more frequent in breech than in natural vertex deliveries. Other visceral hæmorrhages are much more frequent in breech cases.

Not all visceral hæmorrhages are to be classed as traumatic. They are often seen with the spontaneous hæmorrhages from the skin or mucous membranes. When, however, they are single, they seem to me of traumatic rather than of pathological origin.

The most important of the visceral hæmorrhages are intracranial. These are discussed in the chapter devoted to Birth Paralyses. Rarely there may be large hæmorrhages into the lung. Here the blood fills the air vesicles, the small bronchi, and coagula may be found even in the larger bronchi. A large part of a lobe or an entire lobe may be involved. On section the condition resembles atelectasis, and it may give the physical signs of consolidation.

The abdominal viscera suffer more than those of the thorax because less protected against pressure. Small hæmorrhages are not uncommon upon the surface of any of the viscera covered by peritonæum. Intraperitoneal hæmorrhages are rare, but may be very extensive, amounting to one or two pints. Sometimes no ruptured vessel can be found. The hæmorrhage may be primarily in the peritoneal cavity, or it may result from rupture of one of the viscera, especially the suprarenal capsule. It may be large enough to produce death from loss of blood.

Small surface hæmorrhages of the liver are not infrequent. Occasionally one of considerable size occurs separating the peritoneal covering and forming a tumour generally upon the superior surface. Such laceration may be produced during labour, and a slow accumulation of blood may take place beneath the capsule, death resulting, as in the case reported by Mendelson (New York), from rupture into the peritoneal cavity on the third day. Steffen reports a case of laceration of the capsule of the liver in a still-born infant. Of the large hæmorrhages, those into the suprarenal capsules are perhaps the most frequent. Two cases have recently occurred in the Sloane Maternity Hospital. In one of these, the specimen of which I examined, the capsule was distended nearly to the size of an orange, and the kidney surrounded by a mass of blood-clots. Blood was extravasated into the retroperitoneal connective tissue, and rupture had taken place into the peritoneal cavity, which contained half a pint of partly coagulated blood. The child died on the fifth day. This case has been reported in full by Tuley.* Ahlfeld has reported a case of hæmorrhage into both suprarenals.

Except in the intracranial variety, visceral hæmorrhages cause few symptoms, and in the great majority of cases the diagnosis is not made. Intrapulmonary hæmorrhages have given rise to the signs of consolidation of the lung and even to hæmoptysis (Miram's case). The abdominal hæmorrhages are the most obscure. There may be a general abdominal distention with the usual symptoms of loss of blood, or there may be a circumscribed swelling. In many cases nothing is noticed until a rupture of a subperitoneal hæmorrhage takes place into the general peritoneal cavity, when there may be sudden collapse and death.

The visceral hæmorrhages are not amenable to treatment. The prognosis depends upon the size and position of the hæmorrhage. In the cases of abdominal hæmorrhage the diagnosis is extremely obscure and is rarely made during life.

SPONTANEOUS HÆMORRHAGES—THE HÆMORRHAGIC DISEASE OF THE NEWLY BORN.

A disposition to bleeding is seen with many diseases of the first few days of life, especially those of an infectious character, like syphilis and pyæmia. With most of these, however, the hæmorrhages are small, and the condition may be compared to the hæmorrhagic tendency seen in certain forms of infection of later life, such as measles, smallpox, and malignant endocarditis. There is, however, a class of cases in which the hæmorrhages are not associated with any other known process, and in which the escape of blood from the small blood-vessels is the chief or essential symptom. In these cases the bleeding is much more extensive than in the others mentioned. These hæmorrhages are characterized by the fact that they are spontaneous in origin, having no connection with delivery, they are multiple in location, and, while little influenced by treatment, they tend to cease spontaneously after quite a limited time. They are most often from the umbilicus, the mucous membranes of the stomach and intestines, or beneath the skin, but they may be from almost any mucous surface or into any organ of the body.

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* Archives of Pædiatrics, November, 1892.

Etiology.—Exactly what causes these hæmorrhages is as yet unknown, but it is something which produces changes in the blood or in the blood-vessels, or in both, whereby the vessels are no longer able to hold their contents. In this class, as well as in the traumatic hæmorrhages, the predisposing causes of bleeding in early life must be emphasized—viz., the fragile condition of the blood-vessels and the great changes taking place soon after birth both in the circulation and in the blood itself. These hæmorrhages are not common, and are met with much more often in institutions than in private practice. In 5,225 births in the Boston Lying-in Asylum, Townsend reports 32 cases of hæmorrhage, or 0·6 per cent. In the Lying-in Asylum of Prague, Ritter observed 190 cases in 13,000 births, or 1·4 per cent. In the Foundling Asylum of Prague, Epstein reports hæmorrhages in 8 per cent of 740 infants.

These cases, except in very rare instances, are not manifestations of hæmophilia. Of 576 bleeders collected by Grandidier, only 12 had a history of hæmorrhage at the time of falling off of the cord, and symptoms very rarely appeared before the end of the first year. Hæmorrhages in the newly born are only slightly more frequent in males, while in hæmophilia they predominate 13 to 1. The hæmorrhagic disease of the newly born is self-limited, and runs a definite course to recovery or death. The tendency to bleed does not extend beyond a few weeks, and often lasts but a few days; those who survive, recover perfectly. Circumcision has been done within a few days after the cessation of the hæmorrhages without any unusual bleeding. In a case lately under observation with the most extensive subcutaneous hæmorrhages I have ever seen, all tendency to bleed had ceased before the separation of the cord, although there had previously been bleeding at the navel. A similar case is reported by Townsend. These cases are not associated with difficult delivery. In only 6 of Townsend's * 50 cases was the labour abnormal. This is borne out by my own experience. Many of the children who bleed have previously been anæmic and in poor general condition; but, on the other hand, many have been strong and given every indication of being well nourished. Hereditary syphilis is associated in a small proportion of the cases—from 2 to 6 per cent, according to the observations of Epstein, Ritter, and Townsend. In 132 cases of congenital syphilis observed by Mracek, 14 per cent suffered from hæmorrhages.

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* Archives of Pædiatrics, 1894, p. 559.

A more frequent association with sepsis (pyogenic infection) has been observed. Of the 61 cases observed by Epstein not less than 29, and of the 190 cases of Ritter,* 24 were associated with sepsis. During the year 1895 there were no less than 8 marked cases of hæmorrhage in the Nursery and Child's Hospital in about 225 deliveries. While it is true that more cases of sepsis (pyogenic infection) occurred among the children during this period than usual, it was striking that not one of these hæmorrhagic cases gave any evidence of sepsis, and that none of the septic cases had bleeding. An epidemic of 10 cases of hæmorrhages among 54 births at the New York Infirmary for Women and Children was studied in 1899 by Kilham and Mercelis.† These all occurred in the course of two months; the epidemic ceased as soon as the cases were properly isolated.

From the foregoing facts it is quite evident that not all the cases of bleeding are due to the same cause, and that while this symptom occurs in some cases of pyogenic infection, the latter does not explain most of those seen. The circumstances in which the hæmorrhagic disease occurs point strongly to an infectious origin, but with our present knowledge we can not believe this cause to be the same as in ordinary sepsis—viz., the entrance of common pyogenic bacteria. Quite a number of these cases have now been studied bacteriologically, but with no very uniform results. In two cases by Gaertner ‡ there was found in the blood a short bacillus resembling in some respects the colon bacillus, which, injected into the peritoneal cavity in young animals, chiefly dogs a few days old, produced a disease accompanied by hæmorrhages resembling that seen in the newly born. The bacillus was recovered from the blood and all the organs of these animals. Several observers have confirmed his findings. Other organisms that have been isolated are the streptococcus, staphylococcus, bacillus pyocyaneus, an organism closely resembling the pneumococcus, and several others; but no one of these is constantly present. It seems likely that the specific cause, whatever its nature, produces changes not so much in the blood as in the blood-vessels themselves. Its action seems to be similar to that of a constituent found by Flexner and Nagouchi in rattlesnake venom, which produces rapid destruction of the vascular endothelium, and which has been called by them *hæmorrhagin.*

While these hæmorrhages are not traumatic, bleeding is exceedingly prone to occur in the skin over pressure points such as the back, the elbows, the occiput, and the sacrum. It is also common from the mucous membranes which are the seat of pathological processes, especially from the eyes, the nose, and the genitals.

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* Oesterreiches Jahrbuch für Pädiatrik, 1871, 127.

† Archives of Pædiatrics, March, 1899.

‡ Archiv für Kinderheilkunde, 1895.

Lesions.—In very many of the cases the autopsy shows nothing except the hæmorrhages in the various situations and the blanching of the organs due to the loss of blood. The hæmorrhages of the brain are usually meningeal and diffuse. They are considered more at length in the chapter upon Birth Paralyses. The pulmonary hæmorrhages are usually small and unimportant, amounting only to small extravasations into the substance of the lung or ecchymoses of the mucous membrane of the bronchi. Ecchymoses may be seen upon the surface of the pleura, the pericardium, or the peritoneum, but large hæmorrhages into the pleura or pericardium are very rare. The thymus gland is often the seat of small extravasations. The stomach and intestines may contain considerable blood variously disorganized in the different parts of the canal, and there may be ecchymoses of the mucous membrane. In addition, ulcers may be found in the stomach and duodenum. In twenty-four autopsies upon cases with hæmorrhage from the stomach and intestines collected by Dusser,* ulcers were found in the stomach in nine cases, and in the intestines in four. These ulcers are multiple and are small, resembling the follicular ulcers of the colon. They are usually superficial, but may extend to the muscular coat and may even perforate. I have myself found ulcers in the stomach in a single case. They were associated with a moderate amount of follicular gastritis. The intestinal ulcers are found only in the duodenum and resemble those of the stomach. The cause of these ulcers is somewhat obscure; some of them are undoubtedly dependent upon inflammatory changes probably of infectious origin; others have been compared to the peptic ulcers of later life, and are attributed to thrombi in the blood-vessels of the mucous membrane. These ulcers are found in but a small proportion of the cases in which bleeding occurs from the alimentary tract, and they may be wanting even where it has been very profuse.

Small extravasations may be seen upon the surface of the liver, the spleen, or the kidneys. They may also be found in the substance of these organs. The large hæmorrhages upon the surface of the liver, into the suprarenal capsules and other subperitoneal extravasations have been included, improperly perhaps, in the group of traumatic hæmorrhages discussed in the preceding chapter. From a rupture of any of these there may be large extravasations into the peritoneal cavity. Microscopical examinations of the blood-vessels have been made in but a small number of cases. Mracek claims to have found evidences of endarteritis in some of the syphilitic cases in which there was bleeding. The changes found in the blood have not been uniform and have as yet been only imperfectly studied. The associated lesions found are most frequently those due to sepsis.

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* Thèse, Paris, 1889.

Symptoms.—The time of beginning is most frequently in the first week of life, rarely after the twelfth day, although it has been observed as late as the sixth week. As a rule, the hæmorrhages from the stomach and intestines begin earlier than those from the navel or the skin. The location of the hæmorrhage in Ritter's 190 cases was as follows: Umbilicus, 138 (umbilicus alone, 97); intestines, 39; mouth, 28; stomach, 20; conjunctivæ, 20; ears, 9. In Townsend's 50 cases: Intestines, 20; stomach, 14; mouth, 14; nose, 12; umbilicus, 18 (umbilicus alone, 3); subcutaneous ecchymoses, 21; abrasion of skin, 1; meninges, 4; cephalhæmatoma, 3; abdomen, 2; pleura, lungs, and thymus, 1 each.

In many cases nothing is noticed until the hæmorrhage begins. The child may be previously healthy or feeble. The first bleeding noticed may be from the stomach, intestines, or any of the mucous surfaces, beneath the skin, or from the umbilicus. The amount of blood lost in most cases is not great, but there is a continuous oozing. The total hæmorrhage may be only one or two drachms or it may reach several ounces. The skin is usually pale, the pulse feeble, and the general condition one of considerable prostration, often from the outset. In all cases there is rapid loss of weight. The temperature may be high, low, or subnormal. A marked elevation of temperature may depend not upon the hæmorrhage but upon associated conditions. Fluctuations in temperature during the first three days are so common from disturbances of nutrition, that I attach much less importance than have some writers to this symptom. Icterus is not more frequent than among other infants. In a large number of the cases there is diarrhœa. Convulsions often occur at the close of the disease.

The duration of the disease in cases which recover is usually but one or two days. In fatal cases it is rarely more than three days, and often less than one. Death more frequently results from the gradual failure of all the vital forces than from a rapid loss of blood.

Umbilical hæmorrhage.—A slight oozing from the umbilicus not infrequently occurs when the ligature has been improperly applied, or when there is so much shrinking of the cord that the ligature has loosened. Sometimes rough handling at the time of the separation of the cord may excite a little bleeding. All the above conditions, however, are usually of trivial importance and are readily controlled by simple measures. Spontaneous hæmorrhage is quite a different matter. It is rather later than bleeding from the mucous membranes, usually occurring between the fourth and the seventh day. There may be bleeding into the cord as well as from its free extremity before it separates; after separation, from the stump. A slight stain upon the dressing is usually the first note of warning, but in exceptional circumstances a gush of blood is the first symptom. The hæmorrhage may be temporarily arrested by various means, but it shows a strong tendency to occur in spite of everything which is done. The general symptoms depend upon the amount of bleeding and the rapidity with which it occurs. It is the same as in other hæmorrhages of the newly born. The usual duration is two or three days. It has been known, however, to persist for twelve or fourteen days, and it may be fatal in less than twenty-four hours from the time it is noticed.

Hæmorrhage from the stomach and intestines.—Bleeding occurs much less frequently from the stomach than from the intestines. The latter is called melæna. Gastro-enteric hæmorrhages begin, in the great majority of cases, during the first three days of life. Of Dusser's 75 cases, the hæmorrhage began on the first day in 24 cases; on the second day in 22 cases; on the third day in 9 cases; in only 10 cases later than the ninth day, and in no instance later than the twelfth day. The appearance of the blood vomited depends upon the length of time it has remained in the stomach. Usually it is in dark brown masses, and not very abundant; more rarely bright red blood may be ejected. The quantity varies from one drachm to half an ounce. Vomiting is liable to be excited by nursing. The blood discharged from the bowels is always dark coloured, usually intimately mixed with the stool, very rarely in clots. If in doubt between blood and meconium, one should look for the corpuscles with the microscope. When this is not conclusive on account of the disorganization of the corpuscles, a chemical test for hæmoglobin should be made. Concealed hæmorrhage into the stomach may take place, which may even be sufficient to produce death, no blood being vomited or passed by the bowels. In such cases the autopsy may reveal quite a large quantity of blood, both in the stomach and intestines.

Hæmorrhage from the mouth.—The quantity of blood is rarely large; but it is here that it is often first seen. Its source may be the mucous membrane of the mouth, pharynx, œsophagus, stomach, or bronchi. It may be associated with ulceration of the hard palate, with thrush, or with fissures of the lips.

Hæmorrhages from the nose are infrequent, and are more often due to syphilis than to other causes. These are rarely profuse, but are frequently repeated.

Subcutaneous hæmorrhages.—These may appear in places exposed to pressure, such as the sacrum, heels, occiput, or back; or in others which are not so exposed, as the abdomen, axillæ, or thighs. They may follow other lesions of the skin, such as pemphigus, eczema, or furunculosis. In some cases these hæmorrhages are very extensive, as in one recently under observation, where nearly one third of the thorax was covered. The extravasations are surrounded by an indurated border. Where they occur alone or form the principal lesion, the prognosis is favourable.

Hæmaturia.—The urine is not only stained with blood, but sometimes contains clots. This hæmorrhage may have its origin in the bladder, urethra, or kidney. Blood coming from the kidney is sometimes due to the irritation of uric-acid infarctions, and may have nothing to do with the general hæmorrhagic disease.

Hæmorrhage from the conjunctiva.—The blood usually comes in drops from between the eyelids, chiefly from the tarsal surface. It is generally preceded by conjunctivitis.

Hæmorrhage from the ears may originate in the external meatus or the middle ear. It is generally preceded by otitis.

Hæmorrhage from the female genitals.—This not infrequently occurs without hæmorrhages elsewhere, and under such circumstances is rarely serious. Cullingsworth has collected thirty-two cases in children under six weeks of age—no case having resulted fatally. These are not to be regarded as cases of precocious menstruation. They are frequently preceded by catarrhal inflammations of the vagina.

Diagnosis.—This is generally easy, as the hæmorrhages are usually multiple and some of them external. A slight hæmorrhage from the intestine may be easily overlooked. Large hæmorrhages into the internal organs also are obscure and not often recognised. Spurious hæmorrhages from the stomach may occur, blood being vomited which has been swallowed during birth or nursing. The source of bleeding may also be the mouth, nose or pharynx, and sometimes blood is swallowed in large quantities and afterward vomited. These cavities should therefore always be examined, since local treatment may be efficacious. Syphilis should be suspected when the bleeding is chiefly nasal.

Prognosis.—In all circumstances the hæmorrhagic disease in the newly born has a bad prognosis. Of seven hundred and nine cases collected by Townsend, the mortality was seventy-nine per cent. No observer has seen more than one third of his cases recover. In any single case the prognosis depends upon the extent and severity of the hæmorrhage, upon the vigour of the child, and upon how well it can be nourished. No case should be looked upon as hopeless, for perfect recovery has repeatedly taken place where it seemed impossible.

Treatment.—Thus far no treatment seems to have any decided influence in controlling this disease. Adrenalin and the suprarenal extract appear to have some effect in bleeding from accessible mucous membranes, and should be applied if the hæmorrhage is from the nose, mouth, or pharynx. For internal use the suprarenal extract is to be preferred. I have seen one case in which benefit seemed to follow its use in severe gastric hæmorrhage, but in others it has failed entirely. It may be given up to two grains every two hours. The subcutaneous injection of a two-per-cent solution of gelatin, which has been sterilized several times, is advocated by many European writers; 40 to 50 cc. may be administered two to three times daily. The general treatment should have reference to maintaining the nutrition by careful feeding, judicious stimulation, and attention to the circulation, the body temperature, and the general condition of the child. Bleeding points on the skin or mucous membranes within reach are best treated by the application of chromic acid fused on a probe, or of nitrate of silver. Umbilical hæmorrhage is best controlled by covering the umbilicus with a small pad of sterile cotton, over which is folded from either side the skin of the abdominal wall. This is held in place by two strips of adhesive plaster crossing the umbilicus obliquely. Astringent injections for intestinal hæmorrhages are practically useless, as the blood is almost invariably either from the stomach or from the upper part of the small intestine.

CHAPTER VI.

BIRTH PARALYSES.

BIRTH paralyses are chiefly due either to pressure upon the child by the parts of the mother or to artificial means employed in delivery. They may be cerebral, spinal, or peripheral.

Cerebral paralyses are in almost every instance due to meningeal hæmorrhage. Very infrequently they depend upon cerebral hæmorrhage, laceration of the brain, or pressure from a depressed fracture.

Spinal paralyses are extremely rare, and only a few examples are on record. They are due to laceration of, or hæmorrhage into the cord or its membranes. These lesions produce paraplegia, the exact distribution of which depends upon the point at which the cord is injured.

Peripheral paralyses usually affect the face or the upper extremity. Paralysis of the face is due in most cases to the application of the forceps. Paralysis of the upper extremity is most frequently of the "upper-arm type," and is known as Erb's paralysis. It usually follows extraction in breech presentations. Peripheral paralysis of the lower extremity is almost unknown.

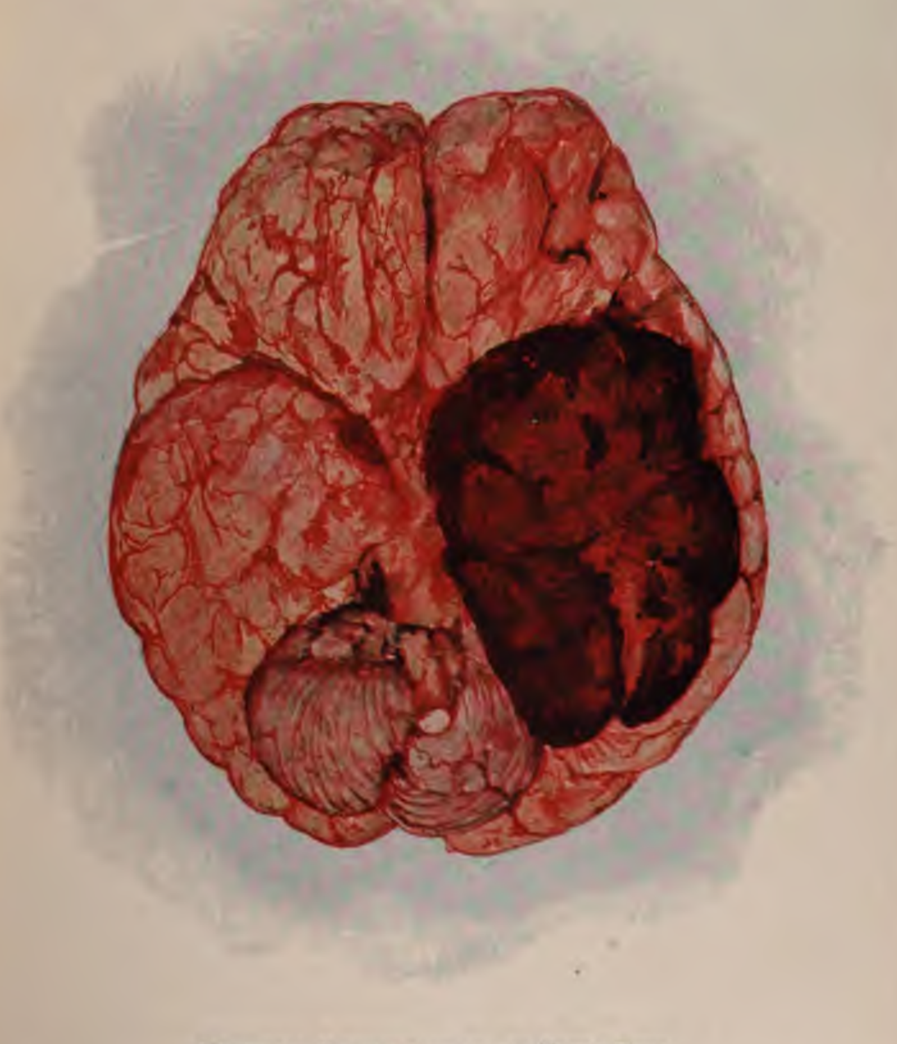
CEREBRAL PARALYSIS.

Cerebral paralysis is often used synonymously with meningeal hæmorrhage. This lesion is not infrequent, and is of great importance not only from its immediate effects, but because upon it depend many of the cerebral paralyses seen in later life. According to Cruveilhier, at least one third of the deaths of infants which occur during parturition are due to this cause.

Etiology.—The same predisposing causes exist in the cases of meningeal hæmorrhages as in others occurring at this time. A small number of cases are associated with syphilis; others with pyogenic infection. In a few cases there is a history of an injury—usually a fall or blow upon the abdomen—during the last months of pregnancy. Meningeal hæmorrhage may occur as one of the lesions in the hæmorrhagic disease of the newly born. The most important causes, however, are connected with parturition. These hæmorrhages are essentially mechanical, and are favoured by everything which increases or prolongs pressure upon the head. The conditions with which they are associated are tedious labour, breech presentations with difficulty in extracting the head, instrumental deliveries, and premature births. The majority occur in first-born children. Certain cases are associated with cardiac malformations—according to Bednar, a small aorta with hypertrophied heart, or the transposition of the large blood-vessels. In many of the cases there is also a hæmorrhage outside the skull.

Lesions.—These hæmorrhages are very much more common at the base than at the convexity, and at the posterior, than at the anterior part of the skull. They are most frequently found over the cerebellum and the occipital lobes of the cerebrum. The entire extravasation is often beneath the tentorium. The extent of the hæmorrhage is exceedingly variable. There may be a single large clot at the convexity or at the base (Plate II), the hæmorrhage may be limited to the convexity of one hemisphere, or it may cover nearly the entire surface of the brain. Diffuse hæmorrhages are more common than a single circumscribed clot. Of the eleven cases collected by McNutt (New York), in seven cases with vertex presentations the lesion was principally at the base, and usually limited to that region. In four breech cases, however, it was principally at the convexity. The source of the blood may be a laceration of one of the sinuses of the dura mater caused by the overlapping of the parietal bones. This was found in one of the cases of Hirst (Philadelphia). Much more frequently the blood comes from one of the cerebral veins, or from the capillary vessels of the pia mater. In thirty-seven of Bednar's fifty-two cases the extravasation was beneath the pia mater. In the remainder it was between the pia mater and the dura—i. e., in the arachnoid cavity. Hæmorrhages between the dura and the skull may be said never to occur except when associated with fracture. If the child is still-born, or if death has occurred on the first or second day, the blood is partly fluid and partly coagulated; later it is entirely coagulated and may have undergone partial absorption. The amount of extravasated blood varies between one drachm and four ounces, the average amount being about one ounce. The blood extends into the fissures between the convolutions and sometimes into the ventricles along the choroid plexus, although this is rare. In large hæmorrhages the brain substance is softened and in places may be quite disintegrated; but with small extravasations these changes are very slight. In cases which survive for two or three weeks there is usually a certain amount of meningitis. The later consequences—those of arrested development of the cortex and cerebral sclerosis—will be considered in the chapter devoted to Cerebral Pa-

PLATE II.



Meningeal Hæmorrhage in the Newly Born.

From a patient in the Nursery and Child's Hospital, dying on the sixth day. Primary respirations poor; child very dull and apathetic, refused to nurse; once vomited blood and had an ecchymosis of the right conjunctiva. On the last day, high temperature (105° F.) and general convulsions. Some changed blood found in the stomach and intestines at the autopsy; brain greatly congested, and at the base was the clot shown in the picture.

ralyses in the section on Diseases of the Nervous System. Hæmorrhages into the membranes of the upper part of the cord are found in a large proportion of the fatal cases. Associated hæmorrhages of the lungs and other organs are not uncommon.

Symptoms.—If the hæmorrhage is large, the child is usually still-born, although its movements may have been active up to the commencement of labour. When the hæmorrhage is not so large as to be immediately fatal, the child may show no symptoms except dulness or torpor, with feeble or irregular respiration, death following within the first twenty-four hours. A large proportion of the cases are born asphyxiated, and frequently they are resuscitated only after considerable effort. They nurse feebly, often with great difficulty. Convulsions are common in cases which last for four or five days, and more with hæmorrhages at the convexity than with those at the base. Opisthotonus is often present, also general rigidity of the extremities, clenching of the hands, and increased knee-jerks. Rarely there is complete relaxation of all the muscles. Sometimes there are automatic movements. The respiration is usually disturbed; in most cases it is slow and irregular. The pulse is feeble and slow. The pupils are more frequently contracted than dilated, and there may be oscillation of the eyeballs. In large hæmorrhages there is marked bulging of the fontanel, and often separation of the sutures. If the hæmorrhage covers one hemisphere, there is complete hemiplegia of the opposite side. Small localized cortical hæmorrhages may cause paralysis of the face, arm, or leg, according to the position of the lesion, or localized convulsions. In large hæmorrhages at the base convulsions are rare, and death occurs early, usually in the first two days. In extensive cortical hæmorrhages convulsions and rigidity of the extremities are frequent, and life is prolonged indefinitely.

The majority of the fatal cases die within the first four days. In those lasting a longer time the symptom is tonic spasm of the trunk, or of one or more of the extremities, with localized paralysis—monoplegia, diplegia, or hemiplegia, according to the lesion—and localized or general convulsions often continuing for two or three weeks and gradually subsiding. In the mildest cases nothing abnormal may be noticed until the child is old enough to walk or talk. In those more severe there may be gradual and continuous improvement of the early symptoms, and the case may go on to apparent recovery, but usually there is some permanent damage to the brain. The following observation of McNutt illustrates the course and termination of one of the severe cases of meningeal hæmorrhage:

Breech presentation, tedious labour, head delivered by forceps, almost continuous convulsions for the first nine days. After the convulsions there was complete paralysis of both sides of the body, not involving the face. The child never walked or spoke; the physical development was very backward; the limbs became contractured; death occurred at two and a half years, from pneumonia. The autopsy showed atrophy of the brain on both sides about the fissure of Rolando.

The main diagnostic symptoms in recent cases are stupor, rigidity, increased reflexes, convulsions, paralysis, and opisthotonus. These vary with the extent and situation of the lesion. Other symptoms are changes in the pupils, oscillation of the eyes, and bulging fontanel.

Prognosis.—A large hæmorrhage at the base quickly causes death; if it is located at the convexity, although the child may survive, there is always serious damage to the brain. Even from small hæmorrhages some permanent injury usually results, though the extent of this may not be evident for years.

Treatment.—This is mainly prophylactic, the chief indication being to shorten tedious labours by the early use of the forceps. Where the hæmorrhage has been attributed to the forceps, the damage has rather been the result of the long-continued pressure before they were used. Nothing can be done after delivery to limit the amount of the hæmorrhage, except to keep the child as quiet as possible. The removal of the clot by surgical operation has twice been successfully accomplished by Cushing (Baltimore). With more accurate diagnosis there seems to be no reason why a considerable number may not be saved. The hopeless outlook for such cases when not relieved, justifies the taking of great risks.

FACIAL PARALYSIS.

The usual cause of facial paralysis is the use of the forceps, but this does not explain all the cases. The etiology of those in which the forceps have not been used is still somewhat obscure. In peripheral facial palsy the nerve is pressed upon, either near its exit from the stylo-mastoid foramen, or where it crosses the ramus of the jaw, at which point the parotid gland gives it but little protection in the newly born. If the lesion is in front of this point, any one of the terminal branches may be affected; most frequently it is the temporo-facial branch. As only one blade of the forceps commonly touches the face in this region, the paralysis is, as a rule, unilateral.

Roulland has reported several cases not due to the forceps. In these the pressure is believed to have been produced by the promontory of the sacrum at the superior strait, or by the ischium at the inferior strait, as paralysis followed when the head was long arrested at one of these points. It was not seen with face or breech presentations. When facial paralysis is of central origin it depends generally upon a meningeal hæmorrhage, and the arm and leg of the same side as the face are involved. It is, however, possible for a very small cortical hæmorrhage to produce paralysis of the face only. This occurred in a case reported by McNutt.

In repose, the only symptom noticed may be that the eye remains open upon the affected side, owing to paralysis of the orbicularis palpebrarum.

When the muscles are called into action, as in crying, the whole side of the face is seen to be affected. The paralyzed side is smooth, full, and often appears to be somewhat swollen. The mouth is drawn to the side not affected. In this paralysis, the tongue, of course, is not implicated. It is therefore rare that nursing is seriously interfered with.* If the paralysis is of central origin, only the lower half of the face is involved, while in peripheral paralysis, as the trunk of the nerve is injured, the upper half of the face, including the orbicularis palpebrarum, is also affected.

The paralysis is generally noticed on the first or second day of life, and does not increase in severity. Its course and termination depend upon the extent of the injury done to the nerve. Some idea of this may often be gained by the amount of injury to the soft parts, although this is not an infallible guide. In cases not due to the forceps, the paralysis is slight and disappears in a few days; the great majority of the forceps cases follow the same favourable course, the paralysis gradually disappearing without treatment in about two weeks. In more serious cases it may last for months, or it may even be permanent. The reaction of degeneration is present in these severe cases, and there may even be perceptible atrophy of the muscles. This symptom is fortunately extremely rare.

Treatment.—Nothing should be done for the first ten days except to protect the eye and keep it clean. If improvement has begun by the end of this time, the probabilities are that the case will require no treatment. If no improvement has taken place by the end of the third or fourth week, electricity should be used regularly and systematically. If the muscles respond to it, the faradic current may be employed; if not, galvanism should be used. The electrical treatment should be continued for several months, or until recovery has taken place.

PARALYSIS OF THE UPPER EXTREMITY.

When this is due to a peripheral lesion it probably never involves the entire arm, but affects only certain muscles or groups of muscles. Although commonly occurring after an artificial delivery, it may be seen in cases where the labour has terminated naturally. Roulland † has reported a case in which deltoid paralysis, occurring in a large child, was attributed to pressure upon the shoulder during labour. In vertex presentations, paralysis is most frequently due to the forceps where one of the blades has extended down upon the neck, injuring the lower cervical nerves. It may be produced by traction with the finger in the axilla. Roulland reports a unique case of paralysis of both extremities, apparently due to the cord being very tightly wound around the neck. The great proportion of all cases of paralysis of the upper extremity follow extraction in breech presentations. The injury is usually inflicted by traction upon the shoulder in the delivery of the head, or in bringing down the arms when they are above the head. In the latter case the paralysis may be double and associated with fracture of the clavicle or humerus. In shoulder presentations, paralysis may be produced by traction upon the arm itself.

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* In this connection it is to be remembered that the principal part in nursing is done by the tongue, and not by the lips.

† Paralysies des nouveau-nés, Paris, 1887.

The most common form of peripheral paralysis is that known as the "upper-arm type," or Erb's paralysis, in which the injury is inflicted at the anterior border of the trapezius muscle at the lower part of the neck, usually in such a position as to affect the fifth and sixth cervical nerves. The muscles paralyzed are the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the supra- and infra-spinatus. All these muscles may be involved, or only part of them, and in varying degrees. In case the injury is slight, the paralysis may not be noticed for some weeks. If severe, it is evident in the first few days. The arm hangs lifeless by the side; it is rotated inward, the forearm pronated, the palm looking outward (Fig. 22). The forearm and hand are not affected. In severe cases there may be anæsthesia of the outer surface of the arm, in the region supplied by the circumflex and external cutaneous nerves. This is rarely marked, and in its slighter degrees it is very difficult to determine. It is characteristic of this paralysis that the triceps is not affected, so that power to extend the forearm remains, although it cannot be flexed. Atrophy of the paralyzed muscles occurs after a few weeks, but the muscles are so small and so covered with fat that it is rarely noticeable before the second year. It is most conspicuous in the deltoid. In all severe cases the reaction of degeneration is present. In some of the cases of long standing there occurs a shortening of the tendon of the subscapularis muscle, often associated with subluxation of the humerus. The paralysis may be complicated with fracture of the clavicle, the neck of

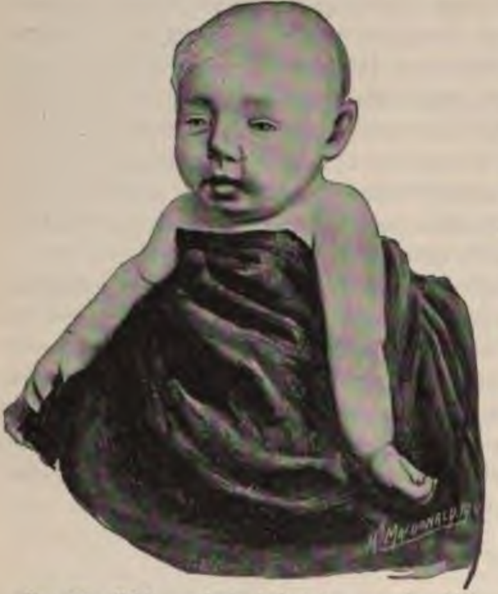

FIG. 22.—Erb's paralysis, infant two months old.

the scapula, or the shaft of the humerus, or with epiphyseal separation of its head.

The *prognosis* depends upon the severity of the injury and also upon the time when treatment is begun. The great majority of cases recover spontaneously in two or three months, improvement being observed within a few weeks, first in the biceps and last in the deltoid. Spontaneous recovery is not to be looked for unless it occurs within the first three months. Not infrequently some degree of paralysis persists until the third or fourth year, and in some of the muscles, usually the deltoid, it may even be permanent. If the muscles respond to faradism, rapid improvement can generally be prophesied. If the reaction of degeneration is present, improvement will be slow and the paralysis may be permanent.

The *diagnosis* is usually not difficult, since the great majority of cases are of the "upper-arm type" with classical symptoms. Peripheral palsy of the arm can scarcely be confounded with that of cerebral origin. If the lesion is central it is one of the rarest occurrences for the arm alone to be involved; either the leg or face, or both, are generally likewise affected. If the case does not come under observation until the child is a year old, it may be difficult, or without a good history, it may be impossible to distinguish peripheral paralysis from that due to polio-myelitis. The peculiar group of muscles involved in Erb's paralysis is the only diagnostic point.

In recent cases the disability resulting from the tenderness or pain of syphilitic epiphysitis may simulate paralysis, but there is lacking the characteristic position of the arm, and a careful examination discloses the fact that the paralysis is only apparent. This may affect both sides. Fracture of the clavicle or epiphyseal separation of the head of the humerus may also be mistaken for paralysis. In cases of long standing, paralysis of the deltoid may resemble dislocation of the humerus. The reaction of degeneration differentiates paralysis from surgical injuries with similar deformities.

The *treatment* consists in the use of electricity, which should be begun at the end of the first month at the latest, and used regularly. If the muscles respond to faradism this may be employed, but in most severe cases they do not, and galvanism must be used, according to the rules laid down for facial paralysis.

CHAPTER VII.

TUMOURS OF THE UMBILICUS.

Granuloma.—This is nothing more than a mass of exuberant granulations at the umbilical stump. The mass is generally about the size of a pea—sometimes larger—bleeds readily, and has a thin, purulent discharge.

It is promptly cured by the application of any simple astringent; powdered alum is probably the best. In case this is not successful, the granulations may be touched with nitrate of silver or snipped off with scissors.

Adenoma, Mucous Polypus, or Diverticulum Tumour—Umbilical Fistula.—The first three terms are used synonymously to describe an umbilical tumour covered with a mucous membrane which is similar in structure to that of the small intestine. It is usually associated with an umbilical fistula. This tumour is formed by a prolapse at the navel of the mucous membrane of Meckel's diverticulum. This diverticulum is the remains of the omphalo-mesenteric duct. When it is present in infants, it is found in various stages of development. Most frequently there is a blind pouch a few inches long given off from the lower part of the ileum. In other cases it may remain patent quite to the umbilicus, causing a fæcal fistula (Fig. 23, A). As the intestine below it is generally normal, this fistula may persist for months or even years, giving rise to no symptoms except a slight fæcal discharge from the umbilicus. In certain cases intestinal worms have been discharged through it. It may close spontaneously or be closed by operation.

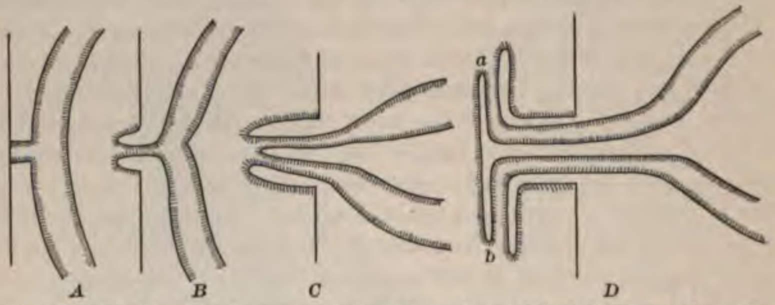

FIG. 23.—Umbilical fistula and tumours produced by prolapse of Meckel's diverticulum. (Barth.)

A prolapse of the mucous membrane lining the diverticulum produces an umbilical tumour with a fistula at its summit (Fig. 23, B). This is the most common form. A cross-section shows under the microscope the structure of the intestinal mucous membrane both as an external covering and lining of the fistulous tract. The prolapse may involve not only the mucous membrane but the entire intestinal wall. There then exists a conical tumour with a fistula which has but one external opening, but at a short distance from the surface it bifurcates, one branch leading upward and one downward (Fig. 23, C). A continuation of the prolapse gives a broad pedunculated tumour (Fig. 23, D), which may reach the size of a man's fist. Its covering is the same as in the other forms. It may contain several coils of intestine. In this form there are usually two fistulous openings (*a*, *b*) which communicate with the intestine.

In all of these cases the tumour is smooth, irreducible, of a rosy pink colour, and from its surface there oozes a mucous discharge. Microscopical examination shows the external covering to be the same in structure as the intestinal mucous membrane. These tumours are generally small, varying in size from a pea to a small cherry, but they may be very much larger. A fæcal fistula usually, but not invariably, coexists. In the condition represented in Fig. 23, B, it is easy to see how an obliteration of the fistula may occur. The small tumours are readily cured by the ligature. The larger ones are usually associated with other serious malformations of the intestines, which make the outlook bad in almost every instance.

UMBILICAL HERNIA.

Hernia into the umbilical cord is a rare congenital condition of a most serious nature. It is due to some fœtal defect, and varies in size from a small protrusion to complete eventration in which nearly all the abdominal organs are outside the body. There is no hernial sac. The prognosis is very bad.

The common umbilical hernia is quite a different condition, and while a source of much annoyance it is rarely serious. It is much more common in females than in males, and occurs especially in those who are poorly nourished and rachitic. The tumour is usually from one-fourth to one-half an inch in diameter; it may, however, be very large, and may even become strangulated, when a surgical operation may become necessary. The ordinary cases, however, require only mechanical treatment. The most important thing is prevention. For this purpose it is necessary, after the cord has separated, to place a firm pad over the navel, and to use a snug abdominal band for the first two or three months. After this period it is uncommon for hernia to develop. In cases coming under observation after the third or fourth month, the pad and abdominal bandage are inadequate, and other means must be employed to retain the hernia. The best of these consists in the use of two adhesive strips applied obliquely over the abdomen, crossing at the umbilicus, the skin along the median line being folded inward so as to overlap the tumour, this forming the retention pad. A simple method of retention is to place over the tumour a coin or button covered with kid and hold it in position by a strip of adhesive plaster ten or twelve inches long. If the skin is made absolutely clean and zinc-oxide plaster used, excoriations are rare. The dressing should be changed every few days and worn for several months. After the first year all mechanical treatment is unsatisfactory. For the very small tumours it is really unnecessary to use any form of apparatus, since these cases ordinarily show little or no tendency to increase in size, and the retention apparatus causes more annoyance than the hernia. These small herniæ seem to disappear spontaneously during childhood, as they certainly are not often seen in children over seven years of age.

MASTITIS.

According to Guillot, a certain amount of secretion in the breasts of the newly born is physiological. It is certainly very common. It is most abundant between the eighth and fifteenth days, but may continue in small quantities as late as the third month. It is seen with equal frequency in both sexes. The quantity of the secretion amounts in most cases only to a few drops; in some, however, as much as a drachm has been obtained. Chemical analysis has shown this secretion to be essentially the same as the adult milk—containing fat, sugar, proteids, and salts. In gross appearance it resembles colostrum. The researches of Sinéty * have shown that the mammary gland of the newly born contains cul-de-sacs lined with secreting cells, resembling those of the adult. During the period of secretion the gland is slightly reddened, its vessels turgid, and all the signs of functional activity are present. This condition in itself is of no practical importance, and in most cases, if left alone, the secretion ceases spontaneously after a week or ten days. If abundant, it can usually be dried up by painting the gland with tincture of belladonna. It sometimes happens, however, that the presence of this secretion tempts the nurse or attendant to rub or squeeze the breast. Such manipulation occasionally leads to serious results by exciting a mastitis which may terminate in abscess. Mastitis is not a very rare condition, and although the inflammation is not usually severe, it may be serious and even fatal. The predisposing cause is the congestion which accompanies functional activity, usually in the second week. The exciting cause is most often some form of traumatism—undue pressure, the squeezing of the breasts, or rough handling by the nurse. Through abrasions or fissures thus produced, micro-organisms find a ready entrance with the same result as in the adult. It seems possible that the germs may enter through the lactiferous ducts without any abrasion of the skin. Want of cleanliness is always a favourable condition for such infection.

The symptoms of mastitis usually begin during the second week of life. There are redness, swelling, and the usual signs of inflammation, which may terminate in resolution or in suppuration. The process may be limited to the mammary region, or a diffuse phlegmonous inflammation may be set up, as in a case reported by Bush,† in which there was extensive sloughing of the tissues of the whole of one side of the chest, with a fatal result. In the great majority of cases the process does not reach this degree of intensity, but suppuration with the formation of single or multiple abscesses is not uncommon. In the female it is possible for the cicatrization which follows such an inflammation to interfere with the sub-

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* Gazette Médicale, No. 17, 1885.

† New York Medical Journal, March, 1881.

sequent development of the gland. The general symptoms are restlessness, loss of sleep, disinclination to nurse, and loss of weight. In cases of diffuse phlegmonous inflammation the general symptoms are those of pyogenic infection. Jourda* has collected fifteen cases of mammary abscess, twelve of which recovered. They began between the fourth and the forty-second days. In eleven cases, only one side was involved; in four, both sides.

Mastitis is usually due to want of cleanliness or to meddlesome interference; the parts should therefore be kept scrupulously clean, and on no account should squeezing of the breasts be permitted. They should be protected by a simple cotton pad. If acute inflammation develops, it should be treated in the beginning by hot applications. Should pus form, early incision with free drainage and general tonic and stimulant treatment are indicated.

INTESTINAL OBSTRUCTION.

The most frequent causes of intestinal obstruction in the newly born are malformations of the intestine; rarely it may be due to pressure from tumours, or from a persistent omphalo-mesenteric duct or artery. The various pathological conditions present in intestinal malformations are considered in the chapter on Diseases of the Intestines. The most common seat of obstruction is at the anus, the bowel being normally formed throughout, lacking only the external orifice. The next most frequent condition is obstruction in the rectum, which may be due either to a membranous septum in the gut, or to obliteration of the tube for some distance. These rectal obstructions are readily recognised. By the examining finger or a bougie the lower limit of the obstruction can be made out, but there is no means by which the upper limit can be determined except by opening the abdomen. When the obstruction is above the rectum, localization is more difficult; but the most frequent seat is the duodenum. Of 38 cases collected by Gaertner, the seat of obstruction was the duodenum in 19 cases, the jejunum in 3, the ileum in 11, the colon in 6, the ileum and colon in 1. There is often obstruction at more than one point.

The symptoms vary with the seat and the degree of the obstruction. In atresia of the anus or rectum there is at first simply an absence of all discharges from the bowel. Later there is abdominal distention from dilatation of the sigmoid flexure and colon. After several days vomiting begins. If there is atresia of the duodenum or any part of the small intestine, vomiting begins early—usually by the second day of life—and it is persistent. Nothing is passed from the bowels after the first dark discharge of the contents of the colon, which is chiefly mucus. There is rapid asthenia, and death from inanition usually occurs in four or five days. The higher the obstruction the shorter the duration of life. If the condition is one of stenosis only, the symptoms are similar to those described but less severe, and life may be prolonged for several weeks, or even months. The constipation in these cases is not absolute. When the cause of obstruction is external pressure, the symptoms do not always begin immediately after birth. I have recently seen a child in whom nothing abnormal was noticed for the first three weeks, but at the end of that time there developed all the signs of acute intestinal obstruction. Laparotomy revealed a loop of intestine constricted by a tiny cord, which was probably the remains of the omphalo-mesenteric duct.

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* Thèse, Paris, 1889.

Cases of imperforate anus and membranous septum in the rectum are readily relieved by proper surgical treatment. In the other varieties of obstruction, whether in the rectum, in the colon, or in the small intestine, although life may be prolonged by the formation of an artificial anus, the ultimate result is almost invariably fatal, death usually occurring from marasmus during the early weeks of life.

DIAPHRAGMATIC HERNIA.

This is due to a congenital deficiency in the diaphragm, which is usually on the left side. Of 118 cases collected by Livingston, 83 were on the left side, 18 on the right, 4 were central, 2 were double, in 1 the diaphragm was absent. With small openings only a single coil of intestine, with large ones a considerable part of the abdominal contents, may be found in the thorax. This causes displacement of the heart, usually to the right side, prevents the full expansion of the left lung, and if the deformity occurs early in intra-uterine life the lung may remain rudimentary. If a large deficiency exists, infants may live but a few hours; with smaller ones, life may be prolonged indefinitely. Booker's * patient lived two and a half months with nearly all the small intestine and omentum and the transverse colon in the thorax; and Northrup's † patient, who died at three years and a half of intercurrent disease, had several coils of the ileum, the cæcum, and the appendix in the chest.

The symptoms are in all cases obscure, the only frequent one being dyspnœa, sometimes constant, sometimes in severe paroxysms resembling asthma, these being apparently produced by an accumulation of gas in the thoracic part of the intestine. The physical signs are those of pneumothorax, generally on the left side, with displacement of the heart to the right. The condition is not amenable to treatment.

SCLEREMA.

Sclerema is a condition characterized by hardening of the skin and subcutaneous tissues. It may occur in circumscribed areas or extend over nearly the entire body. It affects infants who are very feeble and usually terminates fatally. Although sclerema is chiefly seen in the first days of

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* Archives of Pædiatrics, vol. xiv, p. 649.

† Ibid., vol. ix, p. 130.

life, it is not limited to the newly born, but may occur at any time during the first few months. It is not to be confounded with œdema of the newly born, with which condition it is, however, sometimes associated. From published reports it appears to be of not very infrequent occurrence in Europe, chiefly in large foundling asylums. In America, sclerema is an extremely rare disease. In a discussion in the American Pædiatric Society, in 1889, following the report of a case by Northrup, scarcely a dozen cases could be recalled by the members present. I have seen but five cases. In the newly born, sclerema affects those who are premature or very feeble, sometimes those who are syphilitic. Later it may follow any condition leading to extreme exhaustion, especially the different forms of diarrhœal disease.

The first thing to attract attention is usually the induration of the skin. It is often seen first in the calves or the dorsum of the feet, sometimes first in the cheeks, but soon extends over the greater part of the body. It is especially marked in the cheeks, buttocks, thighs and back, and regions where adipose tissue is abundant. It may affect the body uniformly or in circumscribed areas. The skin may be smooth or it may appear somewhat lobulated. The colour is normal or slightly bluish, often tinged with yellow. The lips are blue, and the capillary circulation so feeble that after pressure upon the nails the blood returns slowly or not at all. The limbs are stiff and board-like. The skin is cold to the touch, and often the thermometer in the axilla will not rise above 90° F. In cases reported by Roger and Parrot, an axillary temperature of 71° F. was recorded. The general feeling of the body has been well likened by Northrup to that of a half-frozen cadaver. The tongue and the mucous membrane of the mouth are cold; no radial pulse can be felt; the respiration is slow, irregular, embarrassed, and at times the movements of the thorax are scarcely perceptible. The cry is a feeble whine, scarcely audible. The duration of the disease is usually from three to four days. Death occurs slowly and quietly. If recovery takes place there is gradual improvement in the circulation and nutrition, and, later, a disappearance of the areas of induration.

The causes of sclerema are general, the most important factors being loss of fluids, great feebleness with lowering of the body temperature, and, in consequence, hardening of the subcutaneous fat. If it be true, as stated by Langer, that the fat of early infancy contains more palmitine and stearine than that of adults, it is easy to see how this may occur. There are no essential lesions in this disease. Atelectasis is often present, and may have something more than an accidental association, as incomplete aëration of the blood is no doubt a factor in the production of the symptoms. In Northrup's case, the skin after being injected was studied with great care microscopically, with absolutely negative results.

The prognosis is very bad, because of the grave conditions of which it is the expression, but it is not invariably fatal. In its milder forms, where treatment is begun early, recovery may take place. The diagnosis is to be made from œdema by the fact that there is no pitting upon pressure, by the rigidity of the body, and by the great reduction in the temperature. The most important thing in treatment is artificial heat; nothing but the incubator is efficient. In addition to this, care should be taken to promote the general nutrition by careful feeding and by all other means possible.

ŒDEMA.

Œdema has often been confounded with sclerema, but, although they may sometimes exist together, the conditions are quite distinct. Œdema occurs in delicate infants, and is associated with a feeble heart, especially of the right side, in consequence of which there are insufficient aëration of the blood, overfilling of the veins, and often a lowering of the body temperature. It also depends upon poor blood states, like severe anæmia, and I have seen it occur after hæmorrhages. The kidneys are unaffected.

The swelling is first noticed in the eyelids, the dorsum of the feet, the hands, or in dependent parts of the body. It may come on quite suddenly. In severe cases there may be general anasarca, but dropsy into the serous cavities is rare. Sometimes the first thing observed may be a sudden increase in weight before the œdema of any part is striking enough to be noticed. The general condition is feeble; the surface of the body cool; the temperature often subnormal; the cry weak; the urine often scanty, but rarely albuminous. The diagnosis of œdema is quite easy, the parts having the same appearance as in older patients. They are soft and waxy-looking, and pit upon pressure. While in most cases the prognosis is unfavourable, the disease is not necessarily fatal, since some even of the severe cases recover. The usual duration is five or six days; but there are frequently relapses.

The object of treatment is first to promote the general nutrition by all available means, and then to improve the circulation by the administration of heart stimulants, particularly digitalis and alcohol. In cases of extensive œdema, alkaline diuretics, like the citrate of potash, may be combined with digitalis. The body-temperature must be carefully maintained by artificial heat. The principal complications are diseases of the lungs and of the intestines.

INANITION FEVER.

The term *inanition fever* is not altogether a satisfactory one; but, until these cases are better understood, it is adopted because it emphasizes the very close connection which exists between the rise of temperature and the condition of inanition or starvation. Under this heading are included cases seen during the first five days of life—generally from the second to the fourth day—in which there is an elevation of temperature, apparently due to the fact that the infant gets very little, frequently nothing at all from the breast at which it is being suckled. It is further characteristic of these cases that the temperature falls when the child is put upon a full breast, or when artificial feeding is begun, or even when water is administered, if freely given. Some have ascribed the symptoms to uric-acid infarction of the kidneys.

So far as my knowledge goes, the first to call attention to this condition was McLane (New York), who in 1890 reported to one of the medical societies an extraordinary case of hyperpyrexia in a newly-born child. The infant was found on the sixth day with a temperature of 106° F., near which point it had remained for three days. The child was being suckled at a breast which was found to be absolutely dry. A wet-nurse was procured, the temperature fell to normal in a few hours, and the child, which when first seen was apparently in a hopeless condition, was soon perfectly well.

Since that time very extensive observations, extending to upward of three thousand cases, have been made at the Sloane Maternity and Nursery and Child's Hospitals, which have established the fact that a rise of temperature to 102° or even 104° F. is quite common in newly-born infants during the first few days. This fever is accompanied by no evidences of local disease, and ceases in nursing infants with the establishment of the free secretion of milk. The fall in temperature is often rapid, dropping to the normal in a few hours after having continued for three or four days, and in a large number of cases it does not rise again.

The following case is a fairly typical one of the more severe form: The patient was the second child, the first having died at the age of ten days, from no disease it was said, but simply from exhaustion. At birth the infant, a boy, weighed eight and a quarter pounds and was apparently vigorous. During the first forty-eight hours his loss in weight was five and a half ounces and his condition good. I saw him on the evening of the third day. In the preceding twenty-four hours he had lost eight ounces in weight, and the temperature had gradually risen, until at the time of my visit it was 102·8° F. The body was limp, the child making no resistance to examination. He cried with a feeble whine; the restlessness of the early part of the day having given place to complete apathy. The lips and skin were very dry, the fontanel sunken, the pulse weak. As the father, a physician, expressed it, "he had been wilting through the day like a flower in the sun." Although put to the breast regularly, the child had apparently got very little. It was, in fact, impossible to squeeze any milk from the mother's breasts. Water was freely given and a wet-nurse secured in a few hours. The first milk was taken from the wet-nurse at 11 P. M., and the temperature, which fell gradually during the night, was normal the next morning and did not rise again. (See chart, Fig. 21). During the succeeding four days the child gained eighteen ounces in weight, and at the end of a week was as well as an average infant of his age.

The symptoms are so uniform and so characteristic that they make for these cases of fever a class by themselves. The frequency with which this is seen is shown by the following statistics: Among 200 infants taken successively at the Nursery and Child's Hospital, 20 had fever during the first five days, reaching 101° F. or over, which was not explained by ordinary causes and followed the course above described. In 500 successive children born at the Sloane Maternity Hospital, there were 135 with a similar fever. It was seen in vigorous infants as well as in those who were delicate. The usual duration of the fever was three days, the temperature generally touching the highest point upon the third or fourth day of life. In about two thirds of the cases the temperature did not rise above 102° F.; in 9 it was 104° F. or over, the highest recorded being 106° F. The fall was generally quite abrupt, although not always so. Daily weighings, which were made in these cases, showed that the infants continued to lose weight while the fever continued, and that the loss almost invariably exceeded by several ounces that of the children who had no fever. The maximum loss noted was twenty-eight ounces. In quite a large number of cases it exceeded twenty ounces. As a rule the infants began to gain in weight when the temperature remained at the normal point, but not until then.

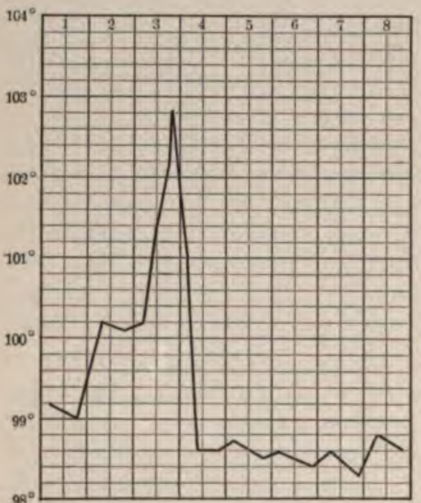

Fig. 24.—Temperature chart. Inanition fever.

The symptoms presented by these infants were a hot, dry skin, marked restlessness, dry lips, and a disposition to suck vigorously anything within reach. With very high temperature there were considerable prostration and weakened pulse. In the less severe cases there were only crying and restlessness. The rapidity with which the symptoms disappeared when the children were wet-nursed or properly fed, was very striking.

It is important that this fever should be recognised, because it gives at times the first warning of a condition which may prove fatal. The extra loss of ten or fifteen ounces in the first week, is a serious handicap to newly-born infants, the effect of which may last for several weeks. The temperature of every child should be taken during the first week. All the usual local causes of fever are first to be excluded by a physical examination. This fever can hardly be confounded with that due to pyogenic infection, which rarely begins before the fifth or sixth day.

The treatment is simple—viz., to give water regularly every two hours, in quantities up to an ounce at a time if required by the thirst of the child. This should be done in every case where the temperature reaches 101° F. When the temperature does not at once begin to fall, the infant should be put upon another breast or artificial feeding should be begun. Examination of the breasts from which the child has been nursing will usually reveal the fact that the secretion of milk is very scanty and often entirely absent.

Such a fever I have occasionally seen in older infants, usually in those who are nursing dry breasts or where fluid food and water have been withheld because of some gastric disturbance. It yields as promptly to treatment as does the same condition in the newly born.

SECTION II.

NUTRITION.

CHAPTER I.

INTRODUCTORY.

NUTRITION in its broadest sense is the most important branch of pædiatrics. Nowhere else and at no other time of life does prophylaxis give such results as in the conditions of nutrition in infancy. The largest part of the immense mortality of the first year is traceable directly to disorders of nutrition. The importance of correct ideas regarding this subject can hardly be overestimated. The problem is not simply to save life during the perilous first year, but to adopt those means which shall tend to healthy growth and normal development. The child must be fed so as to avoid not only the immediate dangers of acute indigestion, diarrhœa, and marasmus, but the more remote ones of chronic indigestion, rickets, scurvy, and general malnutrition, since these conditions are the most important predisposing causes of acute disease in early life.

One of the difficulties has always been that temporary success may mean ultimate failure. If the injurious effects of improper feeding were immediately manifest, there would be very much less of it than exists at the present time. Many things are valuable as temporary foods, which when used permanently are injurious. No better illustration of this is seen than in the too exclusive use of the carbohydrate foods. Infants fed upon many of the proprietary foods often grow very fat, and for the time appear to be properly nourished. The effect of the absence from the diet of some of those elements which are of vital importance may not be evident for months. The physiological laws regarding the requirements of the growing organism can not be ignored without serious consequences, which will sooner or later be evident. Correct ideas of infant feeding are based upon a knowledge of these laws. An accurate understanding of fundamental principles is essential to success and the vast majority of failures may be ascribed to ignorance or disregard of them.

THE FOOD CONSTITUENTS AND THE PURPOSES THEY SUBSERVE IN NUTRITION.

In infancy and childhood, as in adult life, the elements of the food are five in number: proteids, fats, carbohydrates, mineral salts, and water. The forms in which they must be furnished to the child, and the relative quantities in which they are demanded, are different from those required by the adult. One reason for this difference is the delicate structure of the organs of digestion in infancy, and their inability to assimilate certain forms of food. Again, provision must be made not only for the natural waste of the body, but for its rapid growth, nearly trebling in size, as it does, during the first twelve months.

Proteids.—The proteids are essential to life, since they are the only kind of food which is capable of replacing the continuous nitrogenous waste of the cells of the body, upon the healthy condition of which the digestion and assimilation of the other elements of the food depend. Without the aid either of the fats or the carbohydrates, the proteids may sustain life and may even prevent a loss of weight for a time; but in so doing a great excess of such food is required, as twenty-two parts of proteid can do the work of only ten parts of fat. Such a diet taxes severely the digestive organs and the kidneys. When, however, fats and carbohydrates are added to the food, only one-half or one-third as much proteid is required to replace the nitrogenous waste, as in the case of an exclusive proteid diet.

Of all the forms in which proteid food may be furnished to the body, in proportion to its nitrogen content, milk requires for its digestion the smallest amount of gastric and pancreatic juice. This fact is of the greatest importance and indicates the superiority of milk as a food, not only for the first year but throughout childhood. The most easily digested proteids are those of woman's milk. The greatest difficulty in artificial feeding is to supply their place for the nutrition of young infants when woman's milk is not available. The proteids of cow's milk present marked differences which are not yet fully understood.

Although the digestion of the proteids is begun in the stomach, it is principally carried on in the intestines. Disturbances of digestion due to the proteids are, therefore, attended by intestinal rather than gastric symptoms, an important point to be remembered both with nursing infants and with those who are taking cow's milk.

The proteid molecule is a very complex one when compared with that of the fats or carbohydrates. Growing out of this complexity of structure is the relative difficulty of digestion and the possibility of an immense number of side-products which may be formed by the splitting up of the proteid molecule by digestive ferments or by the numbers and varieties of bacteria found in the intestine. While the products of decomposition of the carbohydrates are often very irritating, those formed from the proteids may be very toxic and may be the cause of obscure and severe clinical conditions.

The prolonged use of a diet in which the proteids are insufficient in amount or are furnished in such form that they can not be digested or assimilated, produces a certain definite group of symptoms which are not always referred to their proper cause. In infants the most striking are anæmia, poor circulation, feeble muscular power, disinclination to exertion, and various functional nervous disturbances. Such children are often very fat.

The vegetable proteids can not permanently take the place of the animal proteids in the food of young infants.

Fats.—The uses of the fats in nutrition are many and varied.

They form the most important source of animal heat, their caloric value being a little more than twice as great as that of the carbohydrates or the proteids.

They save nitrogenous waste. The fats should be supplied in the food in such amount that the entire energy of the proteids may be utilized for the growth and nutrition of the cells of the body without being drawn upon to furnish animal heat. The rapid growth of the body in early life makes such demands upon the proteids that it is desirable that other elements of the food should do the work of the proteids whenever possible.

The fats increase the body weight. The large amount of fat stored up in the subcutaneous tissues in infancy is one of the best evidences of health.

The fats supply important elements needed for the normal development of the nervous system. This fact is probably connected with the large amount of fat of various forms which the nerve structures contain. It is a familiar clinical fact that functional nervous disorders are exceedingly common as a result of the long-continued use of foods low in fat. Many such disturbances commonly seen with rickets are regarded by some as a consequence of fat-starvation.

In the growth of bone the fats play an important rôle. The fatty acids formed in the intestine by the splitting up of the neutral fats of the food, combine with the insoluble salts of lime and magnesium and in this way, chiefly, these substances necessary for the growth of the skeleton are absorbed. Normal bony development, therefore, suffers if the food is low in fat.

The unabsorbed fats have a distinct value in preserving the proper consistency of the fæcal mass. While neither the proteids of milk nor the milk sugar appears as such in the stools of the nursing infant, fat is abundant. It forms normally from 10 to 15 per cent of the dry substance of the stool. The amount furnished to the infant is, therefore, considerably in excess of the needs of the body for nutrition. The use of this excess seems to be to increase the volume of the stool and to keep the mass so soft as to be easily expelled. This is readily appreciated by comparing the stool of a healthy nursing infant receiving a food containing 4 or 4·5 per cent fat with that of an infant fed upon diluted cow's milk containing 2 per cent fat. In a sense, therefore, fat may be regarded as a natural laxative.

The amount of fat required in infancy is relatively much greater than in adult life. A well-nourished nursing infant weighing 15 pounds actually receives about one-half as much fat as is allowed in a ration for an adult doing moderate work, who weighs ten times as much.

While it is evident from the foregoing that the fat requirements of the young child are great, it must also be remembered that in certain conditions even the normal amount of fat is badly borne and may do positive harm. Fats do not readily form products injurious to the economy as a consequence of imperfect digestion, but the amount given should be very greatly reduced in the following circumstances: (1) All wasting conditions depending upon disorders of digestion, whether due to functional derangement of the stomach, intestine, liver, or pancreas, or to chronic catarrhal inflammations of the stomach or intestine; (2) all acute disorders of digestion or acute inflammations of the stomach or intestines; (3) all febrile conditions, no matter from what cause. A failure to regard these contraindications is a constant source of trouble in practice.

In the conditions just enumerated the fats must largely be replaced by the carbohydrates, as these substances are capable for the time being of assuming the functions of the fats, and besides are easily digested and assimilated. Such substitution should not be continued too long, as serious results may follow. The importance of fats in nutrition does not end with the first year; they should be supplied liberally throughout childhood in the form of cream, eggs, butter, and cod-liver oil.

Carbohydrates.—Although these, like the fats, can not replace the nitrogenous waste of the body, they are important aids to the proteids, and in this respect they are even more valuable than the fats. The carbohydrates are partly converted into fats, and may thus increase the body-weight. They are capable of replacing the fat-waste of the body. They are one of the most important sources of animal heat.

Carbohydrates are the most abundant of the solid elements of the food, although they form a smaller percentage of the entire quantity of food in infancy than in adult life. The soluble carbohydrates which are used as foods for children are milk sugar, cane sugar, and maltose. Since all of these are converted by digestion into glucose they are to a certain degree interchangeable. In selecting milk sugar as the chief carbohydrate for the first year, we are following Nature, for this is what is furnished in the milk of all mammals. Milk sugar has a decided advantage in not fermenting with the common varieties of yeast present in the stomach, as do both maltose and cane sugar. Like the other sugars, however, milk sugar does readily undergo fermentation in the intestine by the action of bacteria.

The ability of the young infant to digest starches is relatively feeble, although this power does exist to some degree even from birth; but the greater part of the carbohydrates required should be furnished in the form of sugars. To infants of six months and over, starches may advantageously be added to the diet, and after the first year the quantity may be considerably increased. But in whatever form or quantity used thorough cooking is indispensable. Insufficient cooking is responsible for much of the starch indigestion seen in young children.

The advantages of the carbohydrates as foods depend upon their easy digestibility. The transformation of any of the sugars into glucose is a relatively slight chemical change, when compared with that which is necessary in the fats or proteids before they can be absorbed.

The carbohydrates are at a great disadvantage on account of the readiness with which they undergo fermentation in different parts of the alimentary tract. To such fermentations are due many of the symptoms seen in the common functional disorders of digestion.

A diet consisting too exclusively of carbohydrates leads often to a rapid increase in weight, but it is not accompanied by a proportionate increase in strength. Infants so fed have but little resistance, and many of them become rachitic. The easy digestion of a food consisting chiefly of soluble carbohydrates, and the rapidity with which children so fed gain in weight, lead to a great misapprehension in regard to their value as foods. The ultimate results of such one-sided feeding, if long continued, are almost invariably disastrous.

In building up the cells of the body the proteids are first in importance, the carbohydrates second, and the fats third. In the production of animal heat the fats come first, the carbohydrates second; practically the proteids should never be called upon for this purpose. In a proper diet, all of these elements are represented.

Mineral Salts.—These are relatively of greater importance in infancy than in later life, because of the rapid development of the skeleton during infancy and early childhood. The most important for this purpose are the phosphates of lime and magnesium. These are furnished in abundance both in woman's and cow's milk. These salts are also necessary for cell growth. Other inorganic salts furnish the elements from which the mineral constituents of the blood and digestive fluids are formed, and still others facilitate absorption, excretion, and secretion.

Water.—The food of all young mammals consists of from eighty to ninety per cent of water. This is needed for the solution of certain parts of the food, such as the sugar, the salts, and some of the proteids, and for the suspension of the other proteids and the emulsified fat. All the food is thus dissolved or very finely divided so as to be more readily acted upon by the feeble digestive organs of the infant. Water is needed also in large quantities for the rapid elimination of the waste of the body. In proportion to its weight, an average infant during the first year requires about five times as much water as an adult. During the time when the child is upon an entirely fluid diet, the addition of much water other than that supplied by the food is unnecessary; but when the number of feedings becomes less frequent, and solid food is given in larger quantities, water should be given freely between the feedings at all seasons, but especially in the summer.

Caloric Values.—The different foodstuffs have different caloric values:

One gram of fat	yields 9·3	calories.
" " " carbohydrates	" 4·1	"
" " " proteids	" 4·1	"

It is important that these caloric values should be considered in the dietary.

CHAPTER II.

THE INFANT'S DIETARY.

WOMAN'S MILK.

Woman's milk is the ideal infant-food. A thorough knowledge of its character, exact composition, and variations is indispensable, for upon this knowledge are based all our rules for the preparation of foods used as substitutes for woman's milk when this can not be obtained.

Woman's milk is a secretion of the mammary glands and not a mere transudation from the blood-vessels; although under abnormal conditions it may partake more of the character of a transudation than a secretion. A few drops may be squeezed from the breasts before parturition; generally speaking, however, it is only present after delivery. During the first two days the secretion is scanty. Usually upon the third or fourth day it becomes well established, although it may be delayed until the fifth or sixth day. During the period of lactation, milk is constantly formed in the mammary glands, but the process is more active while the child is at the breast.

Physical Characters.—Woman's milk is of a bluish-white colour and quite sweet to the taste. When freshly drawn its reaction is amphoteric to litmus, or slightly acid to phenolphthalein. The specific gravity varies between 1·026 and 1·036, the average being 1·031 at 60° F. On the addition of acetic acid only a slight coagulation is seen, this being in the form of small flocculi, and never in large masses as is the case in cow's milk. Microscopically, there are seen great numbers of fat-globules nearly uniform in size and some granular matter. Occasionally there are present epithelial cells from the milk-ducts or from the nipple.

Colostrum.—The secretion of the first three or four days differs quite markedly from the later milk. To this the name *colostrum* has been given. It is of a deep yellow colour, which is chiefly due to the colostrum-corpuscles. It is not so sweet as the later milk. It has a specific gravity of 1·030 to 1·040, a strongly alkaline reaction, and is coagulated into solid masses by heat, and sometimes coagulates spontaneously. It is very rich in proteids and in salts. Microscopically the fat-globules are of unequal size, and there are present large numbers of granular bodies known as colostrum-corpuscles (Fig. 25, A). These are four or five times the size of the milk-globules (Fig. 25, B), and they are probably epithelial cells which have undergone fatty degeneration.

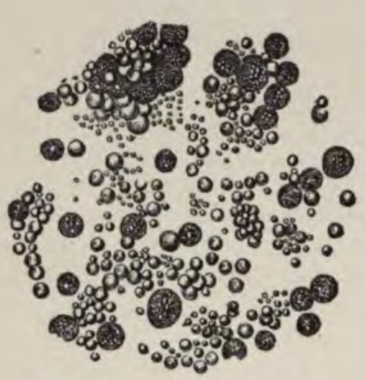

FIG. 25, A.—Colostrum. (Funke.)

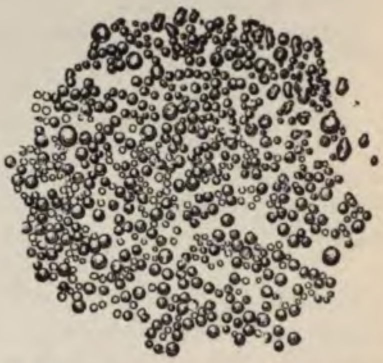

FIG. 25, B.—Woman's milk at a late period. (Funke.)

*Composition of Colostrum.**

Proteids	5·71
Fat	2·04
Sugar	3·74
Salts	0·28
Water	88·23
	100·00

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* From five analyses by Pfeiffer of milk obtained during the first three days.

The colostrum-corpuscles are very abundant during the first few days, but under normal conditions they are not found after the tenth or twelfth day.

Daily Quantity.—Exact information upon this point is difficult to obtain. There are recorded, however, extended observations made with great care upon eight cases,* from which some deductions may safely be drawn. All were healthy infants, nursing exclusively and gaining steadily in weight.

From these observations, and others less extended, the average daily quantity of milk secreted under normal conditions of health may be assumed to be pretty nearly as follows:

	Approximately.
At the end of the first week............	10 to 16 oz. (300 to 500 grm.).
During the second week	13 to 18 oz. (400 to 550 grm.).
During the third week......	14 to 24 oz. (430 to 720 grm.).
During the fourth week................	16 to 26 oz. (500 to 800 grm.).
From the fifth to the thirteenth week...	20 to 34 oz. (600 to 1,030 grm.).
From the fourth to the sixth month.....	24 to 38 oz. (720 to 1,150 grm.).
From the sixth to the ninth month	30 to 40 oz. (900 to 1,220 grm.).

It will be noted that the amount increases very rapidly up to about the eighth week, and after this much more slowly. The amount of milk

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* Haehner's cases (Jahrb. f. Kinderh., xv, 23; xxi, 314). Case I. Female; birth-weight 7 pounds 14 ounces (3,100 grammes). First week, lost 1½ ounce (45 grammes); after this gained steadily during the twenty-three weeks of observation; from second to ninth week, average weekly gain 8 ounces (241 grammes); from tenth to eighteenth week, average gain 4½ ounces (138 grammes); from nineteenth to twenty-third week, average gain 4 ounces (130 grammes); weight at the end of twenty-third week, 14¾ pounds (6,690 grammes).

Case II. Male; birth-weight 6½ pounds (2,950 grammes). Loss, first week, 3 ounces (90 grammes); after this gained steadily during the eleven weeks of observation; from second to eleventh week, average weekly gain 7½ ounces (214 grammes); weight at end of eleventh week, 11 pounds 2 ounces (5,045 grammes).

Case III. Female; birth-weight 3 pounds 9 ounces (1,620 grammes). Gain, first week, 1½ ounce (45 grammes); during the succeeding twenty-one weeks of observation, average weekly gain 5 ounces (141 grammes); weight at the end of twenty-second week, 10 pounds 3 ounces (4,620 grammes).

Laure's case (Thèse, Paris, 1889). Female; birth-weight 8 pounds 13 ounces (4,000 grammes); loss, first week, 8 ounces (225 grammes); after this gained steadily during the nine weeks of observation, on an average 9½ ounces (268 grammes) weekly; at the end of ninth week, weight 13 pounds 3½ ounces (6,000 grammes).

Ahlfeld's case (Deutsch. Ztschr. f. Prakt. Med., 1878). Birth-weight 7 pounds 14 ounces (3,100 grammes). Observations continued from fourth to thirtieth week. During first ten weeks, average weekly gain 5¾ ounces (161 grammes); from eleventh to twentieth week, 7½ ounces (214 grammes); from twenty-first to thirtieth week, 6 ounces (168 grammes); at the end of the thirtieth week, weight 18 pounds 9½ ounces (8,435 grammes).

Feer (Jahrb. f. Kinderh., xlii, 195). Three cases.

In all these cases the amount of milk was determined by weighing the infant both varies also with the demands of the child in a very striking way. The quantities mentioned can not be taken as an absolute guide as to the amount of food to be given to bottle-fed infants. Breast milk contains an average of twelve per cent solids; while the modification of cow's milk best suited to the early months contains only from nine to eleven per cent solids. For this period, therefore, somewhat larger quantities are needed than of breast milk.

A comparison of the daily amount of milk taken with the weight of the child at the different periods, showed that both during the early and the later periods the larger children took not only more milk, but considerably more in proportion to their body-weight than did the smaller ones. This harmonizes with the common observation that small children are much more likely to be overfed than large ones.

The average quantity taken at one nursing by five of the children previously mentioned was as follows:

		Approximately.
During the first week	⅝ to 1½ oz.	(18 to 45 grm.).
During the second week	1 to 3 oz.	(30 to 90 grm.).
During the third week	1½ to 4 oz.	(45 to 120 grm.).
During the fourth week	1½ to 4½ oz.	(45 to 140 grm.).
From the fifth to the seventh week	2 to 5 oz.	(64 to 150 grm.).
From the eighth to the eleventh week	2½ to 5½ oz.	(75 to 160 grm.).
During the fourth month	3 to 6 oz.	(90 to 180 grm.).
During the fifth month	3½ to 6½ oz.	(110 to 200 grm.).
During the sixth month	4 to 7 oz.	(120 to 220 grm.).

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before and after every nursing during the entire period of observation. The following table gives in a condensed form the daily quantity of milk in these cases:

TIME.	Haehner's 1st case.	Haehner's 2d case.	Haehner's 3d case.	Laure's case.	Ahlfeld's case.	Feer's 3 cases. Average.
	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.	Grammes.
1st day	20	75	20	...	...	...
2d day	176	135	45	...	...	...
3d day	265	325	70	125	...	...
4th day	420	295	99	222	...	...
5th day	360	290	124	400	...	256
6th day	374	340	136	475	...	(average
7th day	423	350	156	500	...	1st week)
Average 2d week	497	423	229	556	...	...
Average 3d week	550	468	314	730	...	...
Average 4th week	594	531	379	810	576	610
Average 5th week	663	561	447	944	655	667
Average 6th week	740	661	472	978	791	753
Average 7th week	880	681	525	1,038	811	802
Average 8th week	835	730	568	1,024	845	815
Average 9th week	766	665	584	1,085	810	820
Average 10th to 13th week.	796	...	600	....	869	795
Average 14th to 17th week.	807	...	673	....	983	845
Average 18th to 23d week	870	...	709	....	1,029	919
Average 24th to 30th week.	...	...	...	....	1,145	1,002

Between the limits mentioned the greater number of cases will undoubtedly fall. The amount taken at one time is, however, modified by the frequency of nursing, and is therefore not so good a guide to the amount of food required, as is the quantity taken in twenty-four hours.

Composition.—Many of the older analyses of milk gave erroneous results because of imperfect methods of examination. According to the most recent analyses of Pfeiffer, Koenig, Leeds, Harrington, Adriance, and others, the composition of human milk is as follows:

	Normal average.	Common healthy variations.
	Per cent.	Per cent.
Fat	4·00	3·00 to 5·00
Sugar	7·00	6·00 " 7·00
Proteids	1·50	1·00 " 2·25
Salts	0·20	0·18 " 0·25
Water	87·30	89·82 " 85·50
	100·00	100·00 100·00

In the older analyses, the percentage of proteids is almost invariably too high and the sugar too low.

The milk varies in composition somewhat with the period of lactation. That of the colostrum period is high in proteids and salts and low in sugar. By the end of the second week all these elements have usually reached their normal averages. After this time until near the end of lactation the regular variations are slight. However, there is seen, according to Adriance, a slow but steady fall in the proteids and salts and a very slight rise in the sugar, while the fat is scarcely affected at all.

Proteids.—The proteids are as yet imperfectly understood. The important ones are casein and lactalbumin; others, lactoglobulin and lactoprotein, are also described. The casein is in suspension by virtue of the presence of lime phosphate in the milk, with which it is probably in combination. It coagulates only slightly with rennet, while acetic acid produces a loose flocculent precipitate. The lactalbumin resembles the serum-albumin of the blood. Chemists are by no means agreed in regard to the proportion of the different proteids present in milk. Lactalbumin exists in woman's milk in much larger amount than in cow's milk, and it is more abundant than the casein, the proportion of the two being, according to Koenig, about as five to four.

The total proteids of normal milk are usually from one to two per cent. In abnormal specimens the variations are from 0·7 to 4·5 per cent. The proteids are highest in the milk of the first few days; after the first month they vary but little until toward the close of lactation, when the amount falls very markedly.

Fat.—This exists in the form of minute globules, which are held in a state of permanent emulsion by the albuminous solution in which they are suspended. The fat of woman's milk is chiefly made up of the neutral fats—palmitine, stearine, and oleine; there are also small quantities of the fatty acids, but these are much less than in cow's milk. Like the proteids, the proportion of fat is subject to wide variations, 4 per cent being taken as the normal average. In a series of thirty-four analyses made for me at the laboratory of the College of Physicians and Surgeons, the fat varied between 1·12 and 6·66 per cent. The highest percentage I have known was 10·91. In forty-three analyses by Leeds, the variations were between 2·11 and 6·89 per cent. The proportion is very little affected by the period of lactation.

Sugar.—The sugar is in complete solution. Its proportion is nearly constant, the average being seven per cent. The ordinary variations are usually within the limits of 6 and 7 per cent. The sugar being so important as a heat-producing element, Nature has wisely provided that this shall be the most constant ingredient of the milk. The amount of sugar is smallest in the milk of the first week; after the first month, however, the variations are slight.

Salts.—The average proportion of inorganic salts is 0·20 per cent, or a little more than one-fourth that of cow's milk.

With the exception of calcium phosphate nearly all the salts are in solution. The milk of the first few days is very rich in salts; after the first month the variations are slight but show a gradual fall in the quantity present.*

The Examination of Milk.—The exact composition of human milk is to be determined only by a complete chemical analysis. There are, however, many variations in composition which the physician may readily ascertain for himself by simple methods of examination.

The *quantity* of milk secreted by the breasts may be estimated by the quantity which may be drawn by a breast-pump, although this is not a very reliable test. If the child nurses habitually forty or fifty minutes, the probabilities are very strong that the quantity of milk is small. If the breasts at nursing time are full, hard, and tense, the supply is probably abundant. If the breasts are soft and flabby, and appear to fill only while the child is nursing, it is almost certain that the quantity is small. The most reliable of all tests is weighing the infant before and after nursing, upon an accurate pair of scales, sufficiently sensitive to indicate half-ounces. Two or three weighings will suffice to show conclusively whether an infant at three months, for instance, is getting habitually four or five, or only one or two ounces at a nursing.

The *reaction* of woman's milk even when freshly drawn is rarely alkaline, being amphoteric to litmus, or slightly acid to more delicate tests (phenolphthalein).

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* Bunge's analysis is given on page 150.

The *specific gravity* may be taken with any small hydrometer graduated from 1·010 to 1·040 (Fig. 26, A). The specific gravity is lowered by the fat, but increased by the other solids. An ordinary urinometer will answer every purpose, the only difficulty being the quantity which is required to float the instrument.

Microscopical examination.—The microscope reveals the presence of fat globules, colostrum-corpuscles, blood, pus, epithelium, and granular matter. Colostrum-corpuscles are abnormal after the twelfth day; pus and blood are always abnormal. The presence of any of these elements necessitates the suspension of nursing, at least temporarily. But little importance can be attached to the size and appearance of the fat globules as affecting the nutritive properties of the milk.

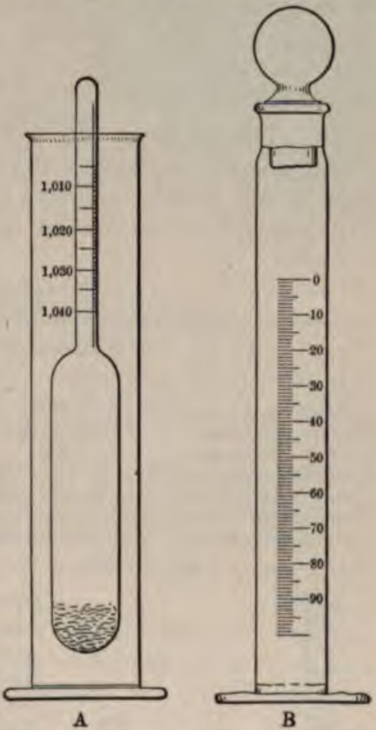

FIG. 26.—Apparatus for examination of woman's milk.
The author's lactometer and cream-gauge.

The determination of fat.—The simplest method is by the cream-gauge (Fig. 26, B). Its results are only approximate, but in most cases sufficiently accurate for clinical purposes. The tube is filled to the zero mark with fresh milk, which stands, corked, at a room temperature for twenty-four hours, when the percentage of cream is read off. The ratio of this to the fat is approximately five to three; thus 5 per cent cream indicates 3 per cent fat, etc.

For an accurate determination the best ready method is the modification by Lewi * of the Leffman and Beam test for cow's milk. This is a centrifugal test requiring special tubes.

Sugar.—The proportion of sugar is so nearly constant that it may be ignored in clinical examinations.

Proteids.—Clinical methods for the estimation of the proteids are not altogether satisfactory. The one giving the best results is that in which the proteids are precipitated by a solution of phosphotungstic and hydrochloric acids in the Esbach tube, the percentages being read off after standing twenty-four hours.* We may also form an approximate idea of the proteids from a knowledge of the specific gravity and the percentage of fat, if we regard the sugar and salts as constant, or so nearly so as not to affect the specific gravity. We may thus determine whether they are greatly in excess or very low, which, after all, is the important thing. The specific gravity will then vary directly with the proportion of proteids, and inversely with the proportion of fat—i. e., high proteids, high specific gravity; high fat, low specific gravity. The application of this principle will be seen by reference to the accompanying table.†

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* Lewi's method is as follows:

(1) Place in the milk flask 2·92 c.c. of woman's milk measured in a special graduated

Woman's Milk.

	Specific gravity 70° F.	Cream—24 hours.	Proteids (calculated).
Average............	1·031	7%	1·5%
Normal variations...	1·028–1·029	8%–12%	Normal (rich milk).
Normal variations...	1·032	5%–6%	Normal (fair milk).
Abnormal variations.	Low (below 1·028).	High (above 10%).	Normal or slightly below.
Abnormal variations.	Low (below 1·028).	Low (below 5%).	Very low (very poor milk).
Abnormal variations.	High (above 1·032).	High.	Very high (very rich milk).
Abnormal variations.	High (above 1·032).	Low.	Normal (or nearly so).

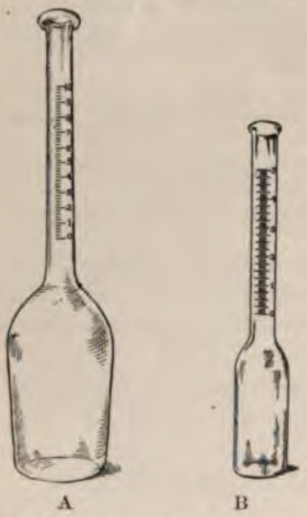

FIG. 27.—Tubes for determining the fat in milk. A, Babcock's tube for cow's milk; B, Lewi's modification for woman's milk. (See also page 147).

pipette; (2) carefully rinse the pipette and add the same quantity of sulphuric acid C. P. of specific gravity 1·830. The acid should be added slowly, and mixed with the milk by gently rotating the flask. The colour turns to a very dark brown from the oxidation of the sugar and proteids; (3) now add 0·6 c.c. of a mixture of equal parts of fusel oil and strong hydrochloric acid; (4) add sufficient of a mixture of the same sulphuric acid and water, equal parts, to bring the level of the fluid well up into the neck of the flask; (5) centrifuge for three or four minutes. The percentage of fat is now read off, each one-tenth gradation in the neck of the flask representing 0·3 per cent of fat in the specimen of milk.

This test has been modified by omitting the addition of strong sulphuric acid—the second step in the test—and in the third step, amyl alcohol is substituted for fusel oil. These reagents are much safer of manipulation and meet all the indications.

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* For description see Boggs, Johns Hopkins Hospital Bulletin, No. 187, October, 1906.

† The author's apparatus may be obtained from Eimer & Amend, Eighteenth Street and Third Avenue, New York. For a fuller discussion of the subject, see Archives of Pædiatrics, March, 1893.

Any specimen taken for examination should be either the middle portion of the milk—i. e., after nursing two-or three minutes—or, better, the entire quantity from one breast, since the composition of the milk will differ very much according to the time when it is drawn. The first milk is slightly richer in proteids and much poorer in fat. The last drawn from the breasts is low in proteids and high in fat. The following analyses from Forster illustrate these differences:

	First portion.	Second portion.	Third portion.
	Per cent.	Per cent.	Per cent.
Fat	1·71	2·77	5·51
Proteids	1·13	0·94	0·71

Conditions Affecting the Composition of Woman's Milk.—*The age of the nurse.*—This has no constant influence. Other things being equal, the milk of very young women, and also of those over thirty-five years of age, is likely to be lower in fat than that of women between twenty and thirty-five years.

Number of pregnancies.—Adriance found that the average milk of 23 primiparæ and 23 multiparæ, both taken at the third month, showed the following differences: The milk of the primiparæ was higher in fat (4·06 against 3·67) and in proteids (1·61 against 1·35), but a little lower in sugar (6·52 against 6·85).

Acute illness.—In the majority of cases of acute illness of a minor character and of short duration there is no perceptible effect upon the milk. In the acute febrile diseases of a severe type the quantity of milk is reduced, the fat is low, and the proteids are apt to be high. In septic conditions bacteria may appear in the milk.

Menstruation.—The effect of this is exceedingly variable, depending much upon the individual and the ease of menstruation.

The nature of the changes in milk sometimes produced by menstruation is illustrated by the following case taken from Rotch:

	Second day of menstruation. Bowels of child loose.	Seven days after menstruation. Bowels regular.	Forty days after menstruation. Child gaining rapidly.
	Per cent.	Per cent.	Per cent.
Fat	1·37	2·02	2·74
Sugar	6·10	6·55	6·35
Proteids	2·78	2·12	0·98
Salts	0·15	0·15	0·14
Water	89·60	89·16	89·79

From observations upon 685 cases, Meyer noted disturbances in the child in over one-half the number. My own experience accords rather with that of Pfeiffer and Schlichter, who consider it quite exceptional for the child to be visibly affected. Schlichter made observations upon infants during 233 menstrual days, noting the condition of the stools and digestion both before and after menstruation. In ninety per cent of the cases there was no perceptible influence. In only eight per cent were the stools bad, and in only three per cent was there disturbance of the stomach with vomiting.

At the present time sufficient observations have not been made to show whether the differences noted in the case cited above—low fat and high proteids—are the rule where disturbances are produced during menstruation. Monti's examinations lead him to the conclusion that the fat is not constantly affected. It is safe to say that the changes are not uniform, and that in very many cases none of importance are produced by menstruation.

Diet.—The fat and the proteids of the milk are much influenced by diet, the sugar but very little. The fat is increased by a diet made up largely of nitrogenous food, meat, eggs, animal broths, etc.; it is reduced by stopping these articles and substituting vegetables and farinaceous food. The proteids are increased by overfeeding and also by too little exercise. Starvation lowers the fat and sometimes also the proteids; the latter may, however, be increased but altered in character. All fluids tend to increase the quantity of milk. Alcohol in the form of malted drinks, and malt extracts increase the quantity of milk and the amount of fat. The effect of alcohol upon the proteids is not constant, but they are usually increased. The following table gives the result of analyses of the milk of two women observed in the New York Infant Asylum before, while taking, and after taking an alcoholic extract of malt:

	I. Without malt.	II. After taking 8 oz. malt daily for 10 days.	III. No malt for 7 days.
Case I:	Per cent.	Per cent.	Per cent.
Fat	1·74	3·83	2·41
Proteids	1·93	1·58	2·95
Sugar	7·02	7·43	6·59
Salts	0·20	0·17	0·19
Case II:			
Fat	1·12	2·75	1·70
Proteids	1·57	2·34	1·26
Sugar	7·11	6·77	6·04
Salts	0·19	0·17	0·18

The child of Case I gained one ounce and a half during the four days preceding the first analysis; that of Case II did not gain at all. During the ten days while taking the malt, the first child gained twelve ounces, the second child eight ounces. During the seven days after the malt was discontinued, the first child gained eight ounces, the second child one ounce. There was a notable increase in the quantity of milk in both cases while taking the malt.

The nursing woman should have a generous diet of simple food, and should drink largely of milk or gruels made with milk. The diet should be a varied one, not excessive in nitrogenous food nor in vegetables. All salads and highly seasoned dishes should be avoided, not so much because they upset the child, although this may happen, as because they are likely to disturb the digestion of the nurse. Nearly all the common vegetables and fruits in season may be allowed in moderation. Strong tea and coffee should be prohibited, although weak tea or coffee may be allowed, each but once a day. Cocoa is less objectionable than either tea or coffee. In addition to her regular meals the nurse should have milk or gruel at bedtime. The diet should in all cases be adapted to her digestion. The bowels should move daily, by the use of laxatives if necessary. Great harm often results from overfeeding with its consequent indigestion. The regular use of alcoholic beverages should be forbidden.

Drugs.—The elimination of drugs through the milk is somewhat uncertain and variable; few of those popularly supposed to affect the child through the milk really do so. Given in full doses, belladonna regularly appears in the milk. Opium does not do so constantly; but when the milk is poor, enough may be excreted to produce serious symptoms, and, in infants a few days old, even to cause death. The iodides and bromides when long administered may be eliminated in sufficient quantity to produce their constitutional effects in the child. Mercury does not appear regularly, but only after prolonged use, and then in variable quantity. Most of the saline cathartics, arsenic, and the salicylates are occasionally found in the milk. Alcohol may seriously disturb the child if taken in considerable quantities by a nurse, although its elimination through the milk is doubtful.

Pregnancy.—The milk of pregnant women is generally small in quantity and poor in quality, especially in fat. (See Weaning.)

Bacteria.—Under normal conditions woman's milk may contain a few bacteria. They are chiefly cocci derived from the external milk ducts and are of no importance. In suppurative inflammation of the mammary gland, numerous bacteria may be found in the milk; also in some cases of puerperal sepsis. Tubercle bacilli have been demonstrated by Roger and Garnier in the milk of a woman with advanced tuberculosis, but ordinarily they are not present unless the gland is the seat of the disease.

The elimination of antitoxin and other protective substances by the milk.—The immunity of nursing infants to most of the contagious diseases has long been noted, but until recently little understood. Roger has published (Révue de Méd., May, 1900) a striking instance in point. In a single year there were admitted to a hospital 36 nursing mothers suffering from contagious diseases: 15 had measles; 19 scarlet fever; 1 diphtheria; 1 mumps. In no case did an infant contract the disease of its mother, although nursing was continued. Animal experiments have demonstrated the constant presence of diphtheria antitoxin in the milk of immunized animals. The Widal reaction has been obtained with the milk of mothers suffering from typhoid and with the blood of their healthy nursing infants. Clinical observations like that of Roger would seem to admit of no other explanation than that these infants did not take the disease of the mothers because something was conveyed to them through the milk, which rendered them immune.

Nervous impressions.—The effect of the nervous condition of a woman upon her milk secretion is very striking. Both the quantity and the composition of the milk are markedly changed by many different nervous impressions. Fright, grief, passion or any great excitement may entirely arrest the secretion, or if not arrested the milk may be so altered in composition as to make the child acutely ill. Worry, anxiety, fatigue, or prolonged nervous strain may so alter the milk as to cause it to disagree with a child who had previously thrived well upon it. It is the nervous condition of the mother more than anything else which determines her success or failure as a nurse. The nervous factor is of far greater importance than the diet. If a mother would nurse successfully, she must have plenty of rest and sleep, keep her mind free from unnecessary worries, avoid social engagements, and lead a simple, regular, natural life. Unless she can and will do this successful nursing can hardly be expected.

The nature of the changes produced in milk by nervous disturbances in the mother are as yet little understood. Some infants are so profoundly affected as to suggest the development of toxic substances in the milk. The milk of the tired and worried mother is nearly always low in fat while the proteids are usually high, possibly at the same time altered in their composition.

COW'S MILK.

The only one of the lower animals whose milk is practically available for infant feeding is the cow. Cow's milk being our main reliance in the artificial feeding of infants and the staple food of nearly all young children, it follows that everything relating to its production and handling is important. The practising physician should therefore familiarize himself with the main facts regarding the production and handling of milk according to modern methods, since no one can do more than he to educate public opinion in these matters, and so to improve the milk supply of the community. Only an outline of the subject can be presented here. For more minute knowledge the reader is referred to special works upon the subject.*

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* Convenient works for a physician's use are Richmond's Dairy Chemistry; Conn's Bacteria in Milk and its Products; Aikman's Milk, Its Nature and Composition, Block, London, 1899; Russell's Outlines of Dairy Bacteriology, 1899; and Belcher's Clean Milk, Hardy Publishing Co., New York.

The essential conditions to be fulfilled in cow's milk which is to be used as a food for infants and young children are: (1) Freshness; (2) it should contain no preservatives; (3) it should be from healthy animals, free from tuberculosis or other taint; (4) it should be clean; (5) it should not be skimmed or otherwise falsified; (6) it should contain no pathogenic organisms; (7) the number of other organisms should not be excessive. It is also desirable for purposes of infant feeding that the composition of the milk, particularly the percentage of fat, should be known, and that the milk should be as nearly uniform as possible from day to day and at different seasons of the year. Mixed or herd milk is therefore to be preferred to that from a single animal, since it is subject to fewer variations. The common varieties or "grade cows" should be chosen rather than highly bred animals, if for no other reason, because they are more hardy, less subject to disease, and less susceptible to other influences which might affect the milk.

As ordinarily handled, milk should be used before it is twenty-four hours old; after this time fermentative changes occur very rapidly, and such milk can not in summer be used with safety for young children. Milk may be safe when more than twenty-four hours old provided special precautions are taken regarding cleanliness in producing and handling it, and special care in keeping it constantly at a temperature below 50° F.

Preservatives are very often added, particularly in hot weather, by unscrupulous dealers to retard the souring of milk, in order thereby to avoid the necessity and expense of proper icing. Formerly boric or salicylic acid were, and recently formaldehyd has been largely employed for this purpose.

Micro-organisms in Milk.—Most of the common bacteria grow readily in milk, and the conditions under which it is produced and handled render it liable to contamination in many ways.

1. *Disease in the cow.*—From disease of the udder streptococci or other pyogenic germs may enter the milk in such numbers as to excite acute gastro-enteritis in a child. Other diseases which may possibly be communicated from the cow are anthrax, the "foot-and-mouth" disease, and tuberculosis. In the State of New York it is estimated that 7 per cent of the cows are tuberculous. Pearson and Ravenel estimate the proportion in Pennsylvania at 2 or 3 per cent, while Marshall states that from 10 to 25 per cent of the Eastern dairy cattle are tuberculous. The best veterinarians regard tuberculosis as steadily increasing among cattle in the United States, particularly in the Eastern States. Of the cattle slaughtered in London, 25 per cent are stated to be tuberculous. Unless the process is advanced or the udder is the seat of disease, very many tuberculous cows do not have tubercle bacilli in their milk. One English writer (Eastes) found tubercle bacilli in 11 of 186 miscella-

neous specimens of milk examined. For reasons given elsewhere (*vide* Tuberculosis), I can not believe the danger of acquiring tuberculosis through milk as great as some have represented. We need further data before we can say positively how often human tuberculosis is acquired from cows; absolute proof being almost impossible and the reported cases in which such transmission seemed highly probable being still few. For the present milk must be regarded as one of the possible sources of tuberculous infection. The sale of milk from cows showing evidence of tuberculosis upon physical examination, and from those having tuberculosis of the udder should not be permitted. Whether we should go further and exclude also the milk of every cow which reacts to the tuberculin test is still an open question.

2. *Specific pathogenic organisms accidentally gaining access to milk.*—The rôle of milk in the spread of infectious disease may be appreciated by the fact that in 1900 Kober collected records of 330 outbreaks which were traced to it. The most important disease communicated in this way is typhoid fever. In the reports of 195 epidemics collected, typhoid existed at the dairy in 148 instances; in 67 the milk was diluted with infected well-water; in 7 the cows probably waded in polluted water; in 24 cases the employees acted as nurses, and in 10 they continued at work, although themselves suffering from the disease; in one case it was found that the milk-pans were washed with cloths used about patients.

Next to typhoid the disease most often spread through milk is scarlet fever. A very small percentage of the cases of scarlet fever, however, can be traced to contaminated milk; but the sudden and simultaneous development of a considerable number of cases of this disease in a community, should lead one to consider the milk supply as a possible cause. Of 99 epidemics of scarlet fever, there was disease at the farm or dairy in 68; in 17, employees were themselves affected, and in 10 they acted as nurses; in 6, persons connected with the dairy either lodged in or had visited infected houses; in 2 infection was brought by cans or bottles from the houses of patients; in 3 the milk was stored near or in the sick-room; in one case milk-utensils were wiped with an infected cloth.

Very infrequently diphtheria has been spread through milk. Of 36 outbreaks of diphtheria collected, there was disease at the farm or dairy in 13; in 3, employees themselves were ill. Twelve of the outbreaks included in this series, however, were of very doubtful character. Besides these diseases mentioned, cholera, dysentery, and certain forms of diarrhœal diseases may probably be spread by milk.

3. *Other bacteria found in milk.*—These are chiefly derived from the air of the stable, the hands and clothing of the milker, and from the dirt which falls from the udder, belly, and tail of the cow into the pail during milking; very many come from the cow's excreta. Freeman exposed a Petri gelatin-plate beneath a cow's udder for one minute dur-

ing milking and obtained 4,450 colonies. The varieties of bacteria found in fresh milk are many and vary with locality. Toward the souring point the great majority are of two or three varieties only; fully 95 per cent at that time belong to the lactic-acid-producing group. They cause the ordinary souring of milk by acting upon the milk sugar. Colon bacilli are very common. Other bacteria act upon the milk proteids, inducing various fermentative or putrefactive changes; and still others have a peptonizing power. Of 15 varieties frequently present which were studied by Russell, 3 belonged to the lactic-acid group, 5 were peptonizing bacteria, while 7 had no recognizable effect upon milk.

Many of the bacteria are no doubt harmless. None have been shown to be beneficial. Others, while not strictly speaking pathogenic, when present in large numbers induce changes in milk that so impair its nutritive properties as to render it unfit for food, and in susceptible infants may cause serious illness. The effects of bacterial contamination of milk are considered in the introductory chapter upon Diarrhœal Diseases.

The number of bacteria in milk.—This depends upon three conditions: (1) Cleanliness in handling; (2) temperature; (3) age of the milk. Hence the bacterial count becomes of the greatest value in furnishing information as to these matters, although of less importance in regard to the production of disease than the nature of the organisms present. The influence of the different factors may be illustrated by the following experiments made at the laboratory of the New York Health Department: A sample of milk taken under good conditions contained immediately after milking 300 bacteria in each drop. It was cooled to 45° F., and kept at this temperature. After twenty-four hours it contained in each drop only 200 bacteria; after forty-eight hours, 900; and after seventy-two hours, 150,000. The milk curdled on the sixth day. Another sample, taken in a dirty barn, cooled and kept at 52° F., contained at first 2,000 bacteria in each drop; in twenty-four hours, 6,000; in forty-eight hours, 245,000; in seventy-two hours, 16,500,000. The milk curdled on the fourth day. The influence of temperature alone upon the multiplication of bacteria in milk is well shown by the following experiment: Four samples of the same milk were kept at different temperatures for twenty-four hours and equal quantities were then plated; No. I was kept at 60° F. and showed 134,340 colonies; No. II was kept at 55° F. and showed 67,170; No. III was kept at 50° F. and showed 1,362; No. IV was kept at 45° F. and showed 448.

The ability of milk to resist the growth of bacteria for a certain time is indicated by these and many other experiments. Exactly to what this is due is not quite clear. There seems, however, to be little doubt that milk, in common with other animal fluids, possesses certain bactericidal properties which render it stable for a limited time, which are soon exhausted if the temperature is allowed to rise, but which assist materially in its preservation during the first twenty-four hours.

The number of bacteria in cream is nearly always far greater than in milk. Cream is usually much older than milk at the time of delivery. Huddleston's investigations of the cream supplied to New York City led him to the conclusion that most of the cream was seventy-two hours old when it reached the consumer. The consistency of much of the heavy cream so popular with many is obtained with age and is largely the result of bacterial growth. Freeman's experiments with gravity cream showed that the bacteria were 300 times as numerous in the cream as in the milk left behind, the bacteria being apparently carried up with the fat globules. Both these facts emphasize the necessity of the greatest care with reference to cream and indicate one great advantage of centrifugal cream, that it can be marketed at least twenty-four hours earlier than gravity cream.

A bacteriological standard for pure milk.—Much discussion has arisen of late, especially among different milk commissions of physicians, regarding the possibility of establishing some such standard. One commission requires that the milk shall not have more than 10,000 bacteria in each cubic centimetre; another fixes the limit at 30,000. Methods of cultivating and counting the bacteria of milk are by no means uniform, and it is often quite impossible to compare the figures of different observers, because not all the conditions were the same. We are not yet ready to fix a standard. For milk sold in cans 100,000 to the cubic centimetre may be considered good; for bottled milk anything under 30,000 is good, and an average under 10,000 is exceedingly good; the count in all cases being made at the time the milk is offered for sale.

The reports made by the bacteriologist of one of the New York milk commissions show that by the most careful handling the number of bacteria * may be kept at an average of a little more than 5,000 bacteria in each cubic centimetre at the time when it is delivered to customers. The bottled milk from single high-class dairies usually ranges from 10,000 to 100,000 under the same conditions. Milk from mixed dairies delivered in cans ranges from 100,000 to 40,000,000, the latter being often reached in very hot summer weather.

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* To accomplish such a result certain special precautions were observed; the most important were the following: The stables had cement floors to admit of ready flushing with a hose; no hay, straw, or fodder were kept in the stables; shavings were used for bedding; the cows were carefully groomed every day and not fed until after they were milked; a few minutes before milking the loose dirt was removed from the udders with a damp cloth. The milkers wore sterilized coats and caps, and washed their hands before milking each cow: all bottles, pails, etc., were sterilized with live steam, the pails just before using. The milk was immediately removed to the milk-house, where it was strained, mixed, cooled to 38° F., bottled and sealed—all within twenty minutes from the time it left the cows.

The means of excluding pathogenic bacteria, and of checking the spread of contagious diseases through milk.—Rules are readily deducible from a study of the records of how milk has usually been infected.

1. No person suffering from, or in contact with a person suffering from, a contagious disease should enter a dairy building or in any way come in contact with the milk or milk-utensils; especially should this rule be enforced in the case of diphtheria, scarlet and typhoid fevers.

2. Milk should not be handled in or near dwellings, privies, or stables; cans and pails should be washed only at the dairy, and after ordinary cleansing they should be washed in boiling water or sterilized with live steam. Especial attention should be given to milk-bottles which have been in infected rooms. The hands of the milker should invariably be carefully washed just before milking.

3. Dairies should be subject to regular city or state inspection. Milk from cows showing physical evidence of tuberculosis should be excluded; also that from animals which are in any way sick or are suffering from disease of the udder should not be used.

4. In all epidemics of contagious disease, both large and small, the milk supply should be carefully investigated; and all cases of such diseases in the families of those who produce or handle the milk should be immediately reported and closely followed up by the authorities.

Means of reducing the number and lessening the growth of bacteria in milk.—A marked diminution in the number of germs present in milk, as it is now handled, may be brought about by attention to two conditions—cleanliness and temperature—and the results will be directly in proportion to the care bestowed upon them.

Cleanliness must have reference, in the first place, to the cows themselves. Since most of the germs in milk come from the cows, it is important that the belly, udder, and tail should be cleansed before milking, to prevent droppings into the pail. The parts should be wiped with a dry or damp cloth. Milking should be done out of doors or in a clean, special shed; if in the stable, this should be clean. No dry fodder should be fed and no sweeping done, nor anything else to raise a dust, just before milking. The milker's hands should be carefully washed and dry, not moistened with milk, as is sometimes done. Milk pails and cans should be washed, as stated above, and always dried upside down, remaining in this position until used. Pails with a small opening partially protected by a hood should be used to lessen the contamination with dirt from the cows during milking. All sieves and straining cloths should be sterilized before each using. Milk should be bottled at the dairy, and so transported. When this is not done the milk, after cooling, should be put into the vessel from which it is delivered; every time the milk is handled, poured from one vessel into another, or in any way manipulated, the danger of contamination is increased.

As to temperature, no point in the care of milk is more important than the rapid first cooling; as soon as possible after being drawn it should be cooled to at least 50° F. Unless the milk is taken at once to a milk-house and some of the special forms of cooling apparatus employed, the cans should be immersed in spring water having a temperature below 50° F., or in ice-water, and remain at least one hour. If a temperature of 50° F. is maintained during transportation, which is quite possible if cans and bottles are properly iced, and during subsequent storage, the growth of bacteria may be so retarded that milk may be a safe food even when forty-eight hours old. If the temperature is not kept as low as 50° F. this result can not be depended upon, and with every degree above that point the increase in bacterial growth is very marked. Since the number of bacteria increases so rapidly with the age of the milk after the first twenty-four hours, it is of the utmost importance that milk be shipped as quickly as possible after it is collected.

A provision of the Sanitary Code of New York city requires that no milk shall be sold having a temperature above 50° F. This ordinance has done more than anything else to improve the milk supply of the city, especially to insure proper icing during transportation.

The desirable results indicated above are to be secured, in the first place, by educating the public to appreciate, and dealers to produce, a better and cleaner milk; secondly, by giving to the health authorities of city and state greater power than heretofore in the matter of milk inspection; thirdly, by the formation of milk commissions,* through which the physicians of a town or city may co-operate to secure adequate supervision of at least a portion of the milk supply.

Composition of Cow's Milk.—Except in the percentage of fat, the composition of mixed or herd milk varies but little, whatever the breed. The fat is lowest in the Holsteins, and highest in the Jerseys.

Composition of Cow's Milk.

	Jerseys.	Holsteins.	Average good herd milk.
Fat	5·61	3·46	4·00
Sugar	5·15	4·84	4·50
Proteids	3·91	3·39	3·50
Salts	0·74	0·74	0·75
Water	84·59	87·57	87·25
Total	100·00	100·00	100·00

In the table the figures for Jersey and Holstein herds are the averages given by the New York State Experiment Station. The legal requirements in New York and most of the States are, fat, 3 per cent; solids not fat, 9 per cent.

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* The first such commission in the United States was organized in Newark, N. J., in 1893, largely through the efforts of Dr. H. L. Coit. It entered into a contract with a

The figures given for herd milk are a little lower for the proteids and a little higher for the sugar than the older analyses. It is with milk of such proportions that the average physician has to do in infant feeding. In a poor milk the only important difference to be considered is that the fat is from 0·5 to 1 per cent lower than the averages given. In a rich Jersey milk the chief difference is that the fat is 1 to 1·5 per cent higher than the averages; there is also an increase in the proteids and sugar which is less important, but should not be ignored. The variations in the fat content of milk are those which are of most practical importance to the physician. As to the relative advantages of the different breeds for this purpose, the difference does not seem great, provided all are equally healthy. Jerseys and all highly bred animals are more prone to serious disease and minor disturbances than the hardier common breeds.

The Examination of Cow's Milk.—The application of heat often causes coagulation in milk which is near the souring point, and also in colostrum milk. Both are unfit for use. The normal *reaction* of cow's milk is amphoteric or slightly acid. If strongly acid it should not be used; if alkaline, it is pretty certain that something has been added to it.

The *specific gravity* is from 1·028 to 1·033. If the milk has been falsified by the removal of cream, the specific gravity is raised; if adulterated by the addition of water, the specific gravity is lowered.

The best of all ready methods of determining fat are by the Leffman and Beam and the Babcock tests.* By both the fat is brought to the surface by the centrifuge after the addition of sulphuric acid and other reagents. These tests are similar, but differ in the reagents used. When carefully made they are very accurate. For institutions such an apparatus is indispensable; and the composition of the milk and the cream used can be determined each day. The optical test by means of Feser's

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dairyman, the terms of which were that the selection of the cows, the details regarding their food and care, and the handling of the milk, should be under the supervision of the Medical Commission. All these matters were to be carried out according to the most improved methods. The animals were to be subjected to a regular inspection by a competent veterinary surgeon; a chemist and bacteriologist to be employed to see that the milk was kept up to the standard both as regards composition and purity. In return, the milk, which was to be delivered only in bottles, was stamped with the approval of the commission as "certified milk," and sold at a slightly higher price than ordinary milk. This plan has proved eminently successful both from a medical and commercial standpoint, and has, with some minor modifications, been imitated in several other cities with equally satisfactory results. (See Archives of Pædiatrics, 1897, p. 824; also Philadelphia Medical Journal, October 20, 1900.)

* The apparatus can be obtained of D. H. Burrell & Co., Little Falls, N. Y. The one sold as the "Facile Junior" may be used for woman's milk, urine, and other fluids as well as for cow's milk, and is very convenient for physicians' use. Price, $10.

lactoscope (Fig. 28) is a good one, and with a little experience in the use of the instrument is quite accurate.*

The cream-gauge may be used as for woman's milk, but it is not to be relied upon unless the milk is put into the cylinder soon after it is drawn and cooled rapidly by being placed in ice-water. Under these conditions, if the reading is made at the end of eight or ten hours the percentage of cream to that of fat is about three to one. If the milk has been first cooled and afterward handled two or three times before the test is made, the cream does not rise regularly, and the above ratio is not maintained.

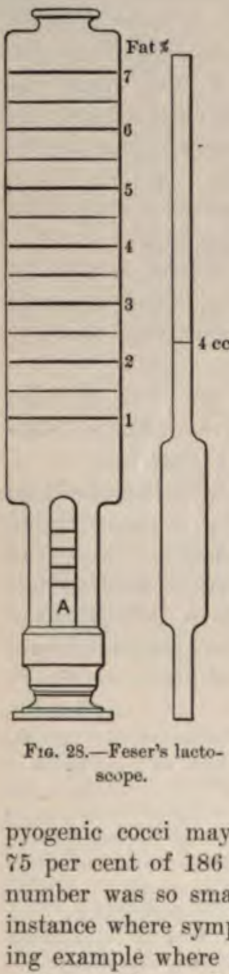

Fig. 28.—Feser's lactoscope.

A microscopical examination of milk is of considerable importance, and in cases where the character of the supply is questionable it may give valuable information. Both the cream and the sediment should be examined. Not much can be learned from a study of the fat globules, but among them may be found colostrum corpuscles, which are usually present for nearly a week after calving. The sediment is best studied after centrifuging. It should be examined for pus cells and blood, and stained for bacteria. A few leucocytes are almost invariably found in normal milk. Stokes and Wegefarth consider that an average of more than five in each field examined with an oil-immersion lens should be regarded as abnormal, and such milk excluded. The most frequent source of pus cells in numbers is inflammation of the udder. Pus cells may be associated with a stringy mucus as muco-pus. Blood may also result from inflammation of the udder, sometimes from traumatism.

Where pus cells are present the specimen should be examined for bacteria. Any of the ordinary pyogenic cocci may be found. Streptococci were found by Eastes in 75 per cent of 186 specimens examined, although in most of these the number was so small that no symptoms were produced. He cites one instance where symptoms were caused. Woodward has reported a striking example where a family of five children were all made seriously ill with vomiting and collapse after taking milk which was found by him to contain large numbers of streptococci. These cases are probably not very rare. In staining milk for tubercle bacilli it should be remembered that the bacilli found are, as a rule, shorter than those found in human sputum.

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* Obtained of Eimer & Amend, Eighteenth Street and Third Avenue, New York.

At the present time it is impossible to lay down definite rules as to what microscopical findings justify one in condemning a sample of milk; but whenever pus cells, muco-pus, blood, or streptococci are at all numerous, the milk should be regarded as unfit for food and a thorough inspection of the herd should be made.

The Differences between Cow's Milk and Woman's Milk.—Cow's milk is more opaque than woman's milk, although the latter may contain the larger proportion of fat. This opacity is due to the large proportion of calcium phosphate with which the casein is combined.

The *reaction* of cow's milk soon after it is drawn becomes acid. It is almost invariably found so unless some alkali has been added. Woman's milk is distinctly less acid.

The *specific gravity* and total solids in the two milks are about the same.

The *sugar* of both cow's and woman's milk is identical in composition; it is lactose in solution. The difference in amount is considerable. Cow's milk usually has 4·5 per cent, while woman's milk usually has from 6 to 7 per cent.

The greater part of the *fat* of cow's milk is neutral fat, as in woman's milk; cow's milk, however, contains in addition much larger quantities of the volatile fatty acids than does woman's milk.

The *proteids* of cow's milk are not only two and a half times as abundant as those of woman's milk, but they show marked differences in character.

Our knowledge of the proteids both of cow's milk and woman's milk is still very imperfect. The separation of the different proteids is difficult, and for this reason chemists are by no means agreed as to the proportions in which the different ones are present. It is well established that in woman's milk the soluble proteids, lactalbumin, etc., are in excess of the insoluble casein, Koenig giving the proportion as 5 to 4; in cow's milk, on the other hand, the proportion of the soluble proteids is much smaller than the insoluble, the latest writers giving the proportion as 1 to 3.

The casein * of cow's milk is readily coagulated by rennet, acids, and many metallic salts. The curd formed by the gastric juice is tough and firm and dissolves slowly by the action of the digestive fluids. The casein of woman's milk is not regularly coagulated by rennet, and only slightly and with difficulty by acids and metallic salts. The curd formed by the gastric juice is loose and flocculent, and is readily and completely dissolved. It is this difference in the proteids which presents the greatest difficulty in the use of cow's milk for infant feeding.

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* By Haliburton and some other chemists the term *caseinogen* is given to this proteid as it exists in milk. When this is acted upon by rennet it splits up into two substances: One, the firm, insoluble coagulum to which only the term casein is applied; the other, a soluble proteid which is known as whey-proteid; this is present in but small amount. Those who use the term casein to designate the proteid as it exists in milk refer to the curd formed by the action of rennet in the stomach as *paracasein*.

The inorganic salts in cow's milk are a little more than three times as abundant as in woman's milk. The most important differences in the composition of these salts are shown in the following analyses:

Ash in 100 Parts of Milk (Bunge).

	Woman's.	Cow's.
Potassium oxide	·0703	·1760
Sodium oxide	·0257	·1110
Calcium oxide	·0343	·1590
Magnesium oxide	·0065	·0210
Ferric oxide	·0006	·0003
Phosphoric acid	·0469	·1970
Chlorine	·0445	·1690
Total	·2288	·7970

It will be noted that cow's milk contains relatively a much larger amount of calcium phosphate and a smaller amount of potassium salts and of iron oxide. The ash does not accurately represent the mineral constituents of milk. About 8 per cent of the phosphoric acid of the ash, according to Richmond, is derived from the phosphorus of the casein; while the traces of sulphuric and carbonic acid found are not true mineral constituents of milk. Most of the more recent analyses show the presence of citric acid in both woman's and cow's milk.

Cow's milk always contains a large number of bacteria, which increase in proportion to the age of the milk; woman's milk is either sterile or contains but a few cocci from the milk ducts.

Cream.—A great misapprehension exists as to its composition. It is often spoken of as if it were entirely different from milk. It should rather be regarded as milk which contains an excess of fat.

Cream is obtained either by skimming—the gravity process—or by the use of a centrifugal machine known as a separator. The latter process has the advantage in point of time, as centrifugal cream can be put upon the market from twenty-four to thirty-six hours earlier than gravity cream. It is, however, attended by a slight disadvantage, as it may break up mechanically some of the fat-globules, so that after heating they may form a thin oily layer at the top of the bottle.

The following table gives the composition of an average milk and of centrifugal cream of different densities removed from the same milk:

	Whole milk.	CREAM.				
		I.	II.	III.	IV.	V.
Fat	4·00	8·00	12·00	16·00	20·00	40·00
Sugar	4·50	4·50	4·20	4·05	3·90	3·00
Proteids	3·50	3·40	3·30	3·20	3·05	2·20
Salts	0·75	0·70	0·65	0·60	0·55	0·45

These will be spoken of hereafter as 8-per-cent cream, 12-per-cent cream, 16-per-cent cream, etc., as indicating the amount of fat which they contain.

The percentages of proteids and sugar in the 8- and 12-per-cent creams are but little lower than in milk; in the very rich creams they are reduced by about one-third.

It is unfortunate that no standard exists as to what shall be sold as cream. In New York, cream sold may contain anywhere between 8 and 40 per cent fat. The very rich, centrifugal cream has from 35 to 40 per cent fat; the ordinary centrifugal cream has about 18 to 20 per cent. Most of the gravity cream sold has from 16 to 20 per cent fat. It is possible to obtain from the milk laboratory cream of any desired percentage.

None of the methods for determining the fat in milk is applicable to cream, except the Babcock or Leffman and Beam test.

Top-Milk.—To secure a milk for infant feeding which is fresh and at the same time one which contains an extra amount of fat, the practice has come largely into vogue of using the upper portion—a third, fourth, or fifth from milk purchased and delivered in bottles—after it has stood only a few hours. To this the term "top-milk" or "upper-milk" has been given. Different percentages of fat may be obtained by varying the amount removed and the length of time the milk has been allowed to stand. Top-milk and thin cream are practically identical in composition, although they may differ in freshness.

If cow's milk from a mixed herd is put into bottles soon after it is drawn and rapidly cooled, it will be found that after four hours the upper fourth will contain nearly all the fat that will rise as cream, and that the upper layers will have nearly the same percentage of fat whether the milk has stood for four hours, for eight hours, or over night. This has been demonstrated in a series of experiments made for me by Messrs. Upton & Jeffers, at the Walker-Gordon Farm at Plainsboro. After the milk had been standing under the conditions mentioned, fat-tests were made with the Babcock apparatus of the different four-ounce layers of bottled milk which contained originally 4 per cent of fat. The different layers were carefully removed with a siphon, with the following results:

Percentage of fat in—	After four hours.	After eight hours.	Over night.
Upper 4 oz.	20·50	21·25	22·00
Second 4 oz.	6·00	6·50	6·50
Third 4 oz.	1·50	1·40	1·00
Fourth 4 oz.	1·20	1·00	0·30
Fifth 4 oz.	1·00	1·00	0·20

Each of these percentages represents the averages, each test having been repeated many times, 110 different tests in all having been made. It will be seen that after four hours the composition of the separate layers does not change very much with the period of standing. With this knowledge, it becomes a comparatively simple matter to secure almost any desired percentage of fat by simply varying the number of ounces removed from the upper part of the quart.*

This will of course not be the same with all milks, but will vary considerably according as the supply is from a good herd of selected cattle of mixed breeds (average 4 per cent fat), a Jersey or Alderney herd (5·25 to 5·50 fat), or from widely scattered farms such as make up the general supply of any large town or city (3·25 to 3·50 fat). It is therefore absolutely necessary for the physician to know with which one of these he is dealing, if the milk for infant feeding is to be modified at home from the different layers of top-milk. More mistakes are made just here than at any other step in this method of feeding.

The tables given below are sufficiently accurate for home modification, provided the fat percentage of the whole milk is known.

From 4 per cent Milk.

To secure approximately a 10% fat, remove the upper 11 oz., or about one third.
" " " 7% " " " 16 " " one half.

From 5·25 to 5·50 per cent (Jersey) Milk.

To secure approximately a 10% fat, remove the upper 15 oz., or nearly one half.
" " " 7% " " " 24 " " three fourths.

From 3·25 to 3·50 per cent Milk.

To secure approximately a 10% fat, remove the upper 8 oz., or about one fourth.
" " " 7% " " " 11 " " one third.

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* A similar plan on a large scale may be followed in institutions by using an apparatus known as the "Cooley creamer." This consists of a wooden tank lined with metal, made of different sizes, holding two, four, or more cans of milk. The cans hold eighteen quarts, and are so covered that they can be submerged. The bottom of the can is inclined, and at the lowest point is placed a faucet. In the side is a glass window, so that the cream level can be distinctly seen. The cans are filled and placed in a tank of ice-water; after six or twelve hours the lower portion is drawn off and the upper creamy layer left behind. In this way a cream of 7 or 10 per cent may be obtained. The Cooley creamer may be obtained at Bellows Falls, Vt.

The physician should make or have made with the Babcock apparatus several fat tests of a given milk supply in order to obtain a basis upon which to make his calculations, and also of his top-milk to control his results. In general it is wise for one who has much to do with infant feeding to have his patients take milk from the same supply to secure uniformity in his results.

In or near large cities it is possible to obtain from the milk laboratories milk with any desired percentage of fat. This of course greatly simplifies the whole matter. How top-milk of different percentages is used will be considered under The Home Modification of Milk.

MILK STERILIZATION.—The term *sterilization* is widely and rather loosely used to signify the heating of milk for the destruction of germs. It should, however, be borne in mind that none of the methods commonly employed renders milk sterile in the bacteriological sense of the word. What is accomplished is the destruction of such pathogenic germs as may be present, and from 95 to 99 per cent of the other bacteria, so as to retard for a considerable time the ordinary fermentative changes. The preservation of milk for infant feeding, by boiling it in small bottles, was advocated by Jacobi many years ago.

The advantages of sterilizing milk are obvious. When we consider the enormous number of bacteria present in cow's milk with the usual methods of handling, and that none of these, so far as is now known, are advantageous, but that they are frequently the cause of disease, it is not strange that after its introduction by Soxhlet in 1886 the practice of heating milk used for infant feeding was rapidly adopted all over the world. Following him, the earlier experiments in sterilization were made at 212° F., usually continued for an hour and a half, and this temperature is still largely employed on the Continent of Europe. Even this does not render milk safe for very long. Spores are not destroyed, and at ordinary room temperatures spore-bearing bacteria may soon develop in such numbers as to make the milk dangerous. Since some of these bacteria act upon the milk-proteids and not upon the sugar, such milk may not be sour, and hence its danger may not be recognised.

There are disadvantages in heating milk. The change in taste and the constipating effects of sterilized milk were soon noticed; other alterations were not so evident and have more recently come to be appreciated, although many of these are not yet fully explained. Some of the lactose is converted into caramel, causing a slight change in colour; the lactalbumin is partially coagulated, this beginning at 160° F. (70° C.); the casein is rendered less coagulable by rennet, and appears to be acted upon more slowly both by pepsin and trypsin; Rettger has shown that when milk is heated above 185° F. (85° C.) a volatile sulphide is liberated, conclusive evidence of a change in the proteids; the organic phosphorus is changed into an inorganic phosphate; citric acid is partially precipitated as calcium citrate, and some lime salts, which are usually soluble, are converted into insoluble compounds. Some changes also occur in the fat. Moreover, certain natural ferments in fresh milk, believed to be of value in digestion, are destroyed by heat.

Many of these changes are but imperfectly understood, and some of them are doubtless without any injurious effect upon nutrition. There is, however, one important clinical reason for believing that the nutritive properties of milk are impaired by heating to 212° F.—viz., the occurrence of scurvy in infants who are fed upon such milk for a long time. Of 379 cases of infantile scurvy brought together in the Report of the American Pædiatric Society in 1898, sterilized milk was the previous diet in 107. At least a score such cases have come under my own notice. Again and again cases of scurvy have been cured by simply ceasing to sterilize the milk.

Sterilizing at Lower Temperatures.—Pasteurizing Milk.—To obviate the disadvantages above referred to, the practice has come largely into use in America of employing much lower temperatures for milk sterilization, owing chiefly to the work of Freeman (New York) and Russell (Wisconsin).

At first 167° F. (75° C.) was used; subsequently, however, a lower temperature was found sufficient, and 150° to 155° F. (65° to 68° C.) are the temperatures which are now generally employed. These temperatures are maintained from twenty to thirty minutes. This is sufficient to kill the bacilli of tuberculosis, diphtheria, and typhoid fever, and from 98 to 99·8 per cent of all the other bacteria in milk. Most of the objectionable changes produced in sterilized milk are avoided when the temperature is raised only to 155° F. (68° C.), while it accomplishes the purpose for which milk is heated. The advantages of this form of sterilization are therefore obvious. But spores are not destroyed, and such milk requires special handling. It should always be rapidly cooled and kept at a low temperature. Pasteurized milk should be used within a few hours after heating; no attempt should be made to keep it more than twenty-four hours, even upon ice.*

Pasteurization vs. High-temperature Sterilization.—From what has already been said it would appear that the argument is altogether in

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* Quite distinct from the process just described is that known as *commercial pasteurization*. In this, by passing milk through hot pipes, it is heated to temperatures ranging from 140° F. for several minutes to 160° F. for a very brief period, usually for 5 to 30 seconds. Such heating destroys from 90 to 99 per cent of the bacteria ordinarily found in milk. According to the experiments made in the laboratory of the New York Health Department, a temperature of 160° F. maintained for 30 seconds under usual conditions kills typhoid, diphtheria, and colon bacilli. In a small percentage of experiments about 1 in 100,000 of these bacteria withstood this exposure.

By this treatment (160° for 30 seconds) the great majority of tubercle bacilli, which are the most resistent of the bacteria exciting disease that are found in milk, are either favour of pasteurization. The lowest temperature and the shortest time that will surely destroy the objectionable bacteria in milk would seem to merit general adoption. Pasteurization, however, requires considerable care, intelligence, and special apparatus; if not properly done it may be worse than nothing. Moreover, pasteurized milk can not, in very hot weather, be kept without ice as long as it may be necessary to keep milk. Sterilization at 212° F. (100° C.) is much simpler; it may be done with many simple and inexpensive forms of apparatus or even without any special apparatus. Where no ice is available, it is certainly safer in hot weather than pasteurization. Among the poor of our large cities, in summer, heating to 212° for an hour is to be advised as the most satisfactory, and indeed the only efficient, method of sterilization. It should not be forgotten that the use of such milk as the sole diet for a long time is attended with a certain amount of risk; and one should always be on the watch for the soreness of the legs and the spongy gums that indicate the beginning of scurvy, as well as for the more general symptoms of malnutrition. Heating to 212° F. on two or three successive days is also to be recommended where milk must be kept for one or two weeks, as upon ocean journeys.

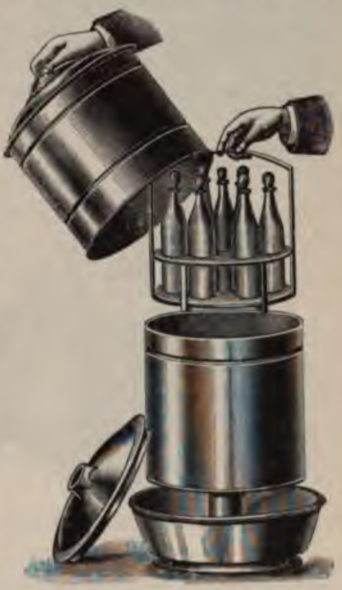
FIG. 29.—The Arnold sterilizer.

Methods of Sterilization.—Milk should be sterilized preferably in small bottles, each one of which contains a sufficient quantity for one

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killed or so injured that they cannot infect. On the average about $\frac{1}{10}$ of 1 per cent survive; 160° for one minute usually kills all.

The pasteurized milk of commerce which is extensively sold in many large cities is chiefly milk that has been heated for from 5 to 30 seconds in the manner described. Such a destruction of bacteria as is accomplished makes it possible to keep milk in warm weather a much longer time before souring occurs. It is therefore a great advantage to the dealer and he is likely to depend upon it rather than upon adequate icing and cleanliness in handling his milk. There are some serious objections to commercial pasteurization. Milk so heated should be quickly cooled, should be received into sterilized vessels and kept at a low temperature (below 50° F.). If these precautions are not taken bacteria develop rapidly and the milk may after 24 hours be more dangerous than if it had not been heated at all; since, unlike raw milk, it does not usually sour and reveal its contaminated condition. Commercial pasteurization should be permitted only under the most careful restrictions, and the can or bottle containing pasteurized milk should indicate the degree and time of heating. Its practical advantages have as yet not been fully demonstrated.

feeding. These bottles may be plugged with cotton or corks, or special rubber stoppers may be used. If the latter, they should be loosely inserted during the process and pressed tightly home at its completion. Soxhlet's apparatus may be employed, or Arnold's (Fig. 29), or any one of a half dozen others sold in the shops. All that is really necessary is to expose the bottles on all sides to live steam in a closed vessel. It can be done effectively in any tin vessel which has a closely fitting cover and a perforated bottom, and which can be placed over a pot of boiling water. Sterilization at 212° is usually continued for one hour. The bottles should then be cooled in water as quickly as possible and placed upon ice or in the coolest place available.

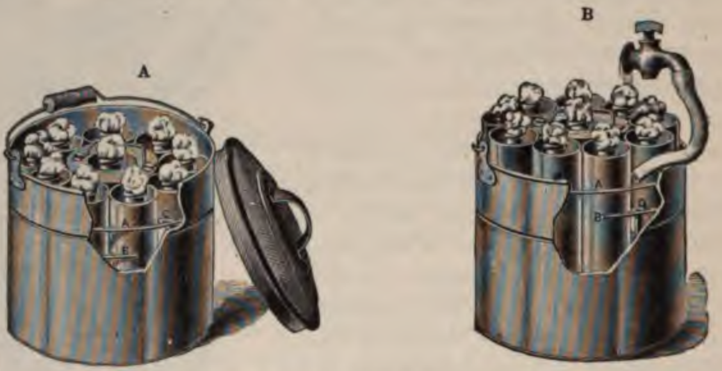

Fig. 30.—Freeman's pasteurizer. A, bottles in position for heating; B, method of cooling.

A simple apparatus for pasteurizing milk has been devised by Freeman (Fig. 30). In this the temperature is raised to 155° F. (68° C.) by hot water, while cold water is used as a conducting medium.* Another useful form of apparatus is that of the Walker-Gordon Laboratory Company, which contains a thermometer so that any desired temperature can be secured. An essential step in pasteurizing milk is rapid cooling. After forty-five minutes the bottles should be removed from the pasteurizer and placed in tepid water and afterward in ice-water, where they should remain half an hour before being placed in the cold room or ice chest.

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* Freeman's apparatus is used as follows: The pail is filled to the groove with water, which is then raised to the boiling point. The bottles of milk are dropped into their places in the cylindrical cups, sufficient water being poured into each cup to surround the bottle, this water acting as the conductor of heat. The pail is now removed from the stove and placed upon a board or other non-conductor, and the receptacle containing the bottles of milk is set inside and the cover replaced. The volumes of milk and water have been so calculated that in ten minutes they are both at a temperature of 155° F. The water contains heat enough to maintain this, with very slight variations, for twenty minutes. In half an hour the bottles of milk are removed and cooled rapidly by being placed in a water-bath, the water being changed once or twice; or, better, by setting the pail in a sink and allowing the cold water to run from a faucet through a piece of rubber pipe into the pail, overflowing into the sink. This rapid cooling is very important. The bottles are then put in the refrigerator. This apparatus may be obtained from James Dougherty, 411 West Fifty-ninth Street, New York. (See Archives of Pædiatrics, August, 1896.)

Limitations of Milk Sterilization.—While pasteurizing or sterilizing milk kills nearly all the living organisms, it destroys few of the spores, and probably but a small proportion, if any, of the toxins. Before sterilization milk may contain the products of bacterial growth in such quantity and of such a character as to render it unfit for food. Even though just sterilized, it may be poisonous to an infant. It is therefore impor- that sterilization be done at the earliest possible moment.

Again, the fewer the spores and spore-bearing bacteria which the milk contains, the more effective the sterilization. Both these have a very close relation to the amount of dirt contained in the milk. Hence the cleaner the milk the better will be the result.

The opinion has gained a certain amount of currency that, if milk has only been "sterilized," it may be fed to a young infant without further modification; but it should be distinctly understood that sterilized milk requires the same modification for infant feeding as raw milk. There is no evidence to show that its digestibility is in any way enhanced by the process of heating.

The sterilization of milk is chiefly valuable by enabling us to feed with safety milk in which, though it may be forty-eight hours old, no important fermentative changes have occurred, because the great proportion of the common bacteria have been destroyed as well as any pathogenic organisms present. As a therapeutic measure sterilized milk is useful in various forms of gastric or intestinal infection such as typhoid fever, dysentery, diarrhœa, etc. In certain of these conditions no milk is admissible; at other times sterilized milk may be given when raw milk would be harmful.

Shall all Milk used for Infant-feeding be Sterilized?—Only the cleanest milk can safely be used in summer without heating. So long as milk is produced and handled as the bulk of it is at present, not being delivered in large cities until it is considerably over twenty-four hours old, and not consumed until over forty-eight hours old, heating should invariably be practised in hot weather; also, where there is any doubt about the dairy hygiene or the health of the cows; and finally, during epidemics of typhoid fever, diphtheria, and scarlet fever.

It is quite possible to produce milk which does not need sterilization; the conditions to be fulfilled have been already detailed. There are special dairies supplying such milk to many of our large cities, and their number may be very greatly increased if the medical profession will use its influence in this direction. My personal preference for routine use in infant-feeding is for a milk so clean and fresh that it may be safely given without heating, feeling as I do that all forms of sterilization do impair, though possibly only to a slight degree, its nutritive properties. It should, however, be borne in mind that there are some delicate infants with feeble digestion who thrive better upon sterilized milk than upon raw milk in which the bacterial content is quite low; for, even though not numerous, bacteria may yet do harm to such children. Healthy infants with good digestion may do well upon raw milk even though the number of bacteria is quite large—i. e., 100,000–1,000,000 per c. c.; while delicate infants or those with digestive disturbances may be seriously affected by such milk. In the country where milk is obtained fresh and used before it is twenty-four hours old, sterilizing is usually unnecessary if the cows are healthy and the milk properly handled.

PEPTONIZED MILK.—Milk is peptonized through the agency of a substance derived from the pancreas, usually that of the pig. This is known in the market as "extractum pancreatis," the active ferment being the trypsin. As this acts only in an alkaline medium, bicarbonate of soda should first be added to the milk. The purpose of peptonizing is to secure a partial digestion of the casein of milk before feeding.

Partially Peptonized Milk.—The process is as follows: * One pint of fresh cow's milk and four ounces of water are put into a bottle, and a powder added containing five grains of extractum pancreatis and fifteen grains of bicarbonate of soda. This is kept at a temperature of 105° to 115° F., or about as warm as the hand can bear comfortably, best by placing the bottle in warm water. It should be shaken from time to time. For partial peptonization, the process is continued for from six to twenty minutes. The peptonizing powder is sold in glass tubes and in tablets. The tubes are to be preferred, as being less liable to deteriorate with age. Milk which has been peptonized ten minutes is not altered in taste; if, however, the process is continued for twenty minutes, a slightly bitter taste is noticed from the formation of peptones. This increases with the duration of the process of artificial digestion. If it is desired to arrest this after ten minutes, the milk may be raised to the boiling point, which destroys the ferment, or its activity may be stopped by placing the milk upon ice. If the milk is to be fed at once, neither of these procedures is necessary. If it is to be kept for several hours, scalding is more certain to arrest the change than lowering the temperature.

Completely Peptonized Milk.—The process is exactly the same as the above, except that it is continued for two hours, which is generally required for the conversion of all the proteids into peptones. The addition of acetic acid to such milk produces no coagulation. Although completely peptonized milk is quite bitter, this is not an obstacle to its use for young infants, who after the first or second bottle do not usually object to its taste. For those who are a little older, the bitter taste may be covered by lemon-juice and sugar—one even teaspoonful of cane sugar and two teaspoonfuls of lemon-juice being added to each four ounces of the milk.

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* Fairchild's process.

Peptonized milk is to be modified according to the age of the child and the condition of his digestion. Peptonized milk is a valuable resource in chronic cases where there is feeble proteid digestion, and during attacks of acute indigestion in infancy. In acute attacks, completely peptonized milk is usually preferable to that which has been partially peptonized. It is not advisable to continue its use indefinitely, for in this case the stomach gradually becomes less and less able to do its work. At most, peptonization should be used only for a month or two at a time; as the child improves the amount of the powder used is gradually diminished and the time of peptonizing shortened.

CONDENSED MILK.—This is prepared by heating fresh cow's milk to 212° F. to destroy the bacteria and then evaporating *in vacuo* at a low temperature to a little less than one-fourth its volume.* It is preserved in tin cans, usually with the addition of cane sugar in the proportion of about six ounces to a pint. The changes, therefore, to which the milk has been subjected are: evaporation of a part of the water, sterilization, and the addition of cane sugar. Fresh condensed milk to which no sugar has been added is to be obtained in many large cities.

The composition of condensed milk is shown in the following table; also the results obtained when it is diluted with six, twelve, and eighteen parts of water, as usually fed:

	Condensed milk.†	With 6 parts of water added.	With 12 parts of water.	With 18 parts of water.
	Per cent.	Per cent.	Per cent.	Per cent.
Fat	6·94	0·99	0·53	0·36
Proteids	8·43	1·20	0·65	0·44
Sugar { Cane, 40 44 Milk, 10·25 }	50·69	7·23	3·90	2·67
Salts	1·39	0·17	0·10	0·07
Water	31·30	90·49	94·82	96·46

The dilution with twelve parts of water is that most frequently employed, although eighteen is often used for very young infants.

The reasons both for the success and for the failure of condensed milk as an infant-food, are apparent from a study of its composition as it is ordinarily used. As a temporary food it is often useful, first because it has been sterilized, but chiefly because both the fats and the proteids of cow's milk have been reduced by the usual dilution to a point at which an infant with a very weak digestion can manage them, while it furnishes an abundance of sugar, the easiest thing for an infant to digest. During the first few months of life it is often apparently very successful for these reasons, but it should not be continued indefinitely. It is rare to see an infant fed exclusively upon it who does not show more or less evidence of rickets. Condensed milk fails as a permanent food because it consists too largely of carbohydrates, and is lacking in fat. It is admissible for temporary use during attacks of indigestion, for infants with feeble digestion, especially in summer, for very young infants during the first two or three months, or among the very poor, where the cow's milk which is available is still more objectionable. It should not be continued as a permanent food where good, fresh cow's milk can be obtained. In travelling it is often the most convenient as well as the safest food to use. It should be diluted twelve times for an infant under one month, and from six to ten times for those who are older.

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* Process followed by the Borden Condensed Milk Company.

† Analysis of Borden's Eagle-brand condensed milk made for the author by E. E. Smith, Ph.D., M.D.

The fresh condensed milk has not the disadvantage of the addition of a large amount of cane sugar, and requires essentially the same modification as ordinary cow's milk. For the poor in cities it is sometimes the best infant-food available. For routine use it should be diluted with from eight to twelve parts of water, and sugar added.

KUMYSS.—The original kumyss made by the Tartars was fermented mare's milk. In this country it is made from cow's milk. The ferment used by the Tartars was kefir grains, consisting of two forms of the ordinary yeast plant and great numbers of lactic-acid bacilli. Kumyss is sometimes made from skimmed milk, but usually from the whole milk, with the addition of cane sugar and a small proportion (about one-sixteenth) of water. The process now most commonly employed is started with ordinary yeast, causing a vinous fermentation. The best results are obtained when this is carried on at a temperature of from 60° to 70° F. in corked bottles. It requires a week or ten days.*

Kumyss contains alcohol, carbon dioxide, lactic acid, and traces of butyric and acetic acids. The casein is first coagulated, and then broken up into minute particles by agitation. Some of it is probably converted into albumoses. Kumyss has an acid reaction and a taste somewhat resembling buttermilk; at first it is often disagreeable, but a fondness for it is soon acquired.

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* The following is perhaps the best formula for the domestic manufacture of kumyss: One quart of fresh milk, half an ounce of sugar, two ounces of water, a piece of fresh yeast cake half an inch square; put into wired bottles, keep at a temperature between 60° and 70° F. for one week, or 85° to 95° F. for twenty-four hours, shaking five or six times a day, and then put upon ice.

Kumyss.

	Made from mare's milk (Koenig).	Made from cow's milk (Koenig).	Made from skimmed milk (Koenig).	Brush's kumyss (Doremus).
Fat	1·46	1·83	0·88	1·91
Proteids	2·24	2·66	2·89	2·04
Sugar	1·47	4·09	3·95	3·26
Alcohol	1·91	1·14	1·38	0·62
Lactic acid	0·91	0·55	0·82	....
Acid	....	....	....	0·30
Carbon dioxide	....	....	....	0·44
Salts	0·42	0·43	0·53	0·44
Water	91·29	89·30	89·55	90·99

The advantages of kumyss are due to the alcohol, carbon dioxide, and lactic acid, and to the changes which have taken place in the casein of the milk. It is more useful for older children than for infants. It is a valuable resource in many forms of indigestion, both of the gastric and intestinal varieties.

For infants, kumyss should be diluted, generally with an equal quantity of water. Many take it better if the gas has been allowed to escape by standing a few minutes. It is important that it be reasonably fresh.

MATZOON.—Matzoon, or Zoolak, is a form of fermented milk first used in Asia Minor. The process of manufacture is given by Dadirrian as follows: The milk is first sterilized by boiling; a ferment is then added which is probably some form of yeast. The fermentation is begun at a temperature of about 105° F. and continued in an open vessel for twelve hours, the temperature being gradually reduced to about 70° F., after which it is cooled, bottled, and kept on ice. A slow fermentation continues after bottling, so that the older matzoon contains a little carbon dioxide and is more sour than the fresh. It keeps on ice for two or three weeks. It is a thick fluid with a taste resembling sour cream. For infant-feeding it should be diluted with water and fed with a spoon, as it is too thick to be drawn through a nipple.

Matzoon, or Zoolak (Leeds).

Proteids	3·48
Fat	3·49
Milk sugar	3·68
Lactic acid	0·90
Alcohol and other products of fermentation	0·13
Mineral salts	0·69
Water	87·63
	100·00

By the process there is a decomposition of the milk sugar into alcohol, lactic and carbonic acids. The changes in the proteids are similar to those in kumyss. It is used in the same conditions.

Buttermilk.—When made from fresh cream this differs but little from skimmed milk, or milk from which the fat has been removed by a separator. Usually, however, as the churned cream is slightly sour, buttermilk contains an appreciable amount of lactic acid. To this chiefly its peculiar taste is due. The proportion of lactic acid depends upon the degree to which the souring process has been allowed to go.

Buttermilk (Vieth).

Fat	0·50
Milk sugar	4·06
Lactic acid	0·80
Proteids	3·60
Inorganic salts	0·75
Water	90·39
	100·00

It is a valuable form of food in chronic intestinal indigestion and in diarrhœal disease. The value of buttermilk in infant-feeding depends upon its low fat, possibly also upon the lactic acid present, and upon some slight change in the milk proteids from the agitation.

A good formula is, buttermilk, one quart; barley flour, two even tablespoonfuls; water, four ounces. Cook slowly, constantly stirring, for twenty minutes; then add two teaspoonfuls of cane sugar, or, better, one tablespoonful of milk sugar.

Junket, Curds and Whey.—Junket is made as follows: To one pint of fresh lukewarm cow's milk are added two teaspoonfuls of essence of pepsin, liquid rennet, or a junket tablet. It is stirred for a moment and then allowed to stand until firmly coagulated. It is given cold. The only change which has taken place is the coagulation of the casein—such as occurs in the stomach as the first step in digestion. Junket is useful in the feeding of older children, but should not be given to infants.

Whey.—The milk is coagulated with rennet as above, the curd is then broken up, and the whey strained off through muslin. The composition of whey varies somewhat, depending upon the way in which it is prepared. If it is desired to have as little fat as possible, skimmed milk should be used, and the whey should be strained through fine muslin without pressure. If it is desired to retain some of the fat, whole milk may be used, coarser muslin, and more pressure. The proteids of whey are chiefly lactalbumin with a small amount of lactoprotein and lactoglobulin.

Whey used alone is valuable in the acute indigestion of infants. It is the basis of the milk modifications, the purpose of which is to give a larger proportion of lactalbumin and a smaller proportion of casein than exist in any dilution of cow's milk. Such modifications of milk have a wide application and form a valuable addition to our means of infant-feeding. Wine whey may be made by adding sherry, usually in the proportion of one part to sixteen of whey.

Whey.

	Average 46 analyses (Koenig).	From whole milk (Adriance).	From fat-free milk (Adriance).
Proteids	0·86	0·94	1·17
Fat	0·32	0·96	0·04
Sugar	4·79	5·49	5·36
Salts	0·65	0·48	0·52
Water	93·38	92·13	92·91
Total	100·00	100·00	100·00

BEEF PREPARATIONS.

The nutrient properties of these preparations are to be measured by the amount of albumin they contain, their stimulant properties by the proportion of extractives.

Beef Juice.—Expressed beef juice is made as follows: A piece of lean steak is slightly broiled, and the juice pressed out by a meat-press or a lemon-squeezer. Two or three ounces can ordinarily be obtained from one pound of steak. This is seasoned with salt and given cold or warm, but not heated sufficiently to coagulate the albumin in solution.

Another excellent method of making beef juice without cooking, is by taking one pound of finely-chopped lean beef and eight ounces of water and allowing this to stand in a covered jar upon ice from six to twelve hours. The juice is then squeezed out by twisting the meat in coarse muslin. It is seasoned with salt and given as above. This is not quite so palatable as that obtained by the first method, because it contains a smaller proportion of extractives. It can be made so, however, by the addition of sherry wine or celery salt. If the raw juice is added to milk in the proportion of two or three teaspoonfuls to each feeding, the taste will not be noticed. The milk should not be warmed above 100° F. before the addition of the juice.

The composition of the two products is shown in the table on the following page.

The only difference in the two preparations is that the first contains about twice as much of the extractives. The second process is much more economical, as more than three times as much juice can be obtained from a given quantity of beef. If a stronger juice is desired, the amount of proteids may be doubled by using only four ounces of water. This is preferable for all except young infants.

Beef extracts are not to be considered in any sense as foods. Kemmerich has shown that animals receiving nothing else died of starvation, and sooner even than when everything was withheld. According to Chittenden, they contain no nitrogen in the form of proteids, but only in combination with the soluble extractives. They are stimulants, and as such are often useful.

*Beef Juice.**

	I. Expressed juice from 1 lb., warm process; quantity, 2½ oz.	II. Cold process, 1 lb. beef, 8 oz. water; quantity, 8½ oz.
Proteids	2·90	3·00
Fat	0·60	....
Extractives	3·40	1·90
Salts	0·20	0·20
Water	92·90	94·90
	100·00	100·00

Of the preparations of beef in the market probably the best are Mosquera's beef jelly, Armour's beef juice, Wyeth's beef juice, and Valentine's beef extract. Many products sold as beef preparations, such as liquid peptonoids, panopeptone and others, contain from 15 to 20 per cent of alcohol, and should, therefore, be classed as stimulants rather than as nutrients. For infants they must be well diluted. Beef preparations are valuable for older children in many cases of general malnutrition.

Raw scraped beef, or that which has been slightly cooked, is easily digested by most young children. There are many conditions in which other forms of proteid, particularly casein, are not well borne, and indeed can not be taken at all, where children even as young as twelve months appear to digest this beef-pulp without any difficulty. It should be made from very rare or raw steak, finely scraped and well salted. A tablespoonful may be given at one feeding to a child of eighteen months. In nutrient properties this far exceeds most of the beef preparations in the market. The alleged danger of tapeworm from the use of raw meat is in this country so slight that it may be disregarded.

Broths.—Animal broths may be made from mutton, veal, chicken, or beef. A good formula for general use is the following: One pound of lean meat, one pint of water; stand for two hours, then cook over a slow fire for two hours down to half a pint. After it has cooled, skim off the fat and strain through a cloth. The composition of a broth so made is given by Cheadle as follows:

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* Analysis made for the author by E. E. Smith, Ph.D., M.D.

PLATE III.

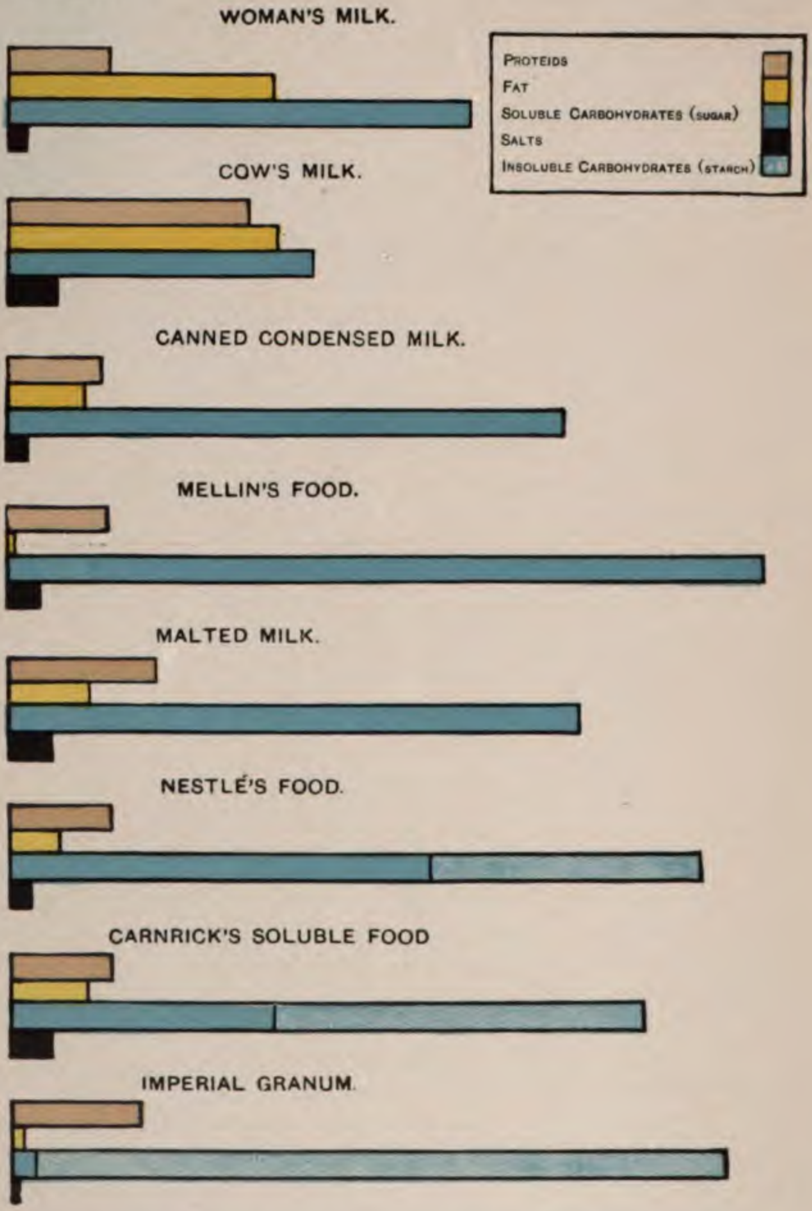

Chart showing the solid ingredients of various infant foods as compared with those of woman's milk

Beef Broth.

Proteids	1·02
Extractives	1·82
Fat	....
Salts	0·88
Water	96·28
	100·00

From their composition it will be seen that broths are not very nutritious; they are, however, quite stimulating, and are at times useful, particularly where milk must be temporarily withheld. They are, however, not adapted to prolonged use alone. Broths which have been thickened with either barley or rice flour are useful for children in the second and third years.

CEREALS.

Barley Water.—This may be made either from the grains or from the barley flour. When the grains are used, the following is the formula which I have been accustomed to employ: To two tablespoonfuls of pearl barley, add one quart of water, and boil continuously for six hours, keeping the quantity up to a quart by the addition of water; strain through coarse muslin. It is an advantage to soak the barley for a few hours, or even over-night, before cooking. The water in which it is soaked is not used. When cold this makes a rather thin jelly. Its composition by analysis is as follows:

Barley Water.

Starch	1·63
Fat	0·05
Proteids	0·09
Inorganic Salts	0·03
Water	98·20
	100·00

Almost an identical product may be obtained in an easier way by using either the prepared barley flour of the Health Food Company, New York, or Robinson's barley, two drachms—one even tablespoonful—to each twelve ounces of water, and cooking for twenty minutes.

Rice Water, Oatmeal Water, etc.—These may be made in the same manner as the barley water, using the same proportions either of the flour or the grains. These are useful as additions to milk for healthy infants who have reached the age of seven or eight months; they may also be given in many cases of acute or chronic indigestion where milk must be omitted or given in small quantities. When there is a tendency to constipation oatmeal is preferred; when to looseness, barley or rice water. The digestibility of cereals is greatly increased by the addition of diastase; such preparations as Forbes's diastase, maltzyme, Trommer's extract of malt, taka-diastase, cereo, etc., may be employed.

INFANT-FOODS.

It is not possible, nor even desirable, for a physician to know all about the infant-foods with which the market is flooded. He should, however, know at least that they are not perfect substitutes for breast-milk, that as permanent foods they are greatly inferior to properly modified cow's milk, and that as often used by the laity, and even by the medical profession, they are capable of doing and have done much positive harm. Rickets and scurvy have so frequently followed their prolonged use, especially when given without the addition of fresh milk, that there can be no escaping the conclusion that they were the active cause. The almost unanimous verdict of intelligent physicians is against their use as permanent foods. On the other hand, there are times when some of these preparations may be of considerable value, but chiefly for temporary use in pathological conditions. Here they are to be prescribed like drugs, but only with a very definite knowledge of exactly what they do and what they do not contain. The most commonly used infant-foods may be grouped as follows:

1. **The Milk Foods.**—Nestlé's food is perhaps the most widely known. The others closely resembling it in composition are the Anglo-Swiss, the Franco-Swiss, the American-Swiss, and Gerber's food. These foods are essentially sweetened condensed milk evaporated to dryness, with the addition of some form of flour which has been dextrinized; they all contain a large proportion of unchanged starch.

2. **The Liebig or Malted Foods.**—Mellin's food may be taken as a type of the class. Others which resemble it more or less closely are Liebig's, Horlick's food, Hawley's food, malted milk, and cereal milk. Mellin's food is composed principally (80 per cent) of soluble carbohydrates. They are derived from malted wheat and barley flour, and are composed chiefly of a mixture of dextrins, dextrose, and maltose.

3. **The Farinaceous Foods.**—These are imperial granum, Ridge's food, Hubbell's prepared wheat, and Robinson's patent barley. The first consists of wheat flour previously prepared by baking, by which a small proportion of the starch—from one to six per cent—has been converted into sugar. In chemical composition these four foods are very similar to each other, consisting mainly of unchanged starch which forms from seventy-five to eighty per cent of their solid constituents.

4. **Miscellaneous Foods.**—Under this head may be mentioned Carnrick's soluble food and Eskay's food. The composition of these is given in the table on the opposite page.

A better idea of the composition of these foods can be obtained by a study of the accompanying chart (Plate III), which shows their solid

The Composition of Infant-Foods.[*]

	Nestlé's food.	Mellin's food.	Eskay's food.	Malted milk.	Ridge's food.	Imperial granum.	Carnrick's food.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Fat	5·50	0·24	1·16	8·78	1·11	1·04	7·45
Proteids	14·34	11·50	5·82	16·35	11·81	14·00	10·25
Cane sugar	25·00	.....	.....	.....	.....	.....	.....
Dextrose	.....	.....	53·46†	.....	0·52	0·42	.....
Lactose (milk sugar)	6·57	.....		49·15‡	.....	.....	.....
Maltose	27·36	60·80	.....		.....	.....	.....
Dextrins		19·20	14·35	18·80	1·28	1·38	.....
Total soluble carbohydrates	58·93	80·00	67·81	67·95	1·80	1·80	27.08
Insoluble carbohydrates (Starch)	15·39	.....	21·21	.....	76·21	73·54	37·37
Inorganic salts	2·03	3·59	1·30	3·86	0·49	0·39	4·42
Moisture	3·81	4·73	2·70	3·06	8·58	9·23	3·42

constituents as compared with those of woman's milk. The essential features of the foods are seen at a glance—i. e., they are all composed principally of carbohydrates and are lacking in fat. Some of them contain a large proportion of unchanged starch. Furthermore, their proteids, though often sufficient in amount, are chiefly vegetable, not animal proteids. No one of them can be regarded in any sense as a proper substitute for breast-milk.

Some of these foods—Nestlé's and other milk foods, malted milk, cereal milk, and Carnrick's food, and even some of the farinaceous foods, like imperial granum—are advertised as substitutes for breast-milk and recommended for use alone. Others, such as Mellin's, Liebig's, and Eskay's foods, are intended to be used with milk. The use of any of the commercial foods alone is admissible only for short periods during derangements of digestion, when we wish to withhold for the time all fat and milk proteids. Their prolonged use almost invariably produces some grave disorder of nutrition, most frequently rickets or scurvy. Those foods which require in their preparation the addition of milk are open to less serious objections. They should not be used with condensed milk. When added to fresh milk they may serve a useful purpose in furnishing the additional carbohydrates required by an infant fed upon a diluted cow's milk. In such a case they take the place of milk sugar or cane sugar in the milk modification. That they themselves exert an important modifying influence upon cow's milk so as to increase its digestibility is certainly to be doubted. The group classed as farinaceous foods, since they furnish starch in a convenient and palatable form, may often be advantageously used as an addition to milk after the seventh or eighth month and during the second year.

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[*] With the exception of Nestlé's food and Carnrick's soluble food, these analyses were made for the author by E. E. Smith, Ph.D., M.D., of samples purchased in the open market, 1901. † Chiefly lactose. ‡ Largely maltose.

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sing. Among the classes of society where most
sing is very poor, but where every facility can be
artificial feeding, one should not be slow to adopt
of doubt. Among the poor and ignorant, however,
feeding can not be carried on with anything like the
success, one should persist in maternal nursing so long
possibility of success.

ursing *should not be attempted.*—(1) No mother of tuberculosis in any form, whether latent or active, infant; it can only hasten the progress of the disease at the same time it exposes the infant to the danger of) Nursing should seldom be allowed where serious com- been connected with parturition, such as severe hæmorrhage, puerperal convulsions, nephritis, or puerperal septicæmia. (3) If the mother is choreic or epileptic. (4) If the mother is suffering from any serious chronic disease or is very delicate, since great harm may be done to her without any corresponding benefit to the child. (5) Where experience on two previous occasions under favourable conditions has shown her inability to nurse her child. (6) When no milk is secreted. With reference to the fourth and fifth conditions, an absolute opinion can not always be given at the outset. My own inclination as a result

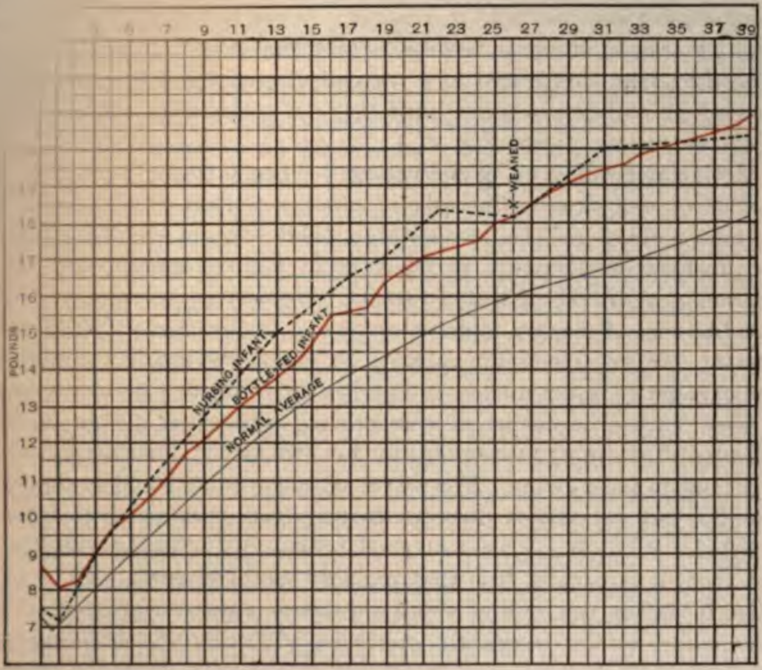

FIG. 31.—Weight curve of nursing and artificial feeding compared.

Both infants were strong, well nourished, and in good surroundings. The bottle-fed infant was never once put to the breast; fed from the milk laboratory. First formula: Fat 1 per cent, sugar 5 per cent, proteids 0·5 per cent. At six weeks taking: Fat 3 per cent, sugar 7 per cent, proteids 1·25 per cent. It will be observed that the nursing infant made more rapid progress during the first few weeks, while the bottle-fed infant more than made up for this between the fifth and ninth month, for weaning became necessary in the other child owing to the gradual failure of the mother's milk. The stationary weight was the result of this condition, and the irregular subsequent gain was incident to the change of food.

of increasing experience is not to allow nursing in either of these conditions, provided the means for proper artificial feeding can be commanded. The chances of success are so small and the difficulties are so increased by even a few weeks of bad nursing that I prefer not to put the child to the breast at all, even for the first two or three days. The breasts are bound up at once and kept bandaged. The theoretical objection that uterine contractions are not likely to be sufficient under these circumstances does not hold in practice. When one begins with healthy digestive organs, artificial feeding is very simple and almost invariably successful; how simple and how successful, one who is in the habit of allowing all children to nurse until they are manifestly upset by it, can hardly appreciate. (See Fig. 31.)

Artificial Feeding vs. Wet-Nursing.—When maternal nursing is impossible or undesirable, the milk of another woman would seem to be the most natural and best substitute. While this is theoretically true, the practical obstacles are so many as to put wet-nursing out of the question as a general method of feeding. We have in America no peasant class like that of Europe to draw upon; and in the class which furnishes most of our wet-nurses the capacity to nurse has steadily diminished. The expense of a wet-nurse—twenty-five to thirty-five dollars a month in New York—the danger of transmitting contagious disease, and the difficulty of obtaining proper care for her own infant, are all very serious objections to wet-nursing. The recent advances in artificial feeding have placed it now on quite a different footing from that which it formerly occupied. While it is true that good breast-milk is unquestionably the best food, it is equally true that properly modified cow's milk is a far better food than the milk of many wet-nurses who are employed. These facts added to the constantly increasing difficulty of obtaining good ones have caused wet-nurses to be pretty generally discarded, even in our large cities, where formerly no other substitute for maternal nursing was considered.

There are, however, some conditions in which they are necessary, even indispensable. Some infants, usually those who have been badly started, can not be made to thrive upon any form of artificial feeding. There are also many premature infants and some very delicate ones whose powers of assimilation are so feeble that they are reared under any circumstances only with the greatest difficulty, but whose chances of life are much increased by a good wet-nurse. Again, in young infants who have been suffering for some time from chronic indigestion and failing nutrition, the symptoms of acute inanition sometimes develop with great rapidity and severity. From such a condition, apparently hopeless, infants may sometimes be rescued by the timely assistance of a good wet-nurse.

The difficulties in the way of successful infant-feeding in foundling asylums and other institutions for young infants are such that in them wet-nursing should be employed whenever possible.

Mixed Feeding.—Mixed feeding, or a combination of nursing and artificial feeding, may be employed whenever the supply of the nurse is insufficient, also to relieve the mother from the strain of nursing entirely, and, during the later months, for the purpose of gradual weaning.

BREAST-FEEDING.

Care of the Breasts during Lactation.—For the safety of both mother and child it is essential that the most scrupulous attention be given to cleanliness. The nipples, and the breasts as well, should always be carefully washed after each nursing. Usually plain water is sufficient, or a weak boric-acid solution may be employed.

Nursing during the First Days of Life.—This is necessary, to accustom the child and the mother to the procedure, and to empty the breasts of the colostrum; it also promotes uterine contractions. All these results can be attained by putting the child to the breast on the first day once in six hours, on the second day once in four hours. The child gets from the breast only from four to six ounces a day during the first two days. Did it require more nourishment before the milk-flow is usually established, we may be sure that Nature would not have been so late with her supply. The common practice of administering to an infant a few hours old all sorts of decoctions, with the idea that because it cries it is suffering from colic, can not be too strongly condemned. A certain amount of crying is necessary. In exceptional circumstances, when an infant is unusually large and strong and cries excessively, it may be necessary to give food even on the first day; but this is not to be the rule. A little warm water, or a five-per-cent solution of milk sugar, should first be given; from two to four teaspoonfuls at a time are sufficient. If this does not satisfy the child, regular feeding should be begun on the second day. Should the milk be delayed beyond the second day, artificial feeding should then be begun at regular intervals.

Nursing Habits.—Good habits of nursing and sleep are almost as easily formed as bad ones, provided one begins at the outset. A vast deal of the wear and tear incident to the nursing period may be avoided if the child is trained to regular habits. Attention to these minor points often makes all the difference between successful and unsuccessful nursing. The physician must have a very clear notion of how often nursing is necessary, must give very explicit directions, and see that they are carried out. After the third day, for the first month, ten nursings in the twenty-four hours are quite sufficient, and no more should be allowed. An infant at this age can usually be depended upon to take at least one long sleep of from four to five hours in the course of the twenty-four.

For the rest of the day the child should be awakened, if necessary, at the regular nursing time, and put to the breast; this plan being continued until nine o'clock at night. It should then be allowed to sleep as long as it will, and but two nursings given between this hour and seven in the morning. In the course of two or three weeks a healthy infant can usually be trained to nurse and sleep with almost perfect regularity, frequently, when a month old, going six hours regularly at night without feeding. A trained nurse of my acquaintance states that out of thirty-three infants of which she had the care from birth, thirty-one were trained without difficulty in the manner stated. Of course, success in training must rest almost entirely with the nurse; but the physician should at least appreciate the importance of proper training and lend it his support. The great gain to the mother is, that she is enabled to have a quiet, undisturbed night. This has more to do with a good milk supply than any other single thing in connection with the mother's habits. So far as the child is concerned, regular habits of feeding and sleep, and regular evacuations from the bowels, which nearly always go with them, are most important factors in infant hygiene.

Schedule for Breast-Feeding.

AGE.	Number of nursings in 24 hours.	Interval during the day.	Night nursings between 9 P.M. and 7 A.M.
		Hours.	
First day	4	6	1
Second day	6	4	1
Third to twentieth day	10	2	2
Third to ninth week	8	2½	1
Third to fifth month	7	3	1
Fifth to twelfth month	6	3	0

These rules can be carried into effect with but little difficulty, and with great benefit to both mother and child. It is to be remembered that we are here speaking only of healthy children. The possibility of training children to eat and sleep in the manner described will be doubted only by one who has not made a careful trial of it. Relieving the mother of night-nursing after the child is five months old is of the greatest value, and will often enable her to continue lactation, when otherwise it would be brought to an abrupt termination. On no account should the child be allowed to sleep upon the mother's breast, nor in the same bed with the mother. The temptation to frequent nursing is thus largely removed. No mere sentiment in regard to these matters should be allowed to interfere with the plain dictates of reason and experience.

Symptoms of Unsuccessful Nursing during the Early Weeks.—Attempts at maternal nursing so often result in failure, jeopardizing the health, and even endangering the life of the child, that it becomes a matter of the greatest importance to decide this question of nursing aright, and as early as possible. On the one hand, one should not hastily wean a child on account of symptoms which may have no connection with the food, nor should one advise weaning when the indigestion from which the infant is suffering is due to causes which are temporary and remediable. On the other hand, nursing should not be continued simply because a conscientious mother desires it, when every indication points to failure. If artificial feeding is to be employed the difficulties are fewer when it is begun early than after the digestive organs have been deranged by several weeks of poor nursing. These cases form a very large group and present peculiar difficulties in practice. While a decision is being reached as to the ability of the mother to nurse, there is required close observation and a careful study of all the conditions, and even then the physician is liable to make mistakes in judgment the results of which may be serious.

The body-weight gives valuable information. The child does not gain or continues to lose after the usual initial loss of the first three or four days. Observations on the weight at least twice a week are necessary, and in cases presenting special difficulties the weight should be taken daily.

At times there may be no vomiting, diarrhœa, or even severe colic, yet the child may fret and worry continually, sleep but little, and show a general discomfort. In other cases definite symptoms of gastric indigestion may be present, usually vomiting or frequent regurgitation of small amounts of undigested milk, later mixed with mucus; eructations of gas with or without vomiting may occur, and distention of the stomach with gas and gastric colic may follow.

More often the symptoms of indigestion are intestinal. Occasionally there is constipation, but as a rule the stools are frequent, thin and green, containing flaky masses of undigested milk, and, after a short time, mucus which is frequently in large amount. The odour of the discharges may be slightly sour or there may be none at all. At times there is much gas and the stools are sour and irritating. If constipation is present there is apt to be severe colic and abdominal distention. The almost uniform absence of any elevation of temperature in these cases points strongly against the existence of an intestinal infection, which is further indicated by the prompt recovery under appropriate treatment. The condition seems to be one of indigestion with a secondary catarrh, which may affect either the stomach or the intestines, or both. In the cases in which the gastric symptoms predominate, the trouble seems primarily due to the fats. When the intestinal symptoms are most marked, it appears to be the proteids which are primarily at fault, but soon fats and sugars also disagree.

Before considering the case one of inadequate nursing, or simple indigestion in a nursing infant, one should be careful to exclude organic conditions in the child, particularly hypertrophic stenosis of the pylorus. The diagnosis of unsuccessful nursing should include the changes in the milk and if possible the causes of these changes.

As the first step one should endeavour to gain some idea as to the quantity of milk secreted. During the first week, particularly from the second to the fourth day, the temperature may be elevated quite apart from septic or inflammatory conditions or even evidences of indigestion. This is particularly seen where the breasts secrete almost nothing (see Inanition Fever). Often when the milk is very scanty something may be learned from the manner in which the child takes the breast. Where the milk is abundant, five or six minutes are often sufficient. If the milk is very scanty, an infant will frequently nurse half or three-quarters of an hour and then stop, more because it is exhausted than because it is satisfied. Sometimes, when the breasts are practically empty, the child will seize the nipple and nurse vigorously for a few moments, then drop it in apparent disgust and refuse to make any further efforts. The only satisfactory way of determining the quantity of milk secreted is to weigh the infant before and after each nursing. If the milk is merely scanty, but not otherwise abnormal, the infant does not gain, but shows no symptoms of indigestion, such as vomiting, colic, or undigested stools, and he frets and cries from hunger only.

An excessively rich milk is usually found under the following conditions: The mother is in good health, has large breasts which are full and tense at nursing time. In most cases she is upon a very abundant diet, getting little or no exercise, and frequently taking some alcoholic beverage with the notion that because the child is not thriving the milk is poor. The child may be colicky, sleepless, and uncomfortable, may vomit, may have frequent stools containing much undigested food, and may be losing in weight. A similar condition is often seen when a wet-nurse makes a change from the simple life and habits of her own home to the more luxurious life and diet of the family to which she goes. The milk then has usually a high specific gravity, is high in fat and high in proteids. The following analyses from Rotch illustrate the point: No. I shows milk of a healthy but under-fed wet-nurse two days before change of food; II, the milk of the same nurse after one month of rich food with very little exercise; III, milk of the same nurse, the food and exercise being regulated. The effect of the exercise and the change in diet is seen in a very marked reduction in the proteids.

	I.	II.	III.
	Per cent.	Per cent.	Per cent.
Fat	0·72	5·44	5·50
Sugar	6·75	6·25	6·60
Proteids	2·53	4·61	2·90
Salts	0·22	0·20	0·14

A scanty milk of a poor quality is most often seen where the mother is delicate or anæmic, or perhaps has had a difficult or complicated labour, and who besides is anxious and careworn. It is often with the greatest difficulty that one can secure the necessary half ounce required for examination. The milk is usually low in total solids and very low in fat. The specific gravity may be only 1·024 to 1·027, and the fat only one per cent or less.

A disturbed or disordered milk secretion is sometimes seen when the milk is scanty, often when it is very abundant. Like the group of cases just mentioned this is frequently met with when the mother's general health is below the normal, but particularly is it influenced by her nervous condition. It is the highly nervous, emotional, worried woman whose milk we are now considering. During the first week or two the secretion may be excessive and then rapidly diminish; or, though the milk continues abundant, the infant shows no improvement. It is most frequently found on examination that the milk is low in fat (0·50 to 1 per cent), while it is high in proteids (1·75 to 3·50 per cent). The child's symptoms are usually those of intestinal indigestion—severe colic, flatulence, and frequent, green, undigested stools.

Management.—The cause of the symptoms being in the food and not in the child, the futility of all medicinal treatment will be at once apparent. He who expects to relieve the symptoms of indigestion by the use of digestive ferments, by giving something before the nursing to dilute the milk, or to check frequent intestinal discharges by opium or astringents, will be disappointed. Temporary benefit often follows a dose of castor oil, but unless the milk can be materially changed in composition no permanent improvement in the child is to be looked for. The question usually to be decided relates to the continuance of nursing. We have a choice of four courses: (1) To continue nursing, endeavouring to correct the milk through treatment of the mother; (2) to partly nurse and partly feed from the bottle; (3) to stop all nursing temporarily, pumping the breasts meanwhile to keep up the secretion while we attempt to improve its character; (4) to wean at once and entirely. In deciding which of these courses is to be adopted we must take into consideration the condition of the child, the severity and duration of its symptoms, the findings of the milk examination, and the condition of the mother.

While the analysis of the milk is of some value in determining the course to be pursued, and should, if possible, be made, it is of much less importance than the child's symptoms. We must be guided not by what the milk contains, but by how seriously it disagrees. The chemical examination may show the milk to be of normal average in the proportion of its different ingredients and yet the child be seriously upset by it; on the other hand, a child may be doing admirably upon a milk which shows proportions which differ very greatly from the normal average.

The question always concerns the effect of the particular milk upon the particular child.

When the symptoms of indigestion are severe or have been prolonged it is usually a mistake to attempt to relieve the condition by simply substituting some other food for part of the nursings. This seldom leads to any material improvement in the symptoms, while it does confuse the result, since we can not now tell whether it is the breast or the bottle feeding which disagrees. A better plan is to stop nursing entirely for a time and try the bottle alone. If the symptoms are at once relieved the weaning should be permanent.

When symptoms point to a scanty milk, but of fair quality—i. e., infant not gaining but without any particular symptoms of indigestion—one is often able to overcome the difficulties and continue the nursing to advantage. Until a decided increase in the milk has occurred the child should have supplementary feedings from the bottle in sufficient number to insure its being properly nourished. Only one or two a day may be required, or it may be desirable to nurse and give the bottle alternately. If the latter plan is followed, both breasts should be given at each nursing period for the stimulating effect upon the secretion.

In the treatment of the mother the first thing is to secure for her an undisturbed rest at night. If possible, she should be entirely relieved of the care of the infant at this time, and if feeding is necessary the bottle should be given. She should have a certain amount of fresh air every day, driving if possible, or walking as soon as she is able to take more active exercise. Gentle massage of the breasts is often useful in stimulating secretion. It should be done with care and with every precaution against infection, and may be repeated two or three times a day for ten minutes. The diet should be abundant, with a large allowance of milk and meat, especially beef. If there is anæmia, iron should be given. Some of the alcoholic extracts of malt are useful (page 138). Every means should be taken to improve the general nutrition, for whatever benefits this improves the milk. If the conditions present are incident to the confinement or the convalescence, the prognosis is good; and in the course of a week or two very marked improvement may be evident, and lactation may be successfully continued. If, however, the conditions depend upon constitutional debility, the prognosis is much worse. Temporary improvement may take place, but it soon becomes evident that the nursing is a failure.

When the symptoms are found to be associated with an over-rich milk the prospects for continuing nursing are much better than when the milk is poor. Unless the infant's digestion is very feeble or has been seriously upset either with vomiting or diarrhœa, one can usually so alter the milk by treating the mother as to make it possible to keep the baby at the breast. Alcohol should be prohibited; the diet, especially the amount of meat, should be reduced, and the mother required to take daily exercise in the open air, particularly by walking. The intervals between nursing should be lengthened, usually to three hours. In many cases there is an advantage in diluting the milk by allowing the child to take water before putting it to the breast. The improvement following such a change in regimen is often immediate, and with increasing age and weight the child gradually becomes accustomed to and is able to digest the rich milk. If, however, the child's symptoms of indigestion are of an aggravated type, whether gastric or intestinal, it will be necessary, even though the weight is increasing normally, to stop nursing entirely for a time. The breasts should be pumped at regular intervals and the child placed upon some other food until the symptoms are relieved, and then brought back gradually to breast feeding. Should the infant's digestion be upset a second time as soon as the breast is resumed, the child should be weaned.

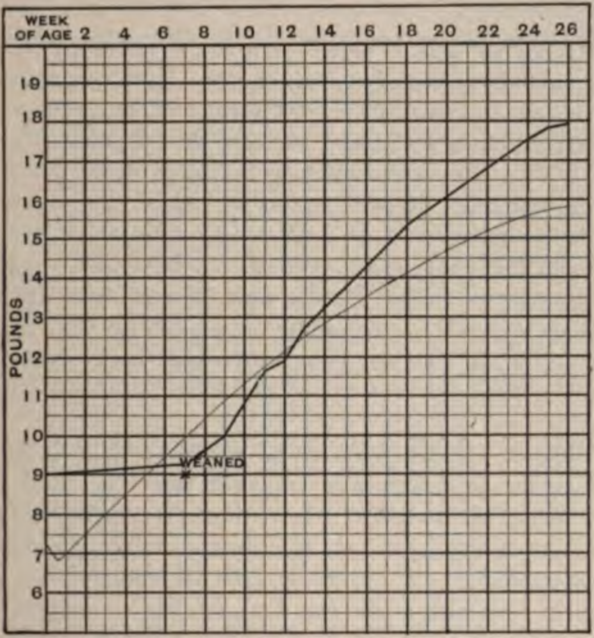

FIG. 32.—Weight curve showing the effect of bad nursing and good feeding. Maternal nursing for seven weeks; continued symptoms of indigestion; colic, frequent green passages, constant discomfort, etc.; other treatment without avail. Immediate improvement when weaned and put on modified milk from the laboratory. Formula: Fat 1·5 per cent, sugar 6 per cent, proteids 0·75 per cent. All symptoms of indigestion rapidly disappeared, the percentages were gradually increased, and steady gain in weight followed.

If the examination shows the milk to be of very poor quality (i. e., low in fat, low in total solids), whether scanty or abundant, the outlook is not good. It is seldom that the conditions affecting the mother to which such a milk is due can be removed.

When we see a fretful, colicky, sleepless infant with either no gain in weight or a loss of a few ounces a week, and with stools which never approach the normal, and these conditions have lasted for three or four weeks, we are justified in taking the child from the breast at once (Fig. 32). When the symptoms are less pronounced, and especially when, in spite of all discomfort and indigestion, the infant is gaining in weight, even though not rapidly, further efforts may be made before weaning is ordered.

Summary.—Poor milk is usually low in fat and scanty in quantity, while the proteids may be either high or low. Very rich milk is usually high both in fats and proteids. Very poor milk can seldom be permanently improved unless the causes are very definite and of a temporary character. Over-rich milk can often be improved if the true explanation for it can be reached. Results are to be judged not so much by the change in the composition of the milk as by improvement in the infant's symptoms. On the whole, since artificial feeding, when it can be properly done, gives much better results than poor or doubtful nursing, I am inclined, as a result of increasing experience, to stop nursing after a fair trial—e. g., of two to three weeks—has been made, and begin feeding, rather than waste time in prolonged efforts to improve the breast-milk.

WET-NURSING.—In the selection of a wet-nurse, it is by no means so essential as has generally been supposed, that her child shall be of about the same age as the child she is to nurse, for, after the first month, the changes in the composition of breast-milk are insignificant. It is always desirable that the wet-nurse shall have nursed her own infant long enough to demonstrate the fact that she has an abundance of good milk; hence, taking a wet-nurse at the end of the first or second week is always fraught with considerable uncertainty. It is the quality of the milk, not its age, which determines whether or not it will agree. For an infant over one month old, a good wet-nurse whose milk is anywhere between one and six months old will usually answer perfectly well; and even for premature infants such a milk may be used without hesitation, but it should at first be diluted.

A good nurse must, first of all, be a healthy woman, free from syphilitic or tuberculous taint, and her throat, teeth, skin, glands, scalp, and legs should be carefully inspected. She must have good mammary glandular development. The breasts should be full and hard three hours after nursing. They may be very large and yet supply very little milk, being then composed almost entirely of fat. On the other hand, some smaller breasts may be almost all glandular tissue. The difference in the size of a breast before and after nursing, is one of the best guides as to the amount of milk it is secreting. The nipples should be free from erosions or fissures, and long enough for the needs of the child. Preferably she should be of a phlegmatic temperament, and of a good moral character. This is desirable for personal reasons, although there is no evidence of moral qualities being transmitted through the milk. It is desirable that a nurse should be between twenty and thirty years of age, although much more depends upon the individual than upon the age. Other things being equal, a primipara should be chosen. An examination of the milk may be of some assistance in selecting a nurse; but the best evidence to be obtained of the character of a woman's milk is the condition of her own child, which should always be seen before she is accepted. It often happens that a woman who has had an abundant supply of milk for her own infant, has very little for another infant for the first few days in her new surroundings. This is usually the result of the nervous disturbance connected with parting from her own child, going to a new place, being carefully watched, etc. In such a case it should not be too readily decided that she is incompetent as a nurse, for, under most circumstances, with proper treatment her normal flow of milk will be re-established.

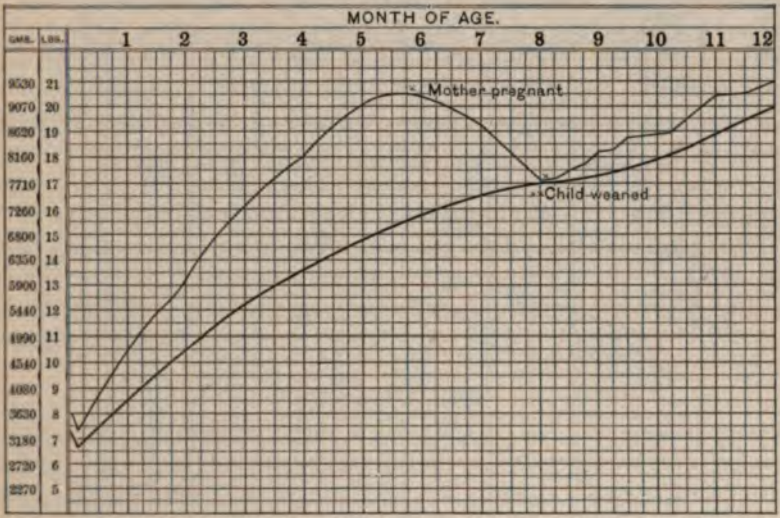

FIG. 33.—Chart showing the effect of pregnancy upon the weight of a nursing infant. The upper line is that of the patient; the lower one is the average line for the first year.

WEANING.—Weaning should always be done gradually, when possible, for the sake of both mother and child. Sudden weaning is apt to be followed by an attack of acute indigestion in the infant. This, however, is not a necessary result, and usually depends upon the fact that the child is given cow's milk without sufficient dilution. Weaning in hot weather is usually to be avoided, but the harm from this is not nearly so great as sometimes results where lactation is unduly prolonged because of a prejudice against a change of food at this time. While there are many women of the lower classes who are able to nurse their children to advantage for the entire first year, the number of such among the better classes is certainly very small. By the latter, nursing can rarely be continued beyond the ninth, and often not beyond the sixth month, without unduly draining the vitality of the mother and at the same time harming the child. The late months of lactation, like the early months, require close watching. It is a common mistake to continue both maternal and wet-nursing too long, owing to a dislike of making a change when things are going tolerably. It is a safe rule to make the ninth month the time to supplement the breast-feeding by other food. But here, as in the early months, the child's weight is the best guide. In the absence of evident signs of disease, a stationary weight for several weeks makes weaning advisable; a steady loss makes it imperative.

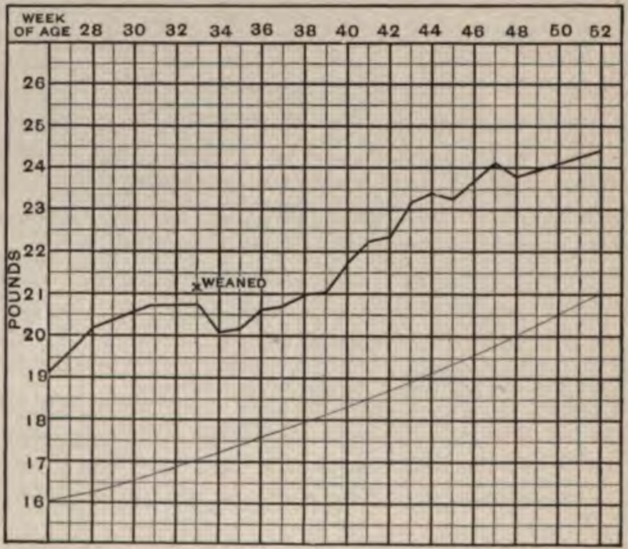

FIG. 34.—Weight curve of a child properly weaned. Abrupt weaning at eight months; loss of weight for the first week due to the child's being put upon cow's milk with low percentages. Formula: Fat 1·6 per cent, sugar 6 per cent, proteids 0·80 per cent. Percentages were rapidly increased, with subsequent steady and regular gain in weight. Weaning accomplished without the slightest symptom of indigestion. The lower is the average line.

The accompanying weight-chart (Fig. 33) illustrates this point. The infant did unusually well until the sixth month. As it did not seem ill, the parents were not disturbed until the loss had reached three pounds. Feeding was at once begun, and the child gradually regained its lost weight. It was subsequently discovered that the mother was pregnant.

When a nursing infant has been accustomed from birth to take one feeding a day from the bottle, always a great convenience to a nursing mother, gradual weaning is generally an easy matter; otherwise it is sometimes an impossibility, the child refusing all food except the breast so long as this is given, and nothing but starvation inducing it to take food either from a bottle or a spoon.

Sudden weaning may be required at any time from the development in the mother of acute disease of a serious nature, such as typhoid fever or pneumonia, of grave chronic disease, such as tuberculosis or nephritis, from the intercurrence of pregnancy, or from disease of the mammary gland. An infant should not be suckled at a breast which is the seat of acute inflammation. Through many of the minor ills—mild attacks of bronchitis, pharyngitis, indigestion, and even malarial fever—mothers frequently nurse their children without any seeming detriment to them or to themselves. In acute illness of short duration, if severe, it is usually better, unless we decide to wean altogether, to feed the child from the bottle and to maintain the flow of milk by the use of the breast-pump three or four times a day rather than allow it to dry up.

In cases of sudden weaning, the food should in the beginning be very much weaker than for an artificially fed child of the same age. The change can then be made without causing much disturbance (Fig. 34). When the infant has become somewhat accustomed to cow's milk the strength of the food may be gradually increased.

MIXED FEEDING.

By mixed feeding is meant a combination of nursing and artificial feeding. There are no objections to this practice; often there are great advantages in giving an infant only a few breast feedings a day when more are impossible. This may frequently be done in hospital practice, and thus a single wet-nurse may assist in the feeding of two or three infants. Mixed feeding may be resorted to whenever the milk supply of the mother is insufficient. If at any time the mother's health begins to suffer, she may be relieved of night nursing or of one or more nursings during the day, and the bottle substituted. In this way she may be enabled to continue lactation for some time longer than would otherwise be possible. Mixed feeding is often necessary during the first few weeks, while the mother's milk is insufficient in consequence of something which has retarded her convalescence. The milk may become abundant and of good quality as soon as the mother is well enough to be up and out of doors, although it was previously scanty and of inferior quality. Two or three feedings a day from the bottle help to bridge over this period and prevent the child's nutrition from suffering. But before allowing a mother partly to nurse and partly to feed her infant, one should be sure that the quality of the milk is good.

It is well from the very outset to accustom the infant to take one of its feedings, or at least to take water, from a bottle each day. In maternal nursing, the occasional feeding which is usually necessary becomes then a simple matter. If the child is being wet-nursed, the same plan is advisable, for it then becomes easy to put an infant upon the bottle entirely in the event of the wet-nurse leaving suddenly—a not uncommon occurrence.

ARTIFICIAL FEEDING.

There are several fundamental principles regarding which nearly the whole scientific world is agreed.

1. Woman's milk is not only the best, it is the ideal infant-food.

2. Any substitute should furnish the same constituents—fat, carbohydrates, proteids, salts, and water; furthermore, they should be in about the same proportion as they exist in a good sample of woman's milk.

3. The food should have a caloric value * sufficient to promote growth and furnish energy.

4. The different constituents should resemble those of woman's milk as nearly as possible both in their chemical composition and in their behaviour toward the digestive fluids.

5. These conditions are fulfilled only by fresh milk from some other animal.

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* From numerous observations, the caloric needs of the average infant in health have been shown to be about 100 calories for each kilo. of body weight from the third week to the sixth month. These gradually diminish until at the end of the first year they reach about 75 to 80 calories per kilo. The caloric requirements are greater for very active infants on account of their more rapid metabolism; also, for premature or wasted infants on account of their relatively larger body surface to radiate heat.

An infant weighing 7 kilos. (15 pounds) requires about 700 calories daily. As the caloric value of a good average specimen of woman's milk is about 650 calories per litre, the requirements would be supplied by a little over one litre of woman's milk.

The practical application of these facts in infant-feeding is that one should be careful to furnish to an infant who is artificially fed what is needed, but no excess. A food much below the normal caloric requirements, or one much above them, may be equally improper and therefore unsuccessful. The physician should be able to calculate the caloric value of the food given when infants are not thriving, to see if possible where the mistake lies.

The caloric value of any modification of cow's milk of known percentages may be calculated as follows: An infant six months old, weighing 15 pounds (7 kilos.), is taking six feedings of 6 ounces, or 36 ounces daily, of a milk containing, fat 3·5 per cent, sugar 7 per cent, proteids 1·75 per cent.

·035 (fat %) × 9·3 (caloric val. of fat) = ·325 caloric val. of fat in 1 grm. food.
·07 (sugar %) × 4·1 " " " sugar) = ·287 " " " sugar " 1 " "
·0175 (proteids %) × 4·1 " " " proteids) = ·072 " " " proteids " 1 " "
·684 caloric value of 1 gram of food.

·684 × 1000 = 684 (caloric value 1 litre food).
36 ounces = 1·06 litres; 1·06 × 684 = 725 (No. calories in food taken daily).
725 ÷ 7 (body wt. in kilos.) = 104 (No. calories per kilo.); which is slightly above the normal requirements.

In the artificial feeding of infants, cow's milk is selected as being the only milk available for general use. Although it furnishes all the constituents required, they are not present in the proportions suited to young infants, and the constituents are not identical with those in woman's milk. Cow's milk, therefore, can not be fed to most infants without some changes. These changes are technically known as the *Modification of Cow's Milk.*

Although there is practical agreement among writers and teachers regarding the foregoing points, there still exists considerable difference of opinion respecting methods of adapting cow's milk to the infant's digestion. To make these changes properly it is necessary to know in the first place what are the exact differences between cow's milk and woman's milk; and, secondly, to devise the simplest method of overcoming them.

The earliest milk modification was simply dilution with water and the addition of enough cane sugar to make it taste like breast milk. The only change made with the age of the child was simply to vary the amount of water. Instead of water as a diluent many have preferred to use gruels made from different cereals—oatmeal, barley, arrowroot, etc. —believing that thereby the casein was rendered more digestible. Upon such simple modifications as these many children have done, and many still do, very well, when the matter of dilution is judiciously managed. But it is equally true that very many do not do well, and that present knowledge enables us to do something better. There are, however, circumstances where anything more complex is impossible in the way of milk modification; then only should the old methods of simple dilution be employed.

Later, when the composition of woman's milk came to be better understood, it was thought that all that was necessary in modified milk was to secure the exact percentages of fat, proteids, sugar, and salts which exist in a good sample of woman's milk, and that this combination would be the best possible substitute for it. Out of this came the various mixtures of milk, cream, sugar, etc., which aimed to reproduce, according to the views of different writers, the exact proportions of woman's milk.

This was a great step in advance, in that some proper relation between the different food constituents was maintained. While frequently successful, such formulas often failed for lack of flexibility. The food was the same, but the child was not always the same. Furthermore, the difference in the digestibility of both the fats and the proteids was not sufficiently taken into account. Experience has shown that no single milk-formula can be made to serve as a substitute for woman's milk; and intelligent students of the problem have ceased to search for one.

The central thought of the newer method of modification—which may very properly be called the "American method"—is to consider the different elements of the food separately and to adapt their proportions to the child's digestion. Like the method just described, it is based upon the percentage composition of woman's milk, and also recognises that there is a difference in the digestibility of cow's milk and woman's milk, particularly of the proteids. It aims to discover the proper proportions of fat, sugar, and proteids, and the best methods of gradational increase for healthy infants with normal digestion; and also to discover for those with abnormal or feeble digestion, the combinations best suited to the individual conditions. Where difficulty exists in the digestion of milk, it is usually with some one of its elements, or at least chiefly with one. In such a condition, instead of stopping milk entirely, or reducing the proportion of all the elements by simply diluting the food still further, that one alone which is causing the disturbance is reduced.

In practice there is necessary an easy method of securing the usual percentages which experience has shown to be best for healthy infants, following in a general way those existing in woman's milk—a method, moreover, which can readily be adapted to special and peculiar conditions. In brief, the American, or, as it is sometimes called, the "percentage method" of milk modification for infant-feeding, aims at something which is definite, exact, and at the same time flexible. It is somewhat more complex possibly than the older methods, but not nearly so difficult as may at first appear. In practical results, however, it is in my judgment, and in the opinion of nearly every one who has taken the trouble to master it, a very great step in advance. By this method infant-feeding has been placed for the first time upon a scientific basis. Percentages are simply a method of stating definitely just what we are giving, and furnish the only means by which our observations can be recorded and compared with those of others.

For the fundamental work along this line the world is indebted to Prof. T. M. Rotch, of Harvard, and Mr. G. E. Gordon, of the Walker-Gordon Laboratory Company.

THE MODIFICATION OF COW'S MILK FOR HEALTHY INFANTS DURING THE FIRST YEAR.—By the modification of cow's milk is meant its adaptation to the purposes of infant-feeding. It is desirable to consider separately the changes required by healthy infants with normal digestion, and those required by infants with feeble digestion, or those suffering from more or less indigestion. From a failure to make this distinction, much confusion has arisen and many errors have crept into the subject of infant-feeding. The digestion of all healthy infants is very much alike, and they can all be fed in much the same way; while, on the contrary, the variations afforded by unhealthy infants are almost endless, and each case must be considered by itself. If it is only healthy infants that can be fed by rule, it is equally true that if fed from the beginning by proper rules most infants will remain healthy.

In adapting cow's milk for infant-feeding we must realize at the outset that, no matter how we may alter it, cow's milk is not a perfect substitute for woman's milk. It should not be lost sight of that there are inherent differences which will never be altogether removed. The following table gives the proportions of the various elements which make up the two milks:

	Woman's milk, average.	Cow's milk, average.
	Per cent.	Per cent.
Fat	4·00	4·00
Sugar	7·00	4·50
Proteids	1·50	3·50
Salts	0·20	0·75
Water	87·30	87·25
	100·00	100·00

These quantitative differences in the constituents are important. It will be seen that cow's milk has an excess of proteids and salts but is deficient in sugar. Far more important, however, for the infant are the qualitative differences. The sugar in the two milks, it is true, is nearly if not quite the same. The fat of cow's milk, however, contains a much larger proportion of volatile fatty acids. The salts are excessive in amount, particularly calcium phosphate, but are deficient in iron and potassium. The most important difference is in the proteids. The total proteids of cow's milk are nearly two and a half times as great as in those of woman's milk. In cow's milk the soluble proteids (lactalbumin, etc.) are only about one-third or one-fourth as abundant as the insoluble proteids (casein); while in woman's milk the soluble proteids form more than half the total. Furthermore, the difference in the digestibility of the proteids, particularly the casein, is even greater than this difference in quantity. Other important conditions relate to the reaction of milk, its freshness, bacterial contamination, etc. The modification of milk must aim, therefore, at something more than overcoming the quantitative differences in the constituents.

Fat.—The average amount of fat of cow's milk which a healthy infant can digest varies from 2 to 4 per cent. With many infants it is often necessary to begin with a slightly lower amount than 2 per cent. The increase is made very gradually, the upper limit being reached usually at four or five months. I have seldom found it advantageous to increase the fat above 4 per cent, and constantly see serious derangements of digestion produced by the use of higher percentages.* The danger of disturbing the infant's digestion by using too high fat is not sufficiently appreciated. This mistake is frequently made when rich Jersey milk is employed, and also when the fat percentage is steadily raised for the purpose of overcoming chronic constipation. There are many healthy infants who can not digest even 4 per cent of fat at any time, and many more who during hot weather do much better when a reduction to 3 or 3·5 per cent is made. No modification of the fat of cow's milk is possible except in the amount. There seems to be no difference in the digestibility of gravity and centrifugal cream. Freshness is a very important consideration in all extra fat added to milk; since undoubtedly the fermentative changes, some of which may take place in the fat quite early, seriously affect its digestibility.

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* Archives of Pædiatrics, January, 1905.

Sugar.—In woman's milk the percentage of sugar varies but little; it is usually between 5·5 and 7 per cent. In feeding cow's milk it is seldom required to have the sugar less than 5 or more than 7 per cent. To obtain the proper proportion of sugar is the simplest part of the modification. It is only necessary to calculate the amount to be added to bring this up to the 5, 6, or 7 per cent desired. The milk sugar should first be dissolved in boiling water, and, when it contains impurities, filtered through absorbent cotton. The advantages of lactose over other sugars have already been considered (page 127). When, however, good milk sugar can not be obtained, cane sugar may be substituted; the amount added should be but little more than half that of milk sugar on account of its sweeter taste and greater liability to undergo fermentation. It should be distinctly understood that the purpose of adding sugar is not to sweeten the food, but to furnish the proper proportion of soluble carbohydrates necessary for the infant's nutrition.

Proteids.—The modification of the proteids is the most important change necessary in cow's milk, for it is the proteids which give most of the trouble to the infant's digestion. The density of the coagulum which forms in the stomach from cow's milk is greatly lessened by diluting the milk, but the coagulum differs much from that formed from woman's milk even when the total proteids are made the same.

Several different methods have been proposed for modifying the proteids of cow's milk: (1) Reducing the total proteids by dilution; (2) partially predigesting them by peptonizing; (3) separating the proteids by removing the casein after precipitation with rennet; (4) adding lime water or other alkalies; (5) adding sodium citrate; (6) using as a diluent, instead of water, gruels made of different cereals—oatmeal, barley, arrowroot, etc., for their mechanical effect upon the coagulation of the casein.

These different methods are more fully discussed in the later pages devoted to Difficult Cases of Feeding. For healthy infants with average digestion, reduction in the quantity of the proteids is often all that is necessary. During the early months it is not enough to reduce the proteids to the average amount present in woman's milk—i. e., 1·5 per cent. Better results are usually obtained by making the proteids for the first few days only 0·5 per cent; then, as the stomach becomes somewhat accustomed to cow's milk, gradually raising the proportion until after a few weeks the child is usually taking 1 per cent; by the end of the second or third month, 1·5 per cent; and by the end of the fourth or fifth month, 2 per cent proteids. It is seldom that the total quantity of proteids present in cow's milk can be given before a child is a year old. I believe the secret of success in feeding cow's milk is to begin with the proteids so low as not to disturb the infant's digestion, and then slowly but steadily to raise the quantity. While the infant's stomach was not intended to digest cow's milk, but woman's milk, it is perfectly certain that by this method it can gradually be trained to digest cow's milk of the percentages mentioned.

Except to start with too high proteids no more common mistake is made than to continue long with too low proteids. Anæmia, malnutrition, and, I believe, not infrequently scurvy are seen as a consequence of this practice. The gradual increase is therefore just as important as the low beginning.

Inorganic Salts.—These may generally be calculated as one-fifth the total proteids. No separate modification of the salts is usually attempted. When the proper dilution is made for the proteids, the proportion of the total salts will be nearly correct. But it should not be forgotten that this dilution, while it brings down those salts which are in excess to a proper proportion, reduces to the same degree those which were originally deficient. The influence of this upon nutrition is something deserving further study.

The amount of reduction obtained by the different dilutions is shown in the following table:

	Cow's milk.	Diluted once.	Diluted twice.	Diluted 3 times.	Diluted 4 times.	Diluted 6 times.	Diluted 9 times.
Proteids...........	3·50	1·75	1·16	0·87	0·70	0·50	0·35
Inorganic salts......	0·75	0·37	0·25	0·18	0·15	0·10	0·07

Reaction.—It has been customary to overcome the acidity of cow's milk by adding either lime-water or bicarbonate of soda. Of the former, there is required about one ounce to each twenty ounces of the food; of the latter, about one grain to each ounce of the food. The value of these additions to milk is probably due more to the retardation of coagulum formation in the stomach than to the neutralization of any increased acidity of the milk taken.

Bacteria.—These are always present in cow's milk. They have been already considered in the pages devoted to the Sterilization of Milk.

The Observation of Cases of Infant-Feeding.—For the first few weeks it is essential that the physician see the infant every few days, inspect the stools, hear the nurse's report, and see how his directions are being carried out. When the child is well started and has begun to gain regularly in weight, a weekly visit will be sufficient. Still later a regular weekly report in writing, to be continued up to the seventh or eighth month, may be all that is required; after that time monthly reports are usually sufficient. My plan is to have the weekly report include only answers to certain questions—viz.:

1. Weight:..........gain or loss since last report.
2. Stools: frequency and general character.
3. Vomiting or regurgitation—when? and how much?
4. Flatulence or colic?
5. Appetite: is the child satisfied? Does he leave any of his food?
6. Is he comfortable and good-natured?
7. How much does he sleep?
8. Date.
9. Date of last report.

An excellent plan is to furnish the patient with printed forms containing these questions to be filled out and returned. This is a simple matter, and there are very few intelligent mothers who will be unwilling to cooperate with the physician to this extent. With information regarding the points indicated, it is possible for the physician to know pretty accurately how the case is doing, what changes, if any, are desirable in the food, and whether he ought to see the patient. It is only by some systematic method of observation that one can secure the best results with any form of infant-feeding.

Milk Laboratories.—The first milk laboratory was established in Boston by the Walker-Gordon Company in 1892; one in New York in 1893, and since that time others in many American cities. They undertake to furnish "modified milk" of any desired proportions, upon the prescription of physicians. The elements chiefly used by the Walker-Gordon laboratories are: (1) Cream containing 32 per cent fat; (2) separated milk, from which the fat has been removed by the centrifugal machine; (3) a standard solution of milk sugar, 20 per cent strength. These contain fat, sugar, and proteids in the following proportions:

	Cream.	Separated milk.	Sugar solution.
	Per cent	Per cent.	Per cent.
Fat..........	32·00	0·05	.....
Sugar..................................	3·40	5·00	20.00
Proteids................................	2·50	3·55	.....

By combining these it is possible to vary the percentages of fat, sugar, and proteids in the milk to almost any degree desired, and to do this with very great accuracy. By using whey, a separate modification of the proteids is accomplished; so that within certain limits a larger proportion of whey proteids, chiefly lactalbumin, can be given. The highest proportion of whey proteids with the lowest proportion of casein can be given when the total proteids do not exceed 1·15 per cent; of this, 0·90 per cent may be whey proteids and 0·25 per cent casein. The alkalinity is usually obtained by adding lime-water in any desired amount. The laboratory uses either gravity or centrifugal cream, as preferred by physicians; it also adds, when requested, gruels of wheat, oats, or barley of any desired strength; and, finally, it delivers the milk raw, or heats it for sterilization to any temperature ordered by the physician.

The food-supply for the entire day is delivered each morning in the bottles from which it is to be fed. The empty bottles returned are washed and sterilized at the laboratory. In ordering the food the physician simply writes for the percentages of fat, sugar, and proteids which he desires, together with the number of feedings for twenty-four hours and the quantity for each feeding, in the following form:

℞ Fat	3 per cent.
Sugar	6 "
Proteids	1 "
Alkalinity, lime-water	5 "
Number of feedings	8
Amount for each feeding	4 ounces.

Heat to 155° F., 30 minutes.

The milk laboratory and the percentage method of milk modification mark a great advance in infant-feeding. The laboratory bears the same relation to the physician as does the apothecary shop. It does not attempt to prescribe; it does not prepare a food. It aims only to supply the physician with any milk modification which he may desire to use. The results with milk from the laboratory will depend, therefore, upon the physician's knowledge and experience in prescribing milk. One who is ignorant of the principles of infant-feeding is not helped by the laboratory, any more than is the careless diagnostician or the uneducated practitioner by a good apothecary. The responsibility of the laboratory is only to see that the patient gets exactly what has been ordered. Too often the physician has wrongly laid the blame for his failures in feeding at the door of the laboratory, when the cause was really his own want of experience in ordering milk.

In using the laboratory, one is not restricted to any method or plan of feeding, but is free to carry out his own ideas with a much greater assurance of accuracy than is possible when the milk is prepared in the average home. He is independent of the ignorance, carelessness, or caprice of the nurse who otherwise would probably prepare the food. While there are many physicians who find little difficulty in calculating percentages from the materials in ordinary use for the home modification of milk, it must be admitted that this calculation is a stumbling-block to the majority. The laboratory makes it an easy matter to vary the percentages at will without making arithmetical calculations. But by whatever method the child is fed the physician who assumes the responsibility to direct must be familiar with the subject and he must keep in touch with the case if he expects good results.

The practical advantages of laboratory-feeding are sufficiently attested by the fact that laboratories have been established in sixteen of the larger cities of the United States and Canada, and have received the indorsement of the great body of the most intelligent physicians of the country. The principal objection to laboratory-feeding is the expense.

After over twelve years' experience with laboratory-feeding I am more than ever convinced of its scientific value and its practical utility, and have, therefore, no hesitation in placing it, when intelligently used, next to maternal nursing. As a general guide to the modification of milk for an average healthy infant the following table is introduced, showing the manner in which the changes required by the development of the child are made:

Table Showing Percentages of Fat, Sugar and Proteids which May Be Ordered from the Milk Laboratory and are Suitable for the First Year.

		Fat.	Sugar.	Proteids.		Whey proteids.		Casein.
Weak Formulas.	I.	1·00	4·00	0·25	or	0·20	and	0·05
	II.	1·00	5·00	0·50	"	0·45	"	0·05
	III.	1·50	5·00	0·75	"	0·70	"	0·05
	IV.	1·50	6·00	1·00	"	0·85	"	0·15
Medium Formulas.	V.	2·00	6·00	1·00	"	0·85	"	0·15
	VI.	2·00	6·00	1·10	"	0·80	"	0·30
	VII.	2·50	6·00	1·20	"	0·80	"	0·40
	VIII.	2·50	6·00	1·30	"	0·80	"	0·50
	IX.	3·00	6·00	1·40	"	0·80	"	0·60
	X.	3·00	6·00	1·40	"	0·60	"	0·80
	XI.	3·00	6·00	1·50	"	0·50	"	1·00
Strong Formulas.	XII.	3·50	7·00	1·60				
	XIII.	3·50	7·00	1·75				
	XIV.	3·50	7·00	2·00				
	XV.	3·50	7·00	2·25				
	XVI.	3·50	7·00	2·50				
	XVII.	4·00	7·00	2·50				
	XVIII.	4·00	6·00	3·00				
	XIX.	4·00	4·50	3·50		(Whole milk.)		

The first group, classed as weak formulas, are designed for normal infants during the first few weeks, or for those with feeble digestion, of whatever age.

The second group are designed for the needs of normal infants from about one month to four or five months, although there are many who can not take a stronger food for a much longer time.

The third group is expected to cover, for children with good digestion, the period from about the fifth month to the twelfth or thirteenth month, gradually leading up to whole milk.

It is important to begin with a weak formula for a young infant, and for one with feeble digestion, whatever its age. One may then gradually increase the strength of the milk according to the indications afforded by the child's appetite and powers of digestion. With some the increase can be made more rapidly than with others, but with all children it is important that the steps of increase should be gradual and not greater than are indicated in the formulas of the table; it may even be desirable at times to make them more slowly than is there suggested.

In the table the total proteids to be used are indicated and also the quantities of whey proteids and casein, when one desires to order these separately. The advantage of so dividing the proteids, when a child has special difficulty in digesting proteids, is very great. By this means one may carry the percentage of total proteids much higher than is otherwise possible. The ability to order the proteids separately and to vary them readily constitutes one of the great advantages of laboratory-feeding. Formulas containing the divided proteids are to be recommended for routine use with young infants or with those with feeble digestion.

Home Modification of Milk.—Inasmuch as milk laboratories are as yet inaccessible to the great body of the profession, the problem presented is how the advantages of the laboratory method may be utilized where milk is prepared at home. No plan of home modification yet proposed secures more than approximate accuracy in the percentages of fat, sugar, proteids, etc. Yet, if the directions given below are carefully carried out, a degree of accuracy sufficient for all practical purposes can be secured. The physician thus can not only know the percentages he is giving, but he can himself readily vary them within the range usually required, according to the indications presented. The thing desired is a method simple enough to be readily grasped by the average mother or nurse who is to carry out the physician's directions. The method here given is one which in principle I have followed for many years; and I have found little difficulty in making patients understand how to use it. Several other methods have been proposed, which have their merits; all require a little study to enable one to use them freely.

The requisites for success in the home modification of milk are:

Good raw materials—the freshest and cleanest milk obtainable.

Knowledge on the part of the physician of at least the approximate composition of the milk and cream used in the home.

Directions which are clear, explicit, and in writing, that they may not be misunderstood.

The cooperation of an intelligent mother or nurse, that they may be properly carried out.

How to Obtain the Formulas Required for General Use.—If one has at command three series or groups of formulas in which the fat has certain definite relations to the proteids, he will be equipped for the great majority of cases met with in practice. The three groups are as follows:

First Series, those in which the fat is three times the proteids.

Second Series, those in which the fat is twice the proteids.

Third Series, those in which the fat and proteids are nearly equal.

Once thoroughly familiar with these groups of formulas, variations from them to suit the needs of the particular case can readily be made. In general, the First and Second Series, in which the fat is considerably higher than the proteids, are adapted to the early months, because at this period the infant as a rule has more difficulty in digesting proteids than in digesting fat. In the later months a higher proportion of proteids can be taken with the same percentage of fat. There are, however, other conditions besides age which must be taken into account, such as the vigour of constitution, the weight, and most of all the peculiarities of the child's digestion. It is, therefore, impossible to say that at certain months certain proportions are desirable, and certain others at another period.

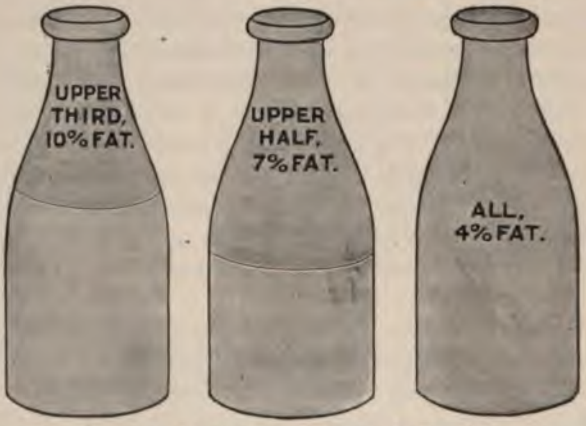

FIG. 35.—The percentage of fat in different layers of milk. (Compare page 152.)

Formulas in which the fat is three times the proteids.—This is nearly the relation which the fat and proteids bear to each other in a good sample of woman's milk. The easiest way to arrive at this would seem to be, first, to secure some milk or milk combination containing three times as much fat as proteids, and then dilute this according to the infant's age and digestion. After such dilution it will be necessary only to add the requisite amount of sugar and, when desired, lime-water to complete the modification. This, in brief, is the whole process.

The most convenient combination for dilution is one containing 10 per cent fat and 3·3 per cent proteids. I shall call it a 10-per-cent milk, and refer to it subsequently as the prímary formula of the First Series. The 10-per-cent milk may be obtained by removing the upper portion (see Fig. 35) from a quart bottle of milk, as described (pp. 151, 152). This method will answer for persons who can obtain milk fresh from the cow, or for those who use bottled milk, provided the bottling is done at the dairy before the cream rises. The upper milk may be taken off with a siphon, spoon, or small dipper (Fig. 36); pouring off is not so accurate. For those who do not get their milk as above described, the additional fat can be secured by adding cream to the milk. To secure a combination containing 10 per cent fat, equal parts of plain milk and the ordinary (16-per-cent) cream should be used.

The next step is the manner and degree of dilution of the primary formula. It is convenient in our calculation to make up 20 ounces of the food at a time. For such a 20-ounce mixture it is seldom necessary to use less than 2 ounces of our 10-per-cent milk. When one wishes to strengthen the food he gradually increases the amount of the 10-per-cent milk, 1 ounce at a time, making it successively 3 ounces, 4 ounces, 5 ounces, 6 ounces, etc., in a 20-ounce mixture, the water, of course, being reduced by the same amount.

These mixtures may readily be translated into percentages by remembering that *the percentage of fat is always exactly one half the number of ounces of the 10-per-cent milk used in a 20-ounce mixture.* Thus using 3 ounces will give 1·5 per cent fat; 4 ounces, 2 per cent fat; 6 ounces, 3 per cent fat, etc. The proteids will continue to be in every instance exactly one third the fat, as in the primary formula.

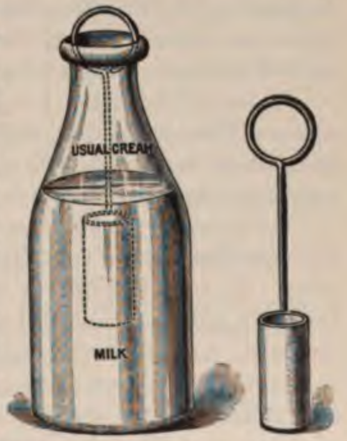

FIG. 36.—Chapin's dipper, for removing the upper layers of milk.

The amount of milk sugar needed to bring this up to the percentage usually required (5·5 to 6·5) is 1 ounce in each 20-ounce mixture. One may obtain from a druggist a box holding exactly 1 ounce of sugar, or may measure in a tablespoon, calculating 2½ even tablespoonfuls as 1 ounce. This sugar is dissolved in the water used for diluting the milk.

The usual proportion of lime-water added is 5 per cent, or 1 ounce in a 20-ounce mixture; this may be easily increased to any desired quantity. The foregoing directions may be expressed in the following table:

First Series of Formulas.—Fat to proteids, 3 : 1.

Primary Formula.—Ten-per-cent milk — or fat 10 per cent, sugar 4·3 per cent, proteids 3·3 per cent. Obtained (1) as upper portion of bottled milk (p. 152), or (2) equal parts milk and (16-per-cent) cream.

Derived Formulas, giving Quantities for Twenty-ounce Mixtures.

			Per cent.	Per cent.	Per cent.
1.	Milk sugar... 1 oz. Lime-water.. 1 oz. Water, q. s. to 20 oz.	with 2 oz. of 10% milk =	fat 1·00,	sugar 5·50,	proteids 0·33.
II.	" " "	" 3 oz. " " " =	" 1·50,	" 5·50,	" 0·50.
III.	" " "	" 4 oz. " " " =	" 2·00,	" 6·00,	" 0·66.
IV.	" " "	" 5 oz. " " " =	" 2·50,	" 6·00,	" 0·83.
V.	" " "	" 6 oz. " " " =	" 3·00,	" 6·00,	" 1·00.
VI.	" " "	" 7 oz. " " " =	" 3·50,	" 6·50,	" 1·16.

Making more than a 20-ounce mixture will be found very simple if we calculate for 25, 30, 35 ounces, etc. Thus for 25 ounces we add one-fourth more of each ingredient; for 30 ounces one-half more, etc. For 25 ounces of II, therefore, the exact formula would be: 10-per-cent milk, 3¾ ounces; milk sugar, 1¼ ounces; lime-water, 1¼ ounces; water q. s. to make 25 ounces—i. e., 20 ounces.*

Formulas in which the fat is twice the proteids.—Here we first obtain a combination, or primary formula, in which the fat and proteids stand in the relation of two to one, and then dilute this, adding milk sugar and lime-water to complete the modification.

The primary formula most conveniently obtained for this purpose is one containing 7 per cent fat and 3·5 per cent proteids, or a 7-per-cent milk. This we may get by removing the upper portion from a quart bottle of milk, as described on page 152. Or in case milk and cream are used, instead of this upper milk, it will be necessary to add one part ordinary (16-per-cent) cream to three parts milk. The dilution is accomplished in the same general way as with the First Series.

These formulas may readily be translated into percentages by remembering that the percentage of fat in any formula is exactly seven-twentieths, or about one-third, the number of ounces of the 7-per-cent milk in a 20-ounce mixture. Thus 3 ounces in the mixture will give about 1 per cent fat; 5 ounces will give 1·6 per cent; 9 ounces about 3 per cent, etc. In the following table these directions are expressed:

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* For method of calculating any number of ounces of any formula derived from 10-per-cent milk, see footnote, page 195.

Second Series of Formulas.—Fat to proteids, 2 : 1.

Primary Formula.—Seven-per-cent milk—or fat 7 per cent, sugar 4·40 per cent, proteids 3·50 per cent. Obtained (1) as upper portion of bottled milk (p. 152), or (2) by using three parts milk and one part (16-per-cent) cream.

Derived Formulas, giving Quantities for Twenty-ounce Mixtures.

			Per cent.	Per cent.	Per cent.
I.	Milk sugar... 1 oz. Lime-water.. 1 oz. Water, q. s. to 20 oz.	with 3 oz. of 7% milk =	fat 1·00,	sugar 5·50,	proteids 0·50.
II.	" " "	" 4 oz. " " " =	" 1·40,	" 5·75,	" 0·70.
III.	" " "	" 5 oz. " " " =	" 1·75,	" 6·00,	" 0·87.
IV.	" " "	" 6 oz. " " " =	" 2·10,	" 6·00,	" 1·05.
V.	" " "	" 7 oz. " " " =	" 2·50,	" 6·50,	" 1·25.
VI.	" " "	" 8 oz. " " " =	" 2·80,	" 6·50,	" 1·40.
VII.	" " "	" 9 oz. " " " =	" 3·15,	" 7·00,	" 1·55.
VIII.	" " "	" 10 oz. " " " =	" 3·50,	" 7·00,	" 1·75.
IX.	Milk sugar... ¾ oz. Lime-water.. 1 oz. Water, q. s. to 20 oz.	" 12 oz. " " " =	" 4·00,	" 7·00,	" 2·00.

With these, as with the First Series, if more than 20 ounces are required, we may make 25, 30, or 40 ounces by using of each ingredient one-quarter more, one-half more, or twice as much.*

Formulas in which the fat and proteids are nearly equal.—In general these formulas are more often used for healthy infants during the later months; but there are many conditions of disturbed digestion in which formulas having this relation of fat and proteids are desirable during the early months. This series of formulas is obtained by using as a starting-point plain milk and variously diluting it. The exact percentages of fat and proteids obtained with the different dilutions of milk, and the amount of sugar necessary to bring this up to the desired quantity, are shown in the table below. The sugar in the higher formulas is reduced for the reason that with them the child will probably be taking a considerable part of his carbohydrates in the form of starch.

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* One may readily calculate any formula of any number of ounces which may be desired in either the first or the second series in the following way:

There is wanted, for example, 35 ounces of a mixture containing 3 per cent fat, 6 per cent sugar, 1·50 per cent proteids. In this combination the fat is twice the proteids. It will therefore be derived from 7-per-cent milk.

35 (No. ounces needed) × 3 (percentage fat desired) = 105 (parts of fat required).
105 ÷ 7 (parts of fat in milk used) = 15 (No. ounces of 7-per-cent milk needed).

The amount of sugar required is found as follows:

7-per-cent milk has 4·40 per cent sugar.
15 × 4·40 = 66 (parts of sugar in the milk used in the formula).
66 ÷ 35 = 1·88 (percentage of sugar in the formula of 35 ounces).

There is needed therefore an addition of about 4 per cent of sugar to bring it to the desired percentage.

4 per cent of 35 = 1·40 (No. ounces sugar to be added).

Third Series of Formulas.—Fat to proteids, 8 : 7.

Primary Formula.—Whole milk: Fat 4 per cent, sugar 4.5 per cent, proteids 3.5 per cent. (When using Jersey or Alderney milk add one-fourth water.)

Derived Formulas, giving Quantities for Twenty-ounce Mixtures.

			Per cent.	Per cent.	Per cent.
I.	Milk sugar... 1 oz. Lime-water.. 1 oz. Water, q. s. to 20 oz.	with 5 oz. whole milk =	fat 1·00,	sugar 6·00,	proteids 0·87.
II.	" " " "	" 6 oz. " " =	" 1·20,	" 6·00.	" 1·00.
III.	" " " "	" 8 oz. " " =	" 1·60,	" 6·50,	" 1.40.
IV.	" " " "	" 10 oz. " " =	" 2·00,	" 7·00,	" 1·75.
V.	Milk sugar... ½ oz. Lime-water.. 1 oz. Water, q. s. to 20 oz.	" 12 oz. " " =	" 2·40,	" 5·00,	" 2·10.
VI.	" " " "	" 14 oz. " " =	" 2·80,	" 5·50,	" 2·50.
VII.	" " " "	" 16 oz. " " =	" 3·20,	" 5·50,	" 2·80.

The Application of the Foregoing Formulas in Practice.—General Rules for Varying Milk Percentages.—We have indicated on page 190 the series of formulas most used in laboratory-feeding and have shown how similar formulas can be obtained when the milk is prepared at home. A schedule like that given in the table is useful to indicate in a general way what percentages an average infant may be expected to take. But no schedule can be closely followed with any given child. One can not conclude that because a child is six weeks old he is able to digest milk containing certain percentages, nor certain others because he is six months old. To attempt to follow a schedule too closely is to violate the fundamental principle of percentage feeding, which is to adapt the milk to the child's digestion at any time. In brief, one should begin with weak formulas and gradually increase their strength according to the child's needs and his ability to digest cow's milk (Fig. 37).

How and where to begin.—With infants having any form of disturbed digestion the formula first used should be determined, as will be more fully explained in the later pages, by the nature of these disturbances. With infants having presumably normal digestion it is desirable to begin with the weak formulas: (1) With a newly born infant; (2) with a delicate infant or one much under average weight, of whatever age; (3) with one just weaned; (4) with one who has not previously taken cow's milk; (5) with any infant whose digestion is unknown.

Having decided that we shall begin with weak formulas, it is not always easy to determine with which series the start shall be made. It is true that most young infants digest fat so much more readily than proteids, that those formulas in which the proteids are only one-third the fat (First Series) are usually to be preferred. However, this is not true of all infants; and in the event of any disturbance of digestion arising, especially vomiting or diarrhœa, the Second Series should be used.

Nothing is easier than to derange the digestion in the beginning by the use of too high percentages; such disturbances, though they may not be severe, often continue for many weeks (Fig. 38). The closest attention is required in the beginning. If a good start is made subsequent progress is easy; but with a bad start there is likely to be trouble most of the time. As soon as an infant's capacity to digest cow's milk is ascertained, the food can be increased accordingly.

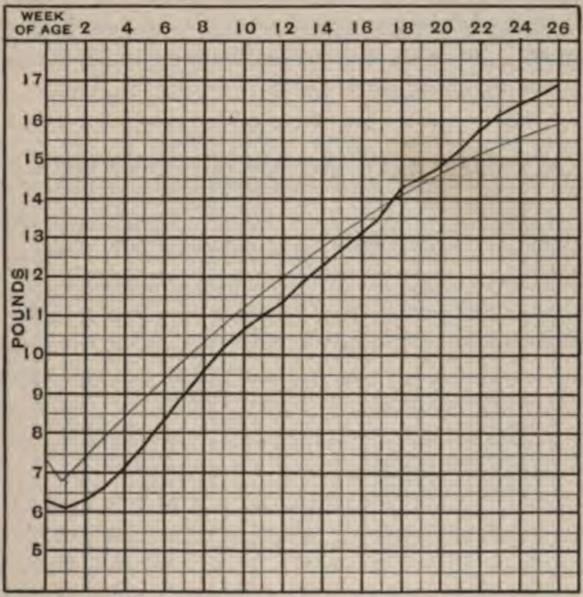

Fig. 37.—Weight curve of bottle-fed infant for first six months. Heavy line that of patient; light line, the normal average. Small child, not particularly vigorous, never put to the breast; feeding begun on the second day from the milk laboratory. Formula: Fat 1 per cent, sugar 5 per cent, proteids 0·33 per cent; at five weeks, taking fat 3 per cent, sugar 6 per cent, proteids 1 per cent; at five months, taking fat 4 per cent, sugar 7 per cent, proteids 2 per cent; not the slightest discomfort or any symptom of indigestion during the entire period. Weight at twelve months, 21 pounds, 8 ounces.

Again, at weaning, or with a child who has previously had no cow's milk, one must begin, even with one whose digestion seems quite normal, with percentages considerably lower than the age and weight would appear to require. A stationary weight for a week or two, or even a loss of a few ounces, is of no importance, provided the change in diet can be effected without deranging digestion; for as soon as a child becomes accustomed to cow's milk the percentages can be raised, and progress is assured (Fig. 34, page 180).

Indications for increasing the food.—While it is important to begin with low percentages, it is a serious mistake to continue with them. We increase the power of digestion by gradually increasing the work the organs are given to do. Abrupt increases are almost certain to disturb digestion. A proper rate of increase is mentioned in the tables of formulas. In them the increase in the fat is usually half of one per cent, and the increase in the proteids one-fourth of one per cent, or less. This is about right for an average child. For many who are delicate the steps of increase should be made only half as great. This can easily be done by using a formula intermediate in strength between any two of those given in the tables.

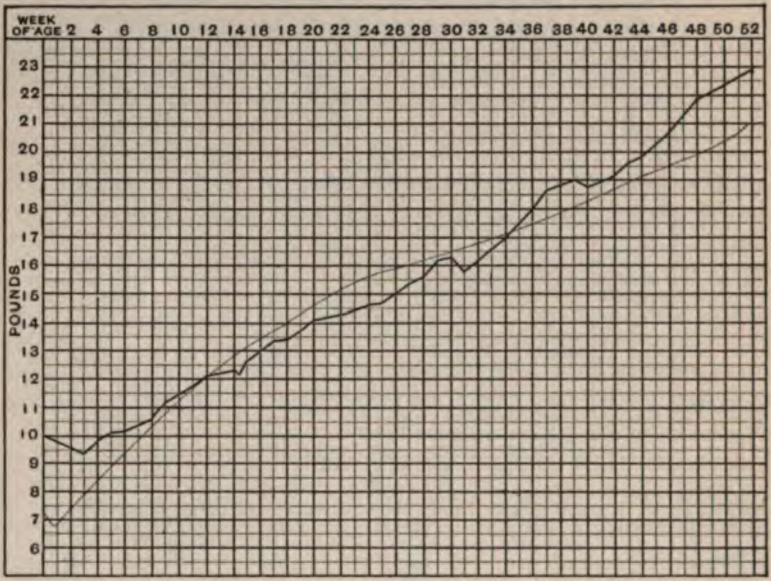

FIG. 38.—Weight curve of artificially fed infant, showing the effect of beginning with too high percentages. Robust child; digestion deranged when a few days old by beginning with fat 2 per cent, sugar 6 per cent, proteids 0·75 per cent; food in two or three days was increased to fat 3 per cent, sugar 6 per cent, proteids 1 per cent. A good deal of indigestion resulted, and the disturbance was such that it was eight weeks before the digestion became normal and the gain in weight regular; progress for the rest of the year satisfactory.

In increasing the quantity, it is seldom wise to add more than a fourth of an ounce to each feeding. During the early weeks both the quantity and the strength of the food should be increased every few days. It may be difficult to tell which of these it is best to do. It is well to alternate; thus, when the infant requires more food, first to increase the quantity; then, after a few days, if still unsatisfied, to increase the strength; the next time, to increase the quantity again, etc. In this way will be avoided the error into which mothers and nurses so often fall, who adopt a single formula and keep on simply increasing the quantity indefinitely whenever the child is unsatisfied. I have frequently seen infants of two or three months taking as much as 7 or 8 ounces every two hours, and even then crying from hunger. After a daily total of 32 to 36 ounces is reached, as happens with most infants by the fourth month, the increase in the food should be chiefly in strength; for the same child at eight months will rarely require more than 40 to 48 ounces.

How rapidly the increase is made will vary much with the individual infant. With a vigorous child, above average weight, with good digestion, the percentages may be raised rather rapidly, and also the quantity given at one feeding. With a small or delicate child, or one with feeble digestion, one must advance much more slowly both with respect to the strength and quantity of food. No greater mistake can be made than to attempt to measure the increase in food by the age of the child. We can not raise the percentages every week or every month regardless of other conditions. The progress in weight is important, yet one should not be guided by it alone in increasing the food. On the low percentages necessary at first no material gain in weight is to be expected. However, if there is no vomiting or colic, if the child is entirely comfortable and sleeps most of the time, and if the stools have a normal colour and odour, conditions may be considered entirely satisfactory. The food may be cautiously strengthened with the demands of the child's appetite, and soon the increase in weight will begin, and when once begun it is likely to continue. On the contrary, if the weight is made the chief concern, there is a constant temptation, when the child is not gaining as rapidly as the mother thinks he should, to increase the food, regardless of conditions, usually with the result of seriously disturbing digestion. The best of all guides to increasing food is the child's demonstrated powers of digestion. If the child is not satisfied and digesting well it is always safe to increase the food.

A caution is necessary against changing the formula too frequently. It is not possible to modify the milk in such a way as to relieve every trivial discomfort or disturbance an infant may have. Nurses are usually ready to ascribe every slight symptom to the food, particularly if they have strong opinions of their own upon the subject of feeding, and are not in full sympathy with modern ideas of milk modification. Very often the cause is outside of the food and even of the organs of digestion. (See Fig. 39, page 207). Unless some very definite symptoms of indigestion, such as severe colic, vomiting, etc., are produced by the formula ordered, it is usually better to continue with it for at least two days, as it is hardly possible in a shorter time to determine what the child's digestive organs are capable of doing. For slight disturbances of a transient nature it is usually enough to dilute the food for a day or more; just before the bottle is given, one ounce or more of milk may be poured off and replaced by boiled water.

To Reduce Milk Formulas to Percentages.—In order to appreciate the composition of any milk formula which a patient may be taking it is necessary to reduce this to its approximate percentages. This is particularly important as regards the fat and proteids. One who forms the habit of making such calculations soon finds it easy, and secures a basis for comparison with the percentages given as proper for the average normal child. A simple method of calculation is as follows: To determine the percentage of any constituent in the food, multiply its percentage in the original milk, cream, or top-milk (compare pp. 146, 151, and 152) by the number of ounces of each in the food, and divide by the total number of ounces of food prepared.*

Special Modifications Required by Particular Symptoms.—Many of the children for whom the physician's advice is sought in matters of feeding are not thriving, or, besides, are suffering from some evident symptoms of indigestion, and for these reasons changes in the food are required. In adapting milk for such cases one must rid his mind entirely of the notion that the food can be prescribed according to the child's age or even its weight, although both must be taken into account. The essential thing is the condition of the digestive organs, and unless this is carefully considered, failure is almost inevitable. To decide as to proportions with which it is best to begin one must know, besides the age and weight, the previous gain or loss, the nature and quantity of the food which has been taken, the appetite, the number and character of the stools, and also whether any such symptoms are present as vomiting, colic, constipation, discomfort, or disturbed sleep. In any case the first prescription is somewhat in the nature of an experiment, but if the symptoms have been intelligently judged the experiment is likely to prove successful.

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* A patient is taking a formula composed of cream 4 ounces, milk 16 ounces, milk sugar 1½ ounces, in a mixture containing 36 ounces. The cream is ordinary centrifugal cream, estimated to have 20 per cent fat; the milk is good average milk, estimated to have 4 per cent fat.

4 × 20 = 80, the parts of fat in the cream
16 × 4 = 64, " " " milk
144, " " " total food
144 ÷ 36 (number of ounces of food) = 4, the percentage of fat in the food.

The proteids are calculated in the same way. In the illustration we estimate the proteids of 20-per-cent cream at 3·05; in the whole milk, at 3·50.

4 × 3·05 = 12·20, the parts proteids in the cream
16 × 3·50 = 56·00, " " " milk
68·20, " " " total food
68·20 ÷ 36 = 1·90, the percentage proteids in the total food.

In a similar way, sugar is calculated. The sugar of a 20-per-cent cream may be estimated at 3·90; in the milk, 4·50.

4 × 3·90 = 15·60, the parts of sugar in the cream
16 × 4·50 = 72·00, " " " milk
87·60, " " " mixture
87·60 ÷ 36 (number of ounces of food) = 2·40, the percentage of sugar in the food before any is added.

To add 1½ ounces to a 36-ounce mixture adds approximately 4 per cent of sugar; for 1·5 is 4 per cent of 36 [1·5 ÷ 36 = ·04].

The total sugar in the mixture therefore is 2·40 + 4, or 6·40 per cent.

The formula contains therefore approximately, 4 per cent of fat, 1·90 per cent of proteids, 6·40 per cent of sugar.

This method of calculating percentages from ounces is exactly the converse of that given on page 195, for calculating ounces from percentages.

Even with infants who are properly fed there are few whose digestion remains perfectly normal throughout the entire first year. Changes in the food are necessary from time to time, even in the most healthy, to meet special symptoms which may arise. Many of these are due to disturbances of a minor character, but are none the less important, as they may lead to serious consequences when not immediately recognized and properly treated.

Vomiting.—The common causes of habitual vomiting are: too frequent feedings, too much food at one time, too high fat or too high sugar, especially if the sugar is maltose or cane sugar.

An infant who vomits should never be fed at shorter intervals than three hours, even if only four or five weeks old. If considerable quantities are ejected almost immediately after feeding, it is usually because too much food has been given. Other causes must be considered also—the food may be too rapidly taken, the child may be moved about too much, the abdominal band may be too tight, etc. The frequent regurgitation, often one or two hours after feeding, of sour, curdled milk or of a watery fluid, is usually an indication that the proportion of fat is too high. Sometimes it is the sugar that is in excess, and sometimes both fat and sugar are at fault. The first indication is to reduce the fat. Formulas from 10-per-cent milk should not be used, and, if the symptom is at all aggravated, formulas from whole milk (page 196) are to be preferred even for very young infants. The sugar also should be reduced by one-third or one-half, and only milk sugar should be used. Other changes which are sometimes helpful are to use twice the usual amount of lime-water, making this 10 per cent, or 2 ounces in each 20-ounce mixture. It is also important that the food be taken slowly and that the child be kept perfectly quiet, on its back, after feeding.

Constipation.—The principal causes of constipation referable to the food are too low total solids, too low fat, too high proteids. Habit and general training are also important factors. Sterilization, and to a slight degree pasteurization, causes milk to be somewhat constipating. During the first few weeks, if the percentages are low, as I believe they should be, there is often a species of constipation present which is simply the result of the low total solids in the milk formula given. The bowels usually move every day, sometimes even twice a day; but the stools are often small and rather dry. Unless there is manifest discomfort on the part of the child, such a condition may be disregarded, especially if the odour and colour of the discharges are nearly normal. As the proportions of all the elements of the food are gradually increased along the general lines previously indicated, this form of constipation passes away. Mothers and physicians often expect that the bottle-fed infant will have during its first month or two the two or three large stools daily to which they have been accustomed with healthy breast-fed infants. But finding instead only one movement a day, and that small and sometimes dry, they at once resort to laxatives or enemata, and by their use really cause much of the trouble they are seeking to remove. Again, if the physician tries to remedy the constipation by rapidly raising only the fat, as is often done, the constipation is rarely relieved, but there is frequently produced a serious disturbance both of the stomach and the intestines.

The low fat is very often the explanation of the constipation seen when infants are fed upon formulas derived from whole milk. If such is the case relief may be afforded by changing to formulas made from 7-per-cent milk or to those from 10-per-cent milk, by which means higher fat with the same, or, if desired, lower proteids may be obtained. The increase in the fat to overcome constipation can only be carried up to a certain point; this is generally about 3 per cent for a young infant and 4 per cent for one who is older. If the fat is raised much beyond this other disturbances of digestion, particularly habitual vomiting, are likely to result. Some other means of overcoming the constipation should be resorted to.

Too high proteids are often given with too low fat, as in mixtures derived from whole milk, and the constipation may be the result of one factor quite as much as the other. A reduction in the proteids and an increase in the fat may be accomplished at the same time by using modifications made from top-milk as suggested just above. The constipating proteid of cow's milk is the casein. By the use of whey modifications (page 210) the amount of casein given can be reduced to a very low point and at the same time the total proteids, in these mixtures chiefly lactalbumin, can be kept sufficiently high for the child's nutrition. This is one of our most effective means of relieving chronic constipation.

The substitution of the milk of magnesia for lime-water as an antacid in milk modifications is often of service. Its use may be continued for several months without harm. One teaspoonful added to the total food for the day is usually sufficient; this amount may be slightly increased or lessened according to the effect produced.

The slightly laxative effect of maltose may be utilized in milk modifications. It may take the place of the lactose which is added in any of the groups of formulas already given. The most convenient form is some one of the malted foods.

Colic and flatulence.—The habitual colic of early infancy is usually due to too high proteids, exceptionally to too high sugar. Excessive flatulence may occur also when cereal gruels are used as diluents in place of plain water. The symptom may be relieved by a reduction in the total proteids by using a weaker formula of any series than the one employed; or, often better still, by the use of whey modifications, it being the casein which is at fault, as in the case of chronic constipation. The coexistence of constipation of course greatly increases the amount of both flatulence and colic.

Curds in the stools.—The appearance of curds in the stools is due to the same cause as habitual colic, and is usually associated with it. The curds generally appear as white masses or lumps; sometimes they are gray or green, coated with mucus, and expelled with effort. Colic, curds in the stools, and constipation are a frequent combination, and are usually due to too high proteids or to inability to digest the casein of the milk given. The treatment of the condition has been considered in the foregoing paragraphs.

Loose, green, or yellowish-green stools of a sour odour.—These are sometimes due to too high a percentage of sugar, sometimes to an excess of fat. The number of stools is usually from two to five daily. In appearance the stools resemble thin scrambled eggs. The small yellowish masses are often mistaken for curds. Stools such as those described are often seen in nursing infants as well as in those artificially fed, and the condition is not incompatible with steady and regular gain in weight. After it has persisted any length of time mucus is regularly present, and an intractable intestinal catarrh may be produced.

Large, dry, white or gray stools.—These are often smooth, and are generally due to an excess of fat. They have usually a peculiarly foul odour, owing to the presence of fatty acids; the masses may be distinguished from curds by their solubility in ether.

No gain in weight without evident symptoms of indigestion.—This is sometimes due to too weak mixtures, all the percentages being too low, the child usually manifesting signs of hunger. Occasionally it is due to the fact that all the percentages, particularly the fat, are too high. In the latter case it frequently happens that the appetite is much reduced, so that the infant takes perhaps less than half his usual allowance. Too frequent feedings and the practice of constantly coaxing the infant to take more food, often produce the same aversion to food. It is much better to offer food at three-hour intervals and take away the bottle as soon as the child shows that he does not want more.

Modifications in the food to meet the indications afforded by more serious conditions than those here described are considered in the later pages devoted to Difficult Cases of Feeding.

The Apparatus required for the Preparation of Milk at Home.—This includes an 8-ounce glass graduate, a glass or agate funnel, a cream dipper, a pitcher for mixing food, feeding-bottles, a tall cup for warming the food, a small ice-box, and a sterilizer. Other articles needed are lime-water, boiled water fresh every day, milk sugar, rubber nipples, absorbent cotton, bottle-brushes, borax or boric acid, bicarbonate of soda, and an alcohol lamp, or better, if gas is available, a Bunsen burner, which should stand upon a zinc-covered table in a room adjoining the nursery. The best style of bottle is that which can be most readily cleaned. The graduated cylindrical bottles with wide mouths are to be preferred. The best nipples are those of plain black rubber, which slip over the neck of the bottle, and are not so thick as to prevent their being turned inside out for cleansing. Those with a long rubber tube going to the bottom of the bottle should not be used. In many cities their use is prohibited by law. The hole in the nipple should be large enough for the milk to drop rapidly when the bottle is inverted, but not so large that it will run in a stream. When not in use, nipples should be kept covered in a solution of borax or boric acid. The most scrupulous care of both nipples and bottles is necessary. Bottles should first be rinsed with cold water, then washed with hot soap-suds and a bottle-brush. When not in use they should stand full of water to which borax or boric acid has been added. Before the milk is put into them they should be rinsed and again boiled.

Directions for Preparing the Food.—All the food needed for twenty-four hours should be prepared at one time. This saves much time and trouble, and is in every way simpler than preparing each feeding separately. The first thing to be decided is the formula to be used; next, the quantity of food for twenty-four hours with the number of feedings into which it is to be divided.

Let us suppose that we wish to give 3 per cent fat, 6 per cent sugar, and 1 per cent proteids—formula V of the First Series—and that we wish to prepare 7 feedings of 5 ounces each, or 35 ounces of food. For a 20-ounce mixture containing 3 per cent fat we will require (see page 194) 6 ounces of 10-per-cent milk, 1 ounce of sugar, and 1 ounce of lime-water; the balance will be water; since the sugar dissolves, 13 ounces of water will be needed. Now to make 35 ounces, we will require three-quarters more of each ingredient than for 20 ounces—i. e., 10½ ounces of the milk, 1¾ ounces of sugar, 1¾ ounces of lime-water, and the balance, or 22¾ ounces, of water. The amount of water need not be calculated each time; enough is added to make the quantity required.

If instead of bottled milk, or milk and cream, the patient is using milk fresh from the cow, as soon as received it should be strained through three thicknesses of cheese cloth or a layer of absorbent cotton, into quart jars or milk bottles, and allowed to stand in ice-water or cold spring water for at least four hours. The top-milk, in this case the upper third, may then be removed.

The milk sugar should be dissolved in boiled water, which is then mixed with the milk in a pitcher and the lime-water added. The food is now divided into the seven bottles, which are stoppered with cotton. They are placed at once in an ice-chest, or first sterilized, then cooled, and afterward placed upon ice.

Directions for Feeding.—The food should be warmed to about 100° F. before feeding, best by placing the bottle in a tall pitcher or cup filled with water at a little above this temperature, not by pouring the food from the bottle into a saucepan. The temperature of the food may be tested by the nurse with a thermometer, or by pouring a few drops upon the front of the wrist; it should feel warm, but not hot. The nurse should never take the nipple of the bottle into her own mouth. A bottle should not be warmed over for a second feeding. A child should not be more than twenty minutes in taking its food, and should not sleep with the nipple of the bottle in its mouth. It is preferable to have a young infant held while taking its bottle. If this is not done, the bottle should at least be held in such a position that the neck of the bottle is kept full, so that the child gets milk, and not air. It is even more necessary than in breast-feeding that rules as to frequency and regularity of meals be observed. The table which follows indicates the size and the number of meals and the intervals of feeding. This is to be taken only as a general guide. The quantity for one feeding can not always be definitely stated. Few children, however, will require less than the smaller quantities, and still fewer will require more than the larger quantities mentioned.

Schedule for Feeding Healthy Infants during the First Year.

AGE.	Interval between meals, by day.	Night feedings 10 P.M. to 7 A.M.	No. of feedings, 24 hours.	Quantity for one feeding.		Quantity for 24 hours.	
	Hours.			Ounces.	Grammes.	Ounces.	Grammes.
2d to 7th day........	2	2	10	1 –1½	30– 45	10–15	310– 460
2d and 3d weeks.....	2	2	10	1½–3½	45–110	15–35	460–1,090
4th and 5th weeks...	2	1	10	2½–3½	75–110	25–35	775–1,090
5 weeks to 2 months.	2½	1	8	3 –5	90–155	24–40	745–1,250
2 to 5 months.......	3	1	7	4 –6	125–185	28–42	870–1,300
5 to 9 months.......	3	0	6	5 –7½	150–235	30–45	930–1,400
9 to 12 months......	4	0	5	7 –9	220–280	35–45	1,090–1,400

The Use of other Food than Milk during the First Year.—In the discussion up to this point nothing but the elements of milk has been considered. Upon these alone I believe that the average healthy infant is best nourished for the first four or five months. The use of the various cereal decoctions as an addition to the milk for young infants is a subject much discussed, and the question can not be regarded as settled. I am quite convinced that this is a useful measure for some infants, but not that it is desirable for all. Surely no point in infant-feeding is better established than that the early use of much farinaceous food often results in serious harm. The addition to milk of farinaceous food in any considerable quantity should, I think, in the feeding of young infants be limited to those in whom some special conditions are present, particularly those who have more difficulty than usual in digesting the milk proteids. This subject will be considered more fully under the discussion of Difficult Cases of Feeding.

For the average healthy infant it is desirable to begin with farinaceous food in some form by the fifth or sixth month. By this time the power of digesting starch is sufficiently strong for the infant to receive some of its carbohydrates in this form, instead of all of it in the form of sugar, as has been previously the case. As starch is added, the sugar should be gradually reduced. The form of starch used may be a gruel made of barley, oatmeal, or arrowroot, or some of the farinaceous foods (page 165). This will take the place of part or all of the boiled water in the preparation of the food. It is thus given with each of the feedings. By the eleventh or twelfth month the quantity of the cereal may be increased. The choice between the different cereals will depend upon the individual case. Where there is a tendency to constipation, oatmeal is to be preferred; at other times barley or wheat flour.

The only other things to be advised during the first year are beef juice (for preparation see page 163) and the juice of some fresh fruit. Beef juice may be begun in the tenth or eleventh month; at first not more than two teaspoonfuls should be given daily. The best fruit juice is that of the orange, which may with advantage be given to most infants over ten months old. Beginning with half an ounce, the quantity may be gradually increased to two ounces, given preferably about one hour before the second milk-feeding.

FEEDING IN DIFFICULT CASES.

Two distinct groups of cases are included under this head: (1) Infants who, owing to feeble digestion or individual peculiarities, do not thrive, even from the outset, upon the usual milk modifications, although they may be used intelligently; (2) the much larger class, who have prolonged disturbances of digestion, or chronic indigestion, the result of previous improper methods of feeding or equally improper nursing. In the aggregate the number of children included in these two groups is quite large, and few cases in the practice of the physician cause him more trouble or anxiety. Even one of large experience often finds himself baffled for a long time by the problems which individual cases present. The difficulties are greatest with young infants, in cities, in institutions, in hot weather, with infants suffering from constitutional debility, and in cases of long standing. That chronic indigestion in a young infant is a serious thing is often not appreciated. The mother is apt to think the problem one easy of solution; she only wants to be told what to feed her baby, imagining that a single food prescription should set the child right at once. The physician, too, sometimes regards the condition lightly because these infants do not seem really ill; he therefore considers the subject hardly important enough for his serious, continuous attention. What I wish to emphasize is that these cases are serious, that they are difficult, that in most of them nothing can be accomplished without close and continuous personal observation, that they do not tend to right themselves, and that infants' lives are often sacrificed as a result of bad management.

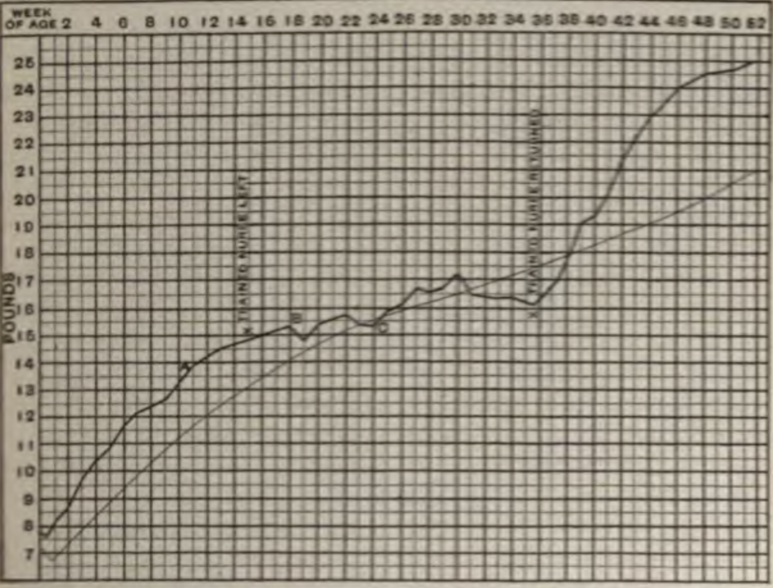

FIG. 39.—Weight chart showing the effect of intelligent care. Maternal nursing in the beginning; *A*, began part feeding; *B*, attack of indigestion; *C*, weaned entirely. The departure and return of the trained nurse are indicated upon the chart. In the interval there was constant indigestion for which no sufficient explanation could be found in the food. Subsequently this was discovered to be due to the carelessness and neglect of the nurse. Immediate improvement on the return of the trained nurse without any important change in the food. It will be noticed that during the four and one-half months of the trained nurse's absence the net gain in weight was only 1 pound 3 ounces.

While these infants present great variety in their symptoms, and must be carefully individualized in their management, there are some general principles applicable to all. One should begin by obtaining a careful history of what has been previously tried, in order to get all possible information respecting the type of indigestion which the child presents. These previous efforts in feeding should be studied with great minuteness; the different changes made and the effect of each one upon the principal symptoms, the vomiting, the stools, and the child's weight should be considered. With a good history obtained from an intelligent mother or nurse one can often at once determine where the mistakes have been made, and in many cases the same mistake has been repeated with each change of food.

A thorough investigation into the nursery routine should be made to ascertain not only what has been tried, but how it has been tried. It is frequently found that the failure is due not to any fault with the food prescription (Fig. 39), but because the food has been improperly prepared or administered—e. g., the food has been cold, the bottles dirty, the nipples sour, the food too rapidly given, too much at one time, or at too short intervals, etc. General statements of nurses and mothers, no matter how experienced, can not be trusted. Success in treatment will depend largely upon how accurately one is able to discover the essential cause or causes of trouble and the nature of the disorder of digestion in the case under treatment. Without such knowledge all is haphazard experimentation.

In dealing with these cases drugs are of little assistance; in most cases they are better omitted altogether.

In carrying out any line of treatment little can be accomplished without continuous observation at fairly frequent intervals on the part of the physician and the co-operation of an intelligent mother or nurse. Particular attention should be paid to the stools, which the physician should always see for himself, to the presence of colic or flatulence, vomiting, the appetite, and the body weight. A daily record is of great assistance. The weight though important is not the only guide as to progress. It should be taken regularly in order that a steady loss may not go on unnoted; but the first signs of improvement are usually observed in other symptoms—the child is more comfortable, sleeps better, and suffers less from its special disturbances of digestion.

Quantities, Intervals of Feeding, Concentration of Food.—With some children one succeeds better with smaller quantities and more frequent feedings; with others, larger quantities and longer intervals are preferable. Generally speaking, the intervals should be longer than in health. It is seldom wise to make them less than three hours for young infants, or less than four hours for those who have passed the eighth or ninth month.

Regarding the effect upon the digestion of the concentration of the food (i. e., a large quantity of a weak food, or a small quantity of a strong food), great variations are seen with different children. The usual tendency when an infant suffers from indigestion is to dilute the food, and in most cases this is perfectly proper; but to continue increasing the dilution because the patient does not do well may be the very worst treatment. This may do harm by causing too much dilution of the digestive fluids. Small feedings, not weak food, are what benefit some of these children most, the balance of the daily amount of water needed by the child being given between the feedings. Thus, instead of giving eight ounces of a weak food every four hours, we may do better with four ounces of a much stronger food, allowing the child three or four ounces of water one hour or one hour and a half before the feeding.

In very troublesome, protracted cases minor variations in the composition of the food or slight changes in the plan of feeding rarely accomplish much. Radical changes are usually necessary. If small feedings and short intervals have failed, one may succeed with larger feedings and much longer intervals. If very dilute food in large quantities has failed, improvement may follow much smaller feedings and a much stronger food. For similar reasons the most brilliant results are often obtained from as complete a change in the diet as possible. An infant who has been long on farinaceous foods is most likely to improve when these are stopped entirely and suitable percentages of cow's milk given. One whose digestion has become seriously deranged while taking milk, and whose symptoms have continued in spite of many variations in percentages, is sometimes helped by nothing so much as temporarily withdrawing all milk. (See Fig. 40, page 216.)

The Modification of Cow's Milk in Difficult Cases.—Many more problems in difficult feeding are solved through a proper adaptation of cow's milk to the digestion of the infant than in any other way, excepting possibly a resort to wet-nursing, which in most cases is not available. One should therefore be slow to discard cow's milk and adopt any of the lauded substitutes which the manufacturer offers; but should seek rather to discover how he can modify the milk to enable the child to digest it, stopping milk entirely only as a last resort.

For purposes of treatment the cases may be divided into several groups according to the nature of the disturbance of digestion which the child presents and the special element of the food with which he has most difficulty. Those who have especial trouble with the proteids constitute probably the largest group. The symptoms are varied, the most frequent ones being colic, flatulence, sometimes diarrhœa, but generally constipation, with stools which are dry, granular, hard, and often coated with mucus. There is also anæmia and general malnutrition. To overcome the difficulty in digesting the proteids of cow's milk several means may be employed.

Reduction in the total proteids.—This may be accomplished without making any change in the proportions of fat or sugar by using weaker formulas derived from 10-per-cent milk, rather than those from 7-per-cent milk, or those from whole milk. For some cases it may be desirable to use with a given percentage of fat even lower proteids than those of the First Series. Such formulas may be obtained from a 16-per-cent cream (the upper 6 ounces from one quart). In this the proteids are approximately one-fifth the fats. Any formula desired may be calculated in the manner indicated on page 195. This plan is suited to a small number of cases, but is not so likely to succeed with the majority as some of the methods which follow.

The use of milk from which the casein has been removed—whey modifications.—After the casein has been coagulated by rennet and then strained out, the whey (page 162) is left, which will contain all the soluble proteids—lactalbumin, lactoglobulin, etc. Most of the fat is removed by the process, but this can be supplied by adding cream, in which the percentage of casein is small.

Table Showing Composition of Formulas Made from Whey.

		Fat.	Sugar.	Whey Proteids.	Casein.
I.	Whey 14 parts; 20% cream 1 part; water 5 parts	1·60	4·00	0·65	0·10
II.	" 19 " " " 1 "	1·90	5·00	0·90	0·10
III.	" 15 " " " 1 "	2·10	5·00	0·90	0·15
IV.	" 11 " " " 1 "	2·50	5·00	0·90	0·20
V.	" 9 " " " 1 "	2·80	5·00	0·90	0·25
VI.	" 8 " " " 1 " milk 1 part	3·00	5·00	0·85	0·50
VII.	" 6 " " " 1 " " 2 "	3·60	5·00	0·85	0·85
VIII.	" 5 " " " 1 " " 2 "	4·00	5·00	0·90	0·90

The sugar may readily be raised to 6 per cent by adding one even tablespoonful of milk sugar to each 40 ounces of the food; to 7 per cent, by adding two tablespoonfuls to each 40 ounces of the food. The addition of one part lime-water to each 20 parts will cause a negligible reduction in all the percentages of the table.

Where slightly lower fat is desired with the same proportion of proteids, it may be readily obtained by substituting 16-per-cent cream for 20-per-cent cream; a still greater reduction in the fat, by using a 10-per-cent top-milk. Lower proteids than are given in the table, with the same proportion of fat, may be obtained by replacing part of the whey with water.

Whey modifications are applicable to a large number of conditions. By using them we are able to raise the total proteids to a much higher point than is otherwise possible, thereby avoiding the dangers incident to keeping infants long on very low proteids. These modifications, on account of the high proportion of soluble proteids which they contain, form a much nearer approach to woman's milk than any other combinations now available. With them constipation is relatively infrequent, while colic and flatulence seldom cause any trouble.

There is no objection to the use of these modifications of milk for several months. With improvement in digestion infants may gradually be brought to digest the larger percentage of casein in the usual modifications, which are somewhat simpler in preparation.

The use of peptonized milk.—This aims at partial predigestion of the milk proteids before the food is given. The method of peptonizing milk has already been described (page 158). It is important that proper percentages be obtained before the peptonizing is done. The proportions usually recommended with the peptogenic milk powder give 4 per cent fat, 7 per cent sugar, and 2 per cent proteids; these are too high for most infants with feeble digestion, as are also the other formulas generally advised for use with the peptonizing tubes or tablets. I have obtained better results with such percentages as those of formulas III, IV, and V of the Second Series; sometimes, however, even with lower fats than these, as in IV, V, and VI of the Third Series. The duration of the predigestion of the food will depend upon the amount of assistance required by the child. As it takes about two hours to peptonize milk completely, the process at the end of fifteen minutes will be only one-eighth completed, and at the end of half an hour only one-fourth, leaving thus in the one case seven-eighths and in the other three-quarters of the work of proteid digestion to be done by the child. Where required at all, I have usually found it best to continue peptonizing for at least fifteen minutes, often for half an hour or even an hour. I prefer to peptonize each bottle separately immediately before feeding, since the ferment in such cases continues its action in the stomach. If the amount for the entire day is peptonized at one time and the milk raised to boiling point the ferment is destroyed. The bitter taste produced at the end of about fifteen minutes is evidence of the conversion of some of the proteids into peptones, but in practice is rarely found to interfere with its use, except with children over seven or eight months old. After the first two or three bottles younger infants take this bitter milk as willingly as any other food.

The partial predigestion of the milk proteids may be continued for several weeks, the amount of assistance given the child being gradually lessened by shortening the duration of the process, as the stomach becomes more and more able to do its normal work. There is a serious objection to the use of predigested foods for as long a period as five or six months; in such cases the organs do not gain, but rather lose in their digestive power.

The addition of the citrate of soda.—The use of the citrate of soda to aid in the digestion of milk was first suggested by Wright (London) a number of years ago. It has been more recently revived by Poynton and others. The theory of the action of the citrate of soda is that it delays casein coagulation in the infant's stomach by uniting with its

	Fat.	Sugar.	Proteids.
I. Skimmed milk (upper 6 ounces removed) has..	1·80	4·50	3·60 per cent.
II. Diluted once gives......................	0·90	2·25	1·80 " "
III. " twice "	0·60	1·50	1·20 " "
IV. " three times "	0·45	1·12	0·90 " "

The sugar can be raised to about 7 per cent by adding half an ounce of milk sugar to each 20 ounces of No. I; one ounce to each 20 ounces of Nos. II and III; 1¼ ounces to each 20 ounces of No. IV. If possible, without disturbing digestion, the percentages of sugar should be raised to 7 per cent or even higher to prevent the loss in weight.

Modifications whose basis is skimmed milk are to be recommended for the relief of special digestive symptoms, particularly vomiting. As they are rather constipating they are also applicable to intestinal conditions if the bowels are loose. But children seldom gain in weight properly upon them, as their caloric value is very low. As soon as possible the fat should be raised to the amount present in whole milk. The value of skimmed milk modifications is often much increased if they are partially or completely peptonized.

There are many infants who have almost equal trouble with both fats and proteids. If the symptoms due to these elements are not severe and the child can tolerate low percentages, very weak formulas made from whole milk should be used—e. g., 1 ounce in a 20-ounce mixture, in which, if a 4-per-cent milk is used, the fat will be 0·20 per cent and the proteids 0·17 per cent. The sugar should be raised to 7·50 per cent (1½ ounces in 20-ounce mixture), and in some cases it can be increased to even 10 per cent (2 ounces in 20-ounce mixture).

The use of cereal gruels as diluents for milk.—I believe cereal gruels to be unnecessary and on the whole during the early months undesirable for healthy infants with normal digestion; also, that used in considerable amounts with young infants they are capable of producing, and as commonly used do actually produce, much intestinal indigestion. But for many infants with disturbed digestion, especially for those whose trouble is particularly with the proteids, they are of undoubted value. Various theories have been held regarding the effect of the addition of cereals upon the digestibility of milk. Some hold that their effect is simply that of diluents, they acting like so much water. The traditional belief, however, has been that their effect is a purely physical one, the admixture of such substances with cow's milk preventing the coagulation of the casein in the stomach into large, solid masses, but instead producing a softer curd, the digestion of which is attended with less difficulty. When a cereal gruel is substituted for water as a diluent for milk, it is sometimes found that the percentage of proteids can be increased without disturbing digestion. At the same time constipation may be relieved because of the possibility of thus increasing the total solids in the food given. Improvement in nutrition and gain in weight may follow.

Cereal gruels are made from the grains, or more readily from the prepared flours of barley, oatmeal, or rice, or from arrowroot, wheat flour or corn starch. One even tablespoonful of any of these flours to one pint of water makes a gruel of about the right consistency. This adds about one per cent of starch to any of the foregoing formulas. Gruels made from flours should be cooked for at least twenty minutes. When made from the grains, from four to six hours' cooking is required. Lately the dextrinization of cereal gruels has been much practised, but when they are used as diluents this has seemed to me to have no marked benefit.

The use of larger amounts of farinaceous food for infants—Keller's "Malt Soup."—The experiments of Keller (Breslau) indicate that carbohydrates may have an important action in checking the decomposition of milk proteids in the intestine, and thus saving nitrogen to the body. He found that a decided diminution in the elimination both of nitrogen and phosphoric acid occurred with the use of additional carbohydrates. He advocates the use of a very much larger amount of farinaceous food with milk than is suggested above. Milk to which starch and malt are added according to his directions, is known as Keller's "Malt Soup." *

Whether Keller's explanation be the correct one or not, it is certainly true that, if used as he has advised, many young infants can take a much larger proportion of starch than was formerly thought possible. Furthermore, it is rare that the stools of infants so fed show evidences of proteid indigestion.

The indications for the use of these additions to milk are found with infants who show no marked symptoms of indigestion, but who can not be made to gain in weight with our ordinary milk modifications. In this class belong many infants of the marasmus type. With them, when the low percentages of the fat or proteids of milk that they can take without disturbing digestion are given, the weight is either stationary or they lose. But if the percentages are raised, digestion is immediately disturbed. The addition of the large amount of carbohydrates in the form specified, is sufficient to raise the caloric value of the food to a point adequate for the needs of the child. But it is essential that the condition of the digestive organs be such that these additional carbohydrates can be tolerated, or disastrous results may follow. If there is present a catarrhal condition of the stomach or intestines, or even marked functional disturbance attended by vomiting or by looseness of the bowels, the large amount of carbohydrates is contraindicated; they almost invariably aggravate the symptoms. It is not wise to continue this food for a long period. If the limitations laid down are carefully observed, it is possible to greatly benefit a large group of infants whose nutrition is very difficult.

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* Keller's formula is as follows: Wheat flour, 3 ounces by volume; Loeflund's malt soup extract, (an extract of malt with potassium carbonate) 3¼ ounces; water, 16 ounces; milk, 16 ounces. The malt extract is mixed with warm water. The wheat flour is carefully rubbed up with the milk and strained; then all the ingredients are mixed and brought slowly to the boiling point with constant stirring. For young infants this is diluted with an equal amount of water.

I have found it advantageous to modify the formula in some important particulars: First, by using smaller amounts both of the wheat flour and malt extract; for most infants half the quantity specified and sometimes even less than this are I think preferable; secondly, by cooking the wheat flour in the water for twenty minutes before the ingredients are mixed and heated.

To secure the best results it is necessary to vary the proportions of milk, flour and malt according to the indications afforded by the symptoms of the individual child. Beginning with the proportions suggested, the amount of milk may gradually be increased until with older infants it may form two-thirds to three-fourths of the total food. Where there is special difficulty in the digestion of fat it is sometimes best to use skimmed milk.

Substitutes for Milk.—There are conditions in which for the time being infants seem incapable of digesting even the smallest proportions of the fat and proteids of milk, no matter how modified. This is most frequently seen in acute derangements of digestion, especially when associated with acute gastro-enteric intoxication. There are also some chronic derangements of digestion in which the same procedure is of value. In ordinary practice, however, the mistake usually made is that of resorting too early to this expedient instead of carefully adjusting the milk percentages to the symptoms. Another mistake is that of continuing for too long a time a food containing no fresh milk.

The advantage which results from stopping milk in these cases is due chiefly to change of diet. Where fat and proteids are very difficult of digestion it may become necessary to give temporarily a food composed almost entirely of carbohydrates. They may be administered either as some of the farinaceous or malted foods. Such a change is more likely to be successful in intestinal than in gastric cases, and chiefly where colic, constipation and failure to gain in weight have long been prominent symptoms. If the bowels are loose, farinaceous foods are more likely to be useful; if they are constipated, the malted foods. These may be continued alone for a limited time—a few days or a few weeks—according to the severity of the symptoms, and then milk in some form added; for it does not follow because a child at one time can not digest milk that it can never do so. While one must begin with something which the child can digest and assimilate, he must get back to rational milk-feeding as soon as possible. For example, it may be advisable to withhold milk for two or three weeks, and then to begin with as small a quantity as one ounce in the total food of a day; after two or three days a second ounce may be added, and so on, gradually increasing the proportion of milk as the child is able to digest it (Fig. 40). In some cases it may be better to begin by adding whey to the farinaceous food, and in still others small quantities of condensed milk. Since some are able to take fat sooner than proteids, very small quantities of cream may be tried as an addition to the food. All substitutes are to be regarded merely as temporary expedients, and the purpose should be to get the child back gradually to a suitable milk formula.

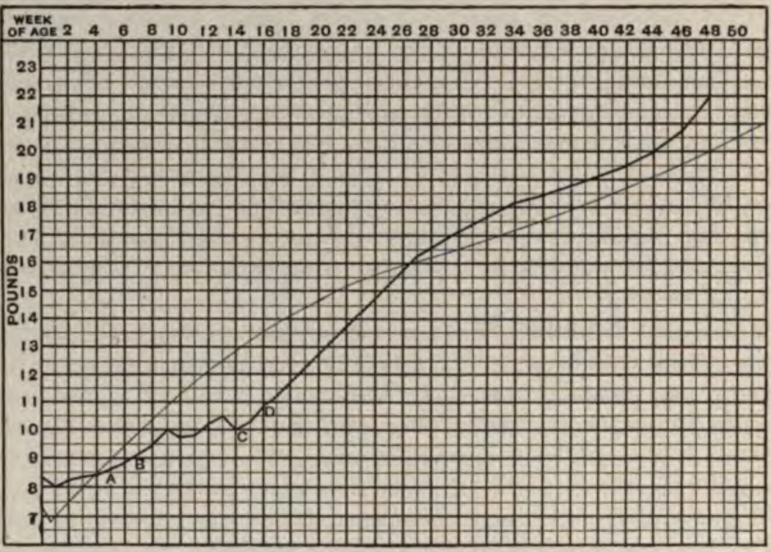

FIG. 40.—Weight curve, showing the advantage of temporarily stopping milk. A fairly vigorous child, nursed entirely by a nervous mother for five weeks, but did badly. *A*, began part feeding; *B*, weaned entirely on account of constant indigestion; *C*, because of continued indigestion, colic, and general discomfort, all milk stopped for two weeks and a malted food substituted; *D*, milk resumed. Subsequent progress satisfactory.

If such addition of fat or milk proteids causes digestive disturbance, nitrogenous food may be supplied in the form of beef juice, beef peptones, broth, white of egg, somatose, plasmon, etc., these being added to the farinaceous or the malted food which is given. There is always great risk in continuing indefinitely a food which does not contain some fresh milk; extreme anæmia, malnutrition, rickets, or scurvy may be the result.

SUMMARY OF INFANT-FEEDING.

Choice of Methods of Feeding.—A faithful trial of maternal nursing should always be made unless there are some very urgent reasons against it; but nursing should not be continued if the child is persistently uncomfortable, suffers constantly from symptoms of indigestion, and does not gain in weight.

Wet-nursing, although theoretically the next choice to maternal nursing, is so difficult that in private practice it should be reserved for certain special cases. In infants' hospitals and foundling asylums the difficulties of artificial feeding are greatly increased, and wet-nursing should be employed when possible.

Artificial feeding has become the general alternative to maternal nursing. If circumstances are such that maternal nursing is almost certain to be a failure, and if at the same time they permit the best artificial feeding, the infant should not be put to the breast at all.

Methods of Artificial Feeding.—The only reliable substitute for breast-feeding is some modification of fresh cow's milk. My own opinion is, that for healthy infants it is best in the early months to use only the milk elements—fat, sugar, proteids, and salts—with lime-water, varying the percentages of these to suit the infant's digestion. The milk laboratories afford facilities for obtaining the best results. Next to laboratory-feeding is milk modified at home by the percentage method. For the very poor in cities results depend less upon exact methods of modification than upon the kind of milk used and the intelligence with which it is fed.

The Principles of Percentage Milk Modification.—In modifying milk for healthy infants the secret of success is to begin with low percentages, especially of the proteids, and gradually increase according to the infant's digestion. To continue with very low proteids frequently leads to disturbances of nutrition, which are sometimes very serious.

During the early weeks the best guide to progress is not the weight, but the comfort of the child and the absence of all signs of disturbance of digestion.

In general, the most important indications for varying the percentages may be stated as follows: If the infant is not gaining in weight and has no special signs of indigestion, increase the proportions of all the ingredients; for habitual colic, diminish the proteids; for vomiting immediately after feeding, reduce the quantity; for the frequent regurgitation of sour masses of food, reduce the fat, and sometimes also the sugar; for obstinate constipation, increase both fat and proteids.

Difficult Cases of Feeding.—One should not ignore the results of previous experience with any infant; in most cases it is unwise to repeat what has once worked badly. One should endeavour to determine whether the trouble is chiefly with the fat, the sugar, or the proteids of the milk; also whether it is the stomach or intestines whose functions are most disturbed.

It should not be forgotten also that failure may be due to other causes than the food ordered—to ignorance or carelessness in preparing or administering it or to the surroundings.

In all protracted cases, change of diet is important; the more protracted the condition, the more radical should the change be. Not much is to be expected from fractional variations in the milk percentages, when those given are producing a great deal of disturbance.

Radical changes are often necessary in the manner of feeding as well as in the food; with reference to intervals between feedings and quantities at single feedings, one often succeeds best by trying the exact opposite of what has previously failed.

A careful regulation of the milk percentages is more often successful than any other method. Success will be proportionate to the accuracy of the diagnosis as to the cause of the symptoms, and to the degree of error in the previous prescriptions employed.

The trouble is most often with the milk proteids. With many infants a proper adjustment of the total proteids given is all that is necessary. Some are helped in digesting proteids by the addition of the citrate of soda. Whey modifications, however, are more often successful than either of the methods just mentioned, and may advantageously be continued for several months. They are particularly useful with young infants. Partially peptonized milk meets the needs of a certain number of infants better than anything else; but caution is necessary not to continue its use too long, and to see that proper percentages are furnished in the milk to be peptonized, especially that the proportion of fat be not too high.

Children who have especial trouble with the fat are often temporarily benefited by the use of formulas made from skimmed milk.

The substitution of cereal gruels for water as diluents for milk possesses a certain amount of value, and is more apt to be beneficial in cases with intestinal than in those with gastric symptoms. The dextrinization of the gruels does not appear to increase their value.

The use of much larger quantities of farinaceous food with malt in the form of Keller's "Malt Soup" is a resource of much value when infants have great difficulty in digesting both the fat and proteids of milk. Infants whose digestive organs can tolerate a large amount of carbohydrates are often very much benefited for a considerable time by their use in this form. But the malt soup should not be continued too long, and if gastric or intestinal catarrh is present with vomiting or diarrhœa, it should not be used at all.

Withholding all milk is often necessary in acute illness, but in cases of chronic indigestion it is done too frequently and often where a better treatment is to discover and give correct milk percentages.

Success in infant-feeding is largely a question of careful attention to details. Without these the proportion of failures by any method will be very large.

CHAPTER IV.

FEEDING AFTER THE FIRST YEAR.

HEALTHY INFANTS DURING THE SECOND YEAR.

THE physician should not relax his vigilance in the feeding of a child after the first year has passed. The ideas of the laity in regard to what a child can digest after it has outgrown an exclusive milk diet, are very erroneous. The majority of infants are given solid food too early and in too large quantities. Most of the attacks of indigestion during the second year are directly traceable to such gross dietetic errors. The diet of a healthy child during the second year should consist of milk, some farinaceous food, bread, a small amount of animal food—beef or mutton, beef juice, eggs—and fruit.

Milk should be the basis of the diet. The popular idea that there are many children who can not take milk is an erroneous one; the real trouble usually is that they will not take it because other food pleases the palate better, and they are allowed to have their own way in this as in other things. It is of the utmost importance that the transition from a purely fluid diet to one of solid food should be made very slowly, and that the habit of drinking milk should not be discontinued.

During the second year with average milk and average infants very little modification of the milk is required. The addition of milk sugar is unnecessary, since the child is now able to take a considerable part of its carbohydrates in the form of starch. If the milk is very rich, such as that from a Jersey herd, it should be diluted with at least one-fourth water. In hot weather a still greater dilution may be necessary. If the milk is poor in fat, and constipation is present, the use of only the upper two-thirds from each quart bottle will make the percentage of fat about right.

Weaning from the bottle.—This should always be begun by the thirteenth month; by the fifteenth month an infant should take all its milk from a cup, except possibly the 10 P. M. feeding, when the bottle may be allowed for the sake of convenience. Early weaning from the bottle is a matter of no small importance. Where the bottle is continued, as it often is, until a child is two or three years old, the greatest difficulty may be experienced in getting rid of it, and this difficulty is increased the longer it is delayed. I have seen many children with the "bottle-habit" so developed that throughout childhood, although at any time they would take milk from the bottle, they could never be induced to take it any other way.

From Twelve to Fourteen Months.—The daily schedule at this period should be about as follows:

6.30 A. M. Milk, six to seven ounces; diluted with barley or oat gruel, two to three ounces.
9 A. M. Orange juice, one to two ounces.
10 A. M. Milk, two parts; oatmeal or barley gruel, one part; from ten to twelve ounces in all may be allowed.
2 P. M. Beef juice, one to two ounces;
or, the white of one egg, slightly cooked; later, the entire egg;
or, mutton or chicken broth, four to six ounces.
Milk and gruel in proportions above given, four to six ounces.
6 P. M. Same as at 10 A. M.
10 P. M. Same as at 6.30 A. M.

In preparing the food, the milk and the gruel are simply mixed together while the latter is warm, and salt and a very small quantity of cane sugar added to make it palatable. It is then divided into as many feedings as are required for the day, each one being placed in a separate bottle. As to handling the bottles and pasteurizing or sterilizing, the same rules apply as during the first year.

From Fourteen to Eighteen Months.—The diet may be increased by the addition of more solid food. The average child will take:

6.30 A. M. Milk, warmed, eight to ten ounces.
9 A. M. Fruit juice, one to three ounces.
10 A. M. Cereal: one, later two or three, tablespoonfuls of oatmeal, hominy or wheaten grits, cooked for at least three hours; for the first month this should be strained; upon the cereal from one to two ounces of thin cream, or milk and cream, with plenty of salt, but without sugar.
Crisp dry toast, one piece; or, unsweetened zwieback;
or, one Huntley and Palmer breakfast biscuit.
Milk, warmed, six to eight ounces.
2 P. M. Beef juice, one to two ounces; and one egg (soft boiled, poached or coddled); and boiled rice, one tablespoonful, cooked four hours;
or, broth (mutton or chicken), four ounces; one or two breakfast biscuits, or zwieback; and (if most of the teeth are present) rare scraped meat, at first one teaspoonful, gradually increasing to one tablespoonful.
6 P. M. Cereal: two tablespoonfuls of farina, cream of wheat, or arrowroot, cooked for at least one half hour, with milk, plenty of salt, but without sugar;
or, bread and milk or milk toast.
Milk, warmed, eight to ten ounces.
10 P. M. Milk, warmed, eight to ten ounces, which may be given from a bottle.

From Eighteen Months to Two Years.—The amount of solid food may be somewhat increased. The number of the meals should be the same as for the preceding period. In addition, cooked fruits, such as the pulp of stewed prunes or baked apple, strained, may be given at the midday meal. It is generally best not to give fruits and milk at the same meal. Nothing but water should be given between meals. Potato and other vegetables are best deferred until the child has passed two years.

DIFFICULT CASES DURING THE SECOND YEAR.

The number of children whose nutrition is a matter of difficulty during the second year is much smaller than during the first year; yet the difficulties may be just as great. Some of these are infants that have been very delicate from birth, and carried through the first year only by the greatest effort. Others are healthy at birth, but their digestion has been badly deranged in consequence of improper feeding. Still others did well until they were weaned. The conditions may be the result of a severe attack of acute disease of the stomach or intestines during the first year. Other important causes are the early use of solid food and the too exclusive use of farinaceous foods of all varieties.

Whatever the special cause of the condition, cases of chronic indigestion in the second year are usually improved by putting them back upon essentially a first-year diet. Usually the first thing to be done is to stop all solid food except possibly rare scraped meat. Starches must be reduced to a minimum or prohibited altogether. In most cases milk, meat, and a little suitable fruit should constitute the diet. While it is undoubtedly true that the use of plain cow's milk often fails entirely, it is certain that nothing is more likely to succeed than cow's milk when properly modified. This must be continued as the principal diet, sometimes as the sole diet, for the greater part of the second year. The milk should be modified as for healthy infants who are from eight to twelve months younger than the patient under treatment. The daily quantity should generally be somewhat larger than for a young, healthy infant taking food of the same strength. The regular intervals of feeding should never be shorter than three hours, and in many cases intervals of four hours are to be preferred.

Striking improvement often follows the administration of rare meat-pulp, especially to those who are over eighteen months old. From one to two ounces may be given daily. Generally the proteids in the food have been previously deficient. Many of these children digest meat when given in this way better than they do the casein of the milk. Raw beef juice and the whites of eggs, partially cooked, may also be given.

The same fruits should be allowed as for healthy infants, the quantity being smaller. As it is with the starches that the greatest difficulty is experienced, the carbohydrates should be administered chiefly in the form of milk sugar or some of the malted foods. When starch is first allowed it should be given with some reliable preparation of diastase.

When the child is once well started and gaining steadily, the food may be gradually modified, until the diet recommended for healthy infants of the same age is reached. All changes must be made very gradually, and it should never be forgotten that there is a constant disposition on the part of all mothers and nurses to over-feed these children.

FEEDING FROM THE THIRD TO THE SIXTH YEAR.

Articles allowed.—From the following list the diet of a healthy child may be arranged:

Milk.—This should be the basis of the diet; most children require about one quart daily. This usually needs no modification, but if somewhat difficult of digestion, it should be prepared as follows: Six ounces of milk, one ounce of cream, and three ounces of water. The milk should usually be given warm.

Cream.—This is of great value, especially when there is a tendency to constipation. From two to eight ounces may be given daily. It may be used upon cereals, upon potato, in broths, and mixed with milk. In many cases it is advisable to withhold milk and give only cream.

Eggs.—These are a valuable form of proteid. They should be fresh, soft-boiled or poached, but never fried. Usually eggs should not be given oftener than every other day, as many children soon tire of them.

Meats.—Some form of meat should be given once a day. The best are beef-steak, mutton chop, and roast beef or lamb; next to these the white meat of chicken, or fresh fish, which should be boiled or broiled. Beef and mutton should be given rare.

Vegetables.—Potato may be given once a day, preferably baked, with the addition of cream or beef juice rather than butter. Of the green vegetables the best are asparagus tops, spinach, stewed celery, string beans, and fresh peas. One of these vegetables should be given daily—always well cooked and mashed.

Cereals.—Nearly all these may be used—oatmeal, wheaten grits, hominy, rice, farina, and arrowroot. The most important part of the preparation is thorough cooking. If the grains are used, cereals should be cooked at least three hours, after having been previously soaked for several hours. They should always be well salted, and given with milk or cream, but with little or no sugar.

Broths and soups.—The meat broths are preferable to the vegetable broths. Nearly all varieties may be given. Plain broths are not very nutritious, but when thickened with arrowroot or cornstarch, and when cream or milk is added, they are very palatable, and at the same time a valuable addition to the diet. Beef juice may be used as directed for the second year.

Bread and biscuits (crackers).—In some form these may be given with nearly every meal, better without butter until the fourth year, as for young children cream is a better form of fat. All varieties of bread may be allowed when stale; also dried bread, zwieback, and oatmeal, Graham, or gluten biscuits.

Desserts.—The only ones that should be allowed up to the sixth year are junket, plain custard, rice pudding without raisins, and, not oftener than once a week, ice-cream. Of the last three, the quantity given should be very moderate.

Fruits.—Some fruit should be given every day. Oranges, baked apple, and stewed prunes are the most to be depended upon. Raw apples should not in most cases be given. Peaches, pears, and grapes (with seeds removed) may be given when thoroughly ripe and fresh, but only in moderate quantity. Special care should be exercised in the use of fruits in very hot weather, and in cities where they may not always be fresh. The juice of fresh berries may be given in the second year; but the whole fruit should be very sparingly given to all young children, and always without cream.

Articles forbidden.—The following articles should not be allowed children under four years of age, and with few exceptions they may be withheld with advantage up to the seventh year:

Meats.—Ham, sausage, pork in all forms, salt fish, corned beef, dried beef, goose, duck, game, kidney, liver and bacon, meat stews, and dressings from roasted meats.

Vegetables.—Fried vegetables of all varieties, cabbage, potatoes (except when boiled or roasted), raw or fried onions, raw celery, radishes, lettuce, cucumbers, tomatoes (raw or cooked), beets, egg-plant, and green corn.

Bread and cake.—All hot bread and rolls; buckwheat and all other griddle cakes; all sweet cakes, particularly those containing dried fruits and those heavily frosted.

Desserts.—All nuts, candies, pies, tarts, and pastry of every description; also all salads, jellies, syrups, and preserves.

Drinks.—Tea, coffee, wine, beer, and cider.

Fruits.—All dried, canned, and preserved fruits; bananas; all fruits out of season and stale fruits, particularly in summer.

From the third to the sixth year four meals should usually be given daily and at regular intervals—e. g., 7 and 10.30 A. M.; 1.30 and 6 P. M. The second meal should, in most cases, be smaller than the others.

The following is a sample diet for a child of four years:

First meal.—Half an orange, two tablespoonfuls of some cereal well salted, with two or three tablespoonfuls of cream, a glass of milk, one piece of bread with a little butter.

Second meal.—A glass of milk or cup of broth with bread or two or three biscuits (crackers).

Third meal.—Two tablespoonfuls of finely divided steak or chop, one tablespoonful of baked potato, one tablespoonful of spinach, bread and butter, a cup of junket, water to drink.

Fourth meal.—Milk with bread, or milk toast.

From the list of articles given above, a sufficient variety in the diet can be secured. The only way for the physician to be sure that proper food is given to young children, is to write out for the guidance of the mother or nurse two lists somewhat similar to the above, of articles forbidden and articles allowed. This plan I have followed for several years with the happiest results. It is rarely safe to trust to the judgment of the mother.

There are a few simple rules in feeding which should always be followed:

A child should be taught to eat slowly and thoroughly masticate his food. The food must always be very finely divided, for, as a rule, mastication is very imperfect even up to the sixth or seventh year. If the child is fed by the nurse, plenty of time should be taken for the meal. It is almost always the case that the food is given too rapidly. It is unwise continually to urge children to eat when they are disinclined to do so at the regular hours of meals, or when the appetite is habitually poor, and under no circumstances should children be forced to eat. Indigestible articles of food should not be given to tempt the appetite when ordinary simple food is refused, nor should these be allowed because of the notion that " the child must eat something." Food should not be allowed between meals when it is habitually declined at meal-time. If a child refuses to eat, and examination reveals no fault with the food prepared, it should seldom be offered again until the next feeding time. In all cases of temporary indisposition, no matter of what nature, and during periods of excessive heat in summer, the amount of solid food should be reduced and more water given. If milk is the food, it should be diluted.

FEEDING DURING ACUTE ILLNESS.

Infants.—This is an important part of the treatment of every acute disease in childhood, but especially so in infancy. Whether the illness is one of the eruptive fevers, diphtheria, pneumonia, or influenza, all cases must be fed in about the same way. It is much easier by proper feeding to prevent disturbances of digestion, than to allay them. In infancy this complication often turns the scale against the patient. In every severe acute illness, especially if it is of a febrile character, the power of digestion is much diminished. One evidence of this is the onset with vomiting; another is the anorexia which accompanies the early stage of nearly all acute diseases. We should respect this disinclination and make it our guide in the treatment. But water is needed; withholding this will often cause the temperature to rise even higher than before.

In all acute febrile diseases the general rule should be, less food and more water than in health.* For bottle-fed infants this is easily accomplished by simply increasing the dilution of the food; for nursing infants by making the nursing time shorter and giving water freely between feedings either from a spoon or bottle.

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* Some valuable suggestions as to the character of food most suitable in acute disease may be obtained from the experiments of Jacubowitch (Jahrbuch für Kinder-

Regularity in feeding is too often entirely ignored. While it is true that with some capricious children all rules must be disregarded, it is with the great majority a decided advantage to adhere to proper food and regular intervals. Food should seldom be given at less than two-hour intervals, and generally a three-hour interval is better, although there is no limit to the frequency with which water may be given, and unless the stomach is irritable, almost no limit as to quantity. Stimulants, when required, are often best given in a very dilute form with the water.

Forced feeding—gavage.—Not a few cases, however, are seen in which, after a child has been several days sick, in consequence of delirium, stupor, sepsis, or some other serious condition, it may refuse all food or take so little that it is in danger of death from inanition. At this juncture forced feeding or gavage (see page 64) serves an excellent purpose. Both food and stimulants can thus be introduced at regular intervals with slight disturbance, and lives saved which would otherwise be lost. If gavage is employed, the stomach should be first washed. The intervals of feeding should be made at least one hour longer than is customary in health, and usually predigested foods given.

Older Children.—The same conditions with reference to digestion exist as in the case of infants. Older patients, however, are not so easily disturbed, and the disturbance of digestion is not so likely to be serious as in the case of infants. Even here the physician should direct the food to be given at regular intervals, usually not oftener than every three hours, but should never—as is so often done—order milk to be given to the child every time it asks for a drink. In most cases, for children under five years old, milk should be somewhat diluted, usually with lime-water, and partially peptonized if the child's digestion is feeble. Children who do not take milk readily may be given beef tea, broth, gruel, or kumyss, but rarely ice-cream or jellies so frequently prescribed, as these, if given in any considerable quantity or very often, are likely to disturb the stomach and take away what little desire for food the

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heilkunde, xlvii, 195) upon the activity of the digestive ferments derived from the different organs of children, removed immediately after death, usually occurring from acute general disease. The greatest activity was found in the diastatic ferment of the pancreas, although its power to emulsify fats was weak, and in one-third the cases it was absent. The peptonizing power both of the stomach and the pancreas was very weak. The practical inference from this is that the food of acutely sick children should consist chiefly of carbohydrates, either as sugars or starches, that fats should be very sparingly given, and that proteids in many cases should be partially predigested. This accords with clinical experience.

child may have. Raw eggs are palatable when beaten up with sherry, a little sugar, and cracked ice. Fruits, particularly oranges, grapes, and grape-fruit, may be allowed in almost every febrile disease, but never given within two hours of a milk feeding.

The water given may be plain boiled water, but better, in most cases, are some of the carbonated waters, Vichy, Seltzer, or Apollinaris, these being less likely to disturb the stomach.

It is certainly a mistake to force food upon older children in any disease in which their condition is not dangerous. But when there is sepsis, delirium, or coma associated with other dangerous symptoms, gavage may be resorted to with but little more difficulty, and with no less satisfactory results, than in infants.

CHAPTER V.

THE DERANGEMENTS OF NUTRITION.

THE derangements of nutrition form a distinct and a very large class in the ailments of infancy, particularly during the first year. The symptoms are sufficiently definite and characteristic for them to be regarded as separate diseases, and to be discussed as such. In adults such symptoms are seldom seen except in connection with organic disease. These cases are often very puzzling, and in a large number of them a diagnosis of some constitutional disease, such as hereditary syphilis, or tuberculosis, or organic disease of the stomach or intestines, is erroneously made. At other times the symptoms resemble those of acute toxæmia. The essential condition in all these cases is the inability of the infant to get from its food what its system needs. It can not digest or assimilate enough to support life. It is unable to replace from its food the daily waste of its tissues. The constructive metabolism is not equal to the destructive metabolism of the body; the process is, therefore, essentially one of starvation, which may be rapid or slow, according to circumstances.

The fault in these cases is partly with the digestion, but principally with the food. The problem is, to adapt the food to the digestion of the individual child under consideration. The solution is often very easy at first, but the difficulties multiply rapidly the longer the condition has lasted. It is therefore essential that the true explanation of the symptoms should be recognised at the earliest possible moment. Changes occur so rapidly in very young infants that a mistake in diagnosis and a consequent delay of a few days, may be sufficient to determine a fatal result. The outcome in cases of imperfect nutrition depends almost entirely upon their management. The condition is not one which tends to right itself. Spontaneous improvement or recovery rarely takes place. In order to recognise the condition and anticipate the result, nothing is so important as a close observation of the body-weight. A child whose nutrition is a matter of difficulty should be weighed regularly, in the early months twice a week, and once a week throughout the first year. If this is done, the first symptoms of failing nutrition are unerringly detected. If a child does not gain in weight something is wrong, and a steady loss in weight in an infant is a warning which should never be unheeded; for, unless the conditions are changed, it is practically certain to continue, and generally with increasing rapidity, until the infant's vitality has been reduced to such a point that no means of treatment can restore it. The younger the child, the more rapid the loss, and the longer it has continued, the greater is the danger.

For convenience of description these derangements of nutrition have been divided into three groups, differing, however, rather in degree than in kind.

1. Cases of acute inanition, which are quite rapid, generally lasting from a few days to a few weeks. They are rare except in young infants, being most frequently seen in the first three months.

2. Cases of malnutrition, in which the symptoms are much less severe than in the other groups, although they may be of long duration. While it is most common in the first two years, malnutrition may be seen at any age.

3. Cases of marasmus. This is similar to inanition, but a much slower process, lasting usually for several months. It may be seen in infants of any age.

ACUTE INANITION.

Inanition, or starvation, is a condition depending upon lack of assimilation. It is common in early infancy, when it often simulates serious organic disease. In older children it is not so frequent, and not usually so obscure. In all the acute diseases of the digestive tract many of the symptoms are due to inanition. The cases considered in the present chapter, however, are those in which there is no such association, or where the digestive symptoms, strictly speaking, are not prominent.

Etiology.—The essential cause of inanition is that the child does not get sufficient food, or that the food taken is not assimilated. It usually develops under one of the following conditions: (1) When a child refuses all food, whether from the breast or the bottle, or can be made to take only an insignificant amount. The cause of this it is often impossible to discover. I have seen it in a variety of circumstances, once in an infant five months old, previously healthy, who was suffering from whooping-cough. This infant utterly refused the breast, and from the spoon would take less than two ounces a day. After four days and the production of most alarming symptoms, gavage was begun, and its life, I think, saved by it. It is sometimes seen at weaning, where a child persistently refuses to take food from a bottle or spoon. (2) When the food given is entirely inadequate, as when an infant is nursing upon a dry breast, or one in which the milk supply is so scanty that the child gets practically nothing. I have occasionally seen it later, when the breast-milk, for some unexplained reason, had suddenly failed. (3) Where the character of the food is improper. Breast-milk may be not only scanty, but of very poor quality. On account of extreme poverty, the infant may be getting only tea, as I have known to be true in several cases before admission to the hospital. Some cases occur in young infants who are fed entirely on starchy food. (4) Where the infant at birth has such feeble powers of digestion, because premature or delicate, that it is unable to take or to digest sufficient food to maintain life. Sometimes this food is breast-milk, which, though abundant, is of inferior quality and can not be assimilated. Very often it is some proprietary food. (5) When a sudden change of food is made to one so difficult of digestion that the child is unable to assimilate it. This may happen after sudden weaning. In such cases the symptoms of inanition are mingled with those of acute indigestion, but the former usually predominate.

In children over one year old, and sometimes in younger ones also, the symptoms of inanition may follow those of some acute disease, such as influenza, malaria, pneumonia, or even otitis. Although the child may recover from the acute process, the general vitality is so much lowered that assimilation is not sufficient to replace the waste of the body.

Symptoms.—The mode of development depends upon the antecedent condition. In young infants inanition often follows malnutrition where perhaps there has been nothing noticeable except a gradual loss in weight; or if the weight has not been watched, it may be observed only that the infant has not been doing well. Severe symptoms may come on quite suddenly, and if the nature and the gravity of the condition are not appreciated the case may terminate fatally in two or three days. The loss in weight is now rapid, amounting often to three or four ounces a day. The temperature in the newly born may be high, but it is more often subnormal. The pulse is always weak and rapid. The urine is scanty and very low in chlorides. The extremities are cold, and the peripheral circulation poor. There is usually complete muscular relaxation, almost collapse. The skin may be dry or covered with a clammy perspiration. There is extreme pallor, and often there is cyanosis. This is always a grave symptom, and when it is marked the case usually ends fatally. Cyanosis may be present in children who have previously cried well and in whom there is no suspicion of atelectasis. The respirations are rapid and may be irregular. There may be constant worrying and fretfulness, or a condition of semi-stupor, in which the child makes no sign of wanting food. The fontanel is sunken and the pupils are often contracted. The stools contain undigested food, or if predigested foods are given they seem to pass through the intestines unchanged. The bowels usually move frequently, although there may be constipation, due to the small amount of food taken. When all food is refused for two or three days the stools may resemble meconium, as I once saw in a child six months old. While no desire for food is manifested, infants will sometimes swallow food when it is offered, retaining everything given for several feedings, when the whole quantity is vomited.

The course of the disease depends much upon the age of the infants. Those under one month succumb most quickly. In them the symptoms sometimes last but two or three days, seldom more than a week or ten days, the children simply drooping steadily until death occurs. With proper treatment complete recovery may take place in a week. In older infants the progress, whether upward or downward, is usually less rapid.

Prognosis.—The outcome of these cases is always uncertain. In few conditions is it more so. It is hard for one who is not familiar with the condition to appreciate the great and even the immediate danger in which a young infant may be from inanition, especially in the absence of both vomiting and diarrhœa. It is difficult to estimate the gravity of an individual case except after twenty-four hours' observation. The best of all guides is perhaps the weight. Where the loss is several ounces each day the chances of recovery are small. The presence also of frequent vomiting or of diarrhœa makes the outlook very bad. A high temperature, very marked relaxation, copious perspiration, cold extremities, and cyanosis are all bad symptoms.

Diagnosis.—Inanition is distinguished from malnutrition by its greater severity, and from marasmus by its more acute character. The usual mistake is that of confounding inanition with some local or constitutional disease. It may be mistaken for acute indigestion, meningitis, gastro-enteritis, pneumonia, and some of the fevers. The temperature when elevated is especially likely to mislead. In some cases the absence of chlorides from the urine may be of diagnostic value.

Treatment.—The existence of inanition in young infants presupposes only the feeblest powers of digestion and assimilation. If possible, a good wet-nurse should be secured, for in most of the cases the time for action is so short that there is no opportunity to experiment with artificial feeding.

The breast-milk should usually be diluted, at first with an equal volume of water or lime-water, and the quantity should be only a few drachms. It may be given with a spoon or a medicine-dropper. If there is diarrhœa, the milk should be pumped from the breasts, and the cream removed, since the high fat of good breast-milk is apt to excite vomiting or copious purgation. Gradually the quantity and strength of the milk are increased until the child is allowed to take the breast entirely.

When no wet-nurse can be obtained, whey mixtures (page 210) may be tried or a milk formula containing low proportions of fat and proteids, such as No. II, Second Series (page 195), or No. I, Third Series (page 196). Sometimes these should be peptonized. When food is not readily taken, it may be given by gavage. Rectal feeding may be of some assistance for a short period. Other things which may be tried are diluted kumyss, animal broths, malted foods, farinaceous foods, and beef peptones.

Often the symptoms are due quite as much to a lack of water as to a lack of food. Injections of a normal salt solution may be given per rectum or even under the skin with very great advantage. Rectal injections should be given at 104° to 110° F. and carried high into the colon by a catheter; they should be repeated every four or five hours.

The other treatment required by these cases is the reduction of high temperatures by sponging or tepid baths, and the raising of subnormal temperatures by hot-water bags, rolling in cotton, or even by the use of an incubator. Stimulants are indicated, but are not very well borne; alcoholic preparations by the mouth often excite vomiting, but by the rectum they may be better tolerated. Drugs are of no use whatever. Oxygen inhalations are of the greatest value, and should be used if possible in all very acute cases whether cyanosis is present or not. Heat, oxygen, and diet are really the sum of treatment.

Inanition in older infants is usually seen at weaning or in connection with or following some acute illness. Completely peptonized milk by gavage is often useful. There are some patients, usually over ten months old, who refuse fluid food of every description, and vomit it when it is coaxed or forced, yet who will take and digest in a most surprising manner some form of solid food, such as beef-steak, oatmeal, bread, crackers, or even potatoes. For the time one must give whatever the child will take, and gradually change to a suitable diet as soon as circumstances will permit. The needed water may be given per rectum.

All children who have suffered from acute inanition need the closest attention for a long time, particularly as to their feeding, regarding which suggestions will be found in the pages devoted to Infant-Feeding.

MALNUTRITION.

Cases of malnutrition are exceedingly common, and occupy a large part of the time and attention of one engaged in practice among children. Although these children can not be said to be actually ill, they are very far from well, and their condition is often the cause of the greatest solicitude on the part of anxious parents, not only from the existing state of health, but from the apprehension of the development of some serious organic or constitutional disease, especially tuberculosis.

Etiology.—Malnutrition may depend upon inherited conditions. Certain children are delicate from birth, possessing only feeble physical vitality, though without giving evidence of any actual disease. They are often the offspring of parents of delicate constitution, or of those with inherited tuberculosis, gout, syphilis, or alcoholism. Very many city children are included in this group. They are a product of modern life, and inherit a too highly developed nervous organization with a corresponding amount of physical deterioration. In another group of cases the children are premature or very small at birth, weighing perhaps only three or four pounds. Many cases are traceable to improper feeding or equally poor nursing during the first few months. These children get a poor start in life, and on that account are handicapped throughout infancy. In many cases malnutrition develops as a result of the patient's surroundings. While this is common among the poor, it is not rare among the better classes. One of the most frequent causes is the pernicious custom of keeping infants in close apartments where the thermometer ranges from 72° to 78° F., and where the greatest anxiety is constantly felt lest the children take cold. Such infants may lose in weight, become anæmic, and exhibit all the signs of malnutrition where nothing else is wrong except the conditions mentioned. In infants, malnutrition often depends upon some previous acute disease, especially of the stomach and intestines, and sometimes of the lungs.

In children who are over two years old the condition of malnutrition may be due to any of the factors above mentioned—inherited feebleness of constitution, bad feeding and its resulting indigestion, too little fresh air, and close confinement indoors. It is, however, at this period much more frequently than in infancy, dependent upon some previous acute disease. This may be acute broncho-pneumonia, acute ileo-colitis, influenza, malaria, or any of the eruptive fevers. As a result, an impression is left upon the child's constitution which lasts for months, often for years, and which manifests itself not by any special local symptoms, but by a general condition of debility or malnutrition. Sometimes such diseases, instead of being directly the cause of the symptoms, are the occasion which brings out some latent inherited taint or constitutional weakness in children who up to this time, perhaps, have appeared exceptionally healthy. In other cases malnutrition depends upon faulty methods in education, especially upon overpressure in schools.

Symptoms.—*In infants.*—The weight is much below the average, and is either stationary or the gain is very slow, often only five or six ounces a month at a period when it should be from one to two pounds. In a case recently under treatment, a child at fourteen months weighed but eight and a half pounds. This infant at birth weighed three and a half pounds, but in a few weeks the weight dropped to two pounds.

Not only the weight but the general physical development is much below the normal. At one year the body length may be three or four inches less than the average. Dentition is usually but not always delayed. Muscular development, too, is backward; many of these children do not sit alone until a year old, and barely walk at two and a half years. The muscles are soft and flabby, and the ligaments so weak that paralysis is often suspected. The body is so small that the head seems unnaturally large, and a diagnosis of incipient hydrocephalus is frequently made. Mentally these infants are often above the average. Some symptoms of rickets may be present, but often there are none; to apply the term rachitic to all of them seems to me a mistake.

Anæmia is invariably present, and varies much in degree, being rarely extreme. The circulation is usually poor, the hands and feet are frequently cold. In many children the skin is unnaturally dry; in others there is a disposition to excessive perspiration, particularly about the head. Nervous symptoms are usually present. These children are restless, fretful, and irritable; they sleep badly during the day, and often worse at night. Enlargement of the lymph glands is common, especially those of the neck. The cervical adenitis may have started from a slight catarrhal cold, but the glands continue to swell after this has subsided and may remain enlarged for months.

One of the most characteristic things about these infants is their feeble powers of digestion and assimilation. Unremitting care and constant watchfulness are required to keep them up to even a moderate standard of health. The most trivial changes in food may upset them. Attacks of acute indigestion are usually brought on by overfeeding—the mistake which is almost invariably made by mothers who are discouraged with the slow progress made, and are anxious to make their children grow fat and strong. The balance is so delicately adjusted that the slightest deviation from proper rules of feeding, either as to the quality of the food or the quantity given, is immediately followed by an attack of acute indigestion, often by severe diarrhœa. As a result, the child may lose as much in two or three days as it has gained in a month or more. These acute attacks, if in summer, not infrequently prove fatal. Not only do these patients have but little resistance to acute disturbances of the stomach and intestines, but any acute disease is serious—measles, whooping-cough, and pneumonia being especially fatal.

Among the poor or in institutions, cases of malnutrition like those described, if in children under nine months old, are almost certain to go on from bad to worse until they have reached the condition described as marasmus. Between this and malnutrition no sharp distinction can be drawn; they are rather different degrees of the same general process. In private practice, where it is possible to have the best care and surroundings, with the co-operation of an intelligent mother or nurse, a very large number of these infants can be reared. After the second year has passed the problem becomes a much simpler one, and if infectious diseases and other forms of acute illness can be avoided, the probabilities are in favour of the child's becoming stronger each year and growing to maturity.

In older children.—In general appearance these children are thin, pale, and very often undersized, particularly if the condition is constitutional or hereditary. Sometimes they are taller than the average for their age, and their symptoms are often attributed to too rapid growth. One of the most striking things about children suffering from malnutrition is their vulnerability. They "take" everything. Catarrhal processes in the nose, pharynx, and bronchi are readily excited, and, once begun, tend to run a protracted course. There is but little resistance to any acute infectious disease which the child may contract. One illness often follows another, so that these children are frequently sick for almost an entire season. Their muscular development is poor, they tire readily, are able to take but little exercise, and their circulation is sluggish. Mentally they are usually bright, often precocious. Many would be called nervous children. They are cross, fretful, and any unusual excitement produces an effect which lasts for some time; for example, after a children's party or a Christmas tree they may lie awake half the succeeding night, and may be really ill for two or three days. Their sleep is usually disturbed and restless; they waken frequently, and occasionally suffer from night-terrors. At a later age they are favourable subjects for chorea, neuralgia, and all functional nervous disorders.

Digestive symptoms, if not constant, are very easily excited. In fact, they do not suffer so much from chronic indigestion as from a delicate or feeble digestion, which is easily upset by the slightest deviation from the regular routine. Children of five or six years have to be fed as carefully as infants of eighteen months or two years. The appetite is usually poor, and mothers are distressed because their children eat so little, yet, when food is urged upon them, attacks of indigestion follow with singular uniformity. The tongue is slightly coated the greater part of the time. The bowels are apt to be constipated, apparently more from lack of muscular tone than from anything else. From time to time, from slight causes, such as exposure to cold, or even from fatigue, there may be large quantities of mucus in the stools for two or three days at a time, although this is not a prominent feature of most of these cases. When they are not fed with the greatest care these children suffer constantly from indigestion. A moderate amount of anæmia is always present, and this may be the most striking feature. In very many children with a marked disturbance of nutrition, there is an excessive elimination of uric acid.

The duration of the condition depends very much upon the cause. If the cause is constitutional or inherited, the condition may last throughout childhood. Where it follows some acute illness it commonly lasts for a few months only; but the effect of an acute attack of bronchopneumonia or of ileo-colitis may last for years. If the malnutrition is the result only of the child's surroundings, like the confinement incident to city life, very rapid improvement may follow a removal to the country. In some children marked improvement is seen about the seventh year; in others, a great change comes at puberty.

Diagnosis.—The physician should not be too ready to make a diagnosis of simple malnutrition. Before accepting such a diagnosis, he should examine the child with the greatest care, to exclude the common organic and constitutional diseases. Much regarding inherited constitutional tendencies can be learned from the family history and from the condition of other children in the family. In the first place, tuberculosis, syphilis, and rickets should be excluded; then chronic malaria and the diseases of the blood; and, finally, organic diseases of the lungs, heart, stomach, intestines, liver, and kidneys. Even malignant disease, though rare, should not be overlooked. It may take careful observation for several days, and sometimes for weeks, with repeated physical examinations, before all these conditions can be positively excluded.

The next step in the diagnosis is to discover upon which one of the many possible causes, malnutrition depends. In private practice the great proportion of cases are due to improper feeding or nursing; next in importance are improper surroundings; and last come inherited constitutional conditions. In other words, most of these children are born healthy, but become ill or delicate in consequence of improper management.

In older children, after excluding constitutional and local diseases, the whole life of the child must be investigated to discover the fundamental condition which is at fault. A carefully obtained history from infancy is of the greatest assistance. It is often difficult, and sometimes impossible, to get at the primary factor, for in cases of long standing there may be symptoms connected with almost every function of the body. One should scrutinize closely the quality and quantity of food given, the amount of sleep, the hours of study and recreation, the amount of exercise in the open air, and the physical conditions surrounding the child. Usually the most important factor in the case can be discovered.

Prognosis.—This depends much upon the cause of the condition; if it is one that can be removed, the prognosis is good not only for improvement but for complete recovery. The longer the condition has lasted and the greater the general disturbance the slower will be the improvement. The great danger is the supervention of some acute disease while the child's resistance is so greatly reduced. Acute indigestion, gastro-enteritis, and broncho-pneumonia are especially to be dreaded.

Since everything depends upon the fidelity with which directions as to diet and general management are carried out, the cases which present the greatest difficulties are those in which these conditions are hardest to control. When a child is not only suffering from malnutrition, but has been indulged and spoiled in every way by anxious but unwise parents, no success is to be expected unless the child can be placed in the hands of an experienced and trustworthy nurse. Cases due to improper feeding or to bad surroundings usually improve when these are corrected, and the worse these conditions have previously been the greater the improvement to be expected. Those depending upon an inherited, delicate constitution are not so hopeful, and require the closest attention throughout childhood.

Treatment.—This is a problem of nutrition to be solved by diet and general management, drugs occupying a very small place.

In infants.—In very young infants treatment is chiefly a question of feeding. This should be carried on according to the rules given in the chapter upon Feeding in Difficult Cases (page 208). These children often do fairly well during the first year, but after this time frequently do very badly, on account of the failure to appreciate the fact that, although over twelve months old, in point of development they resemble healthy infants of four or five months, and are to be managed as such. If they are nursing, weaning should often be deferred until the sixteenth or eighteenth month, or at least partial nursing should be continued until that time. When cow's milk is begun it should always be very largely diluted, usually modified as for a healthy infant two or three months old. It is surprising to see with what uniformity the giving of cow's milk, pure or slightly diluted, will produce attacks of indigestion in some of these infants. I have seen a single feeding in which one ounce of milk was given, and that diluted three times, produce a violent attack of acute indigestion which proved well-nigh fatal. Feeding during the entire second year should be carried on very much as in ordinary healthy children from the sixth to the twelfth month. A deviation from this rule almost invariably results disastrously. One must be guided as to the amount and character of the food not so much by the child's age as by his digestive capacity, and in most cases this is much feebler than the mother or even the physician supposes. In many of these cases, cow's milk—for them the most valuable of all foods—has been excluded from the diet, when the only trouble is that it has not been given in sufficient dilution. For some children it must be partially peptonized during periods when digestion is especially feeble.

Next in importance to diet is fresh air. Often these patients will not improve with any variation in diet until fresh air is secured. Then increased digestive power is seen in the course of a few weeks, sometimes in a few days. The natural tendency of a mother who has a delicate infant, or one suffering from malnutrition, is to house it closely and never allow it a breath of fresh air. It is of the greatest assistance if these children can be sent to a warm climate for the winter. If this is not possible, fresh air may be obtained by changing apartments, or by an airing in the room with the windows open. In the beginning this should be done for a few minutes only, the time being gradually increased to two or three hours each day. The child should be clothed as for the street, and, if necessary, hot bottles should be placed at the feet.

Cold sponging is another valuable tonic. After the morning bath is given, at 95° F., the entire body should be sponged for a moment with water at a temperature of 60°, or even 55° F. This produces a certain amount of shock and causes loud crying, which is of itself beneficial. How frequently this should be done will depend upon the reaction following it. If the child remains blue and cold for some time afterward, the cold sponging should not be repeated. If there is a good reaction, it may be used daily.

Friction and massage are useful in many cases. The child should be laid upon the lap of the nurse, if possible before an open fire, and should always be covered with a blanket. The entire body should then be rubbed for ten or twenty minutes with the bare hand, or, better, with cocoa butter. Simple rubbing may be used, or the movements of massage employed. If the latter, they should be very gentle at first, and only for a short time. Professional operators are inclined to be too energetic for little children. There is no advantage in rubbing with cod-liver oil instead of cocoa butter, while the odour makes it decidedly objectionable.

The only tonics I have found of much value are alcohol, nux vomica, and cod-liver oil. Alcohol may be given in the form of port or sherry wine. Nux vomica may be given alone or with the wine. Cod-liver oil is too much used in these cases, and in too large doses. Many of these infants can not take it at all. It should rarely be given when the tongue is coated and the appetite very poor. The dose should always be small, e. g., ten drops of the pure oil three times a day, or twice as much of an emulsion. In these doses it may be given for a long time without disturbance.

The secret of success in treating cases of malnutrition is, to hold the patient to a regular routine in feeding, sleep, and in everything relating to his life. Experiments are nearly always unfortunate. The physician should lay down in writing for the guidance of the mother, specific rules with regard to the amount of food, the time at which it is to be given, the hours of bathing, sleep, and airing. He should see the patient at regular intervals and often enough to be sure that his orders are being enforced. Good results are obtained only by constant watchfulness, and although improvement may not be seen at once, it is in most cases sure to come if the mother will co-operate. In my own experience no class of patients have given me so much satisfaction as cases of malnutrition in infancy.

In older children.—The same general principles are to be applied to them as to infants. The diet is of the first importance. Only the simplest, plainest, and most easily digested articles of food should be given. Milk, beef, eggs, the lighter and more easily digested cereals, bread, and fruit should form the diet. All sweets, pastry, highly seasoned food, candy, nuts, tea, and coffee should be absolutely prohibited, and, in fact, all the articles mentioned as " forbidden " on page 223. When the appetite is poor and simple food not well taken, the child should not be allowed to take indigestible articles for the sake of eating something. Nothing should be given between meals, and regular hours of feeding must be followed. Usually I have found three meals a day, for children over three years old, better than the practice of giving more frequent feedings. But this is not always the case. Under no circumstances should children be coaxed, urged, or hired to eat; much less should they be forced to do so. There is a popular misapprehension in regard to the variety in diet which children need. Most cases do better when a very simple and fairly uniform diet is continued.

The general habits of children should be directed; there should be regular and early hours for retiring, freedom from undue excitement, and interest should be awakened in out-of-door amusements. A pony or dog will be found useful. Children should be kept as much as possible in the open air; usually they do much better if they can be in the country during the entire year. Only a limited amount of reading and study should be allowed; and if children are at school, care should be taken that overpressure is not the cause of the symptoms, particularly in an ambitious child. The cold sponging given in the morning, as described on page 57, is extremely beneficial to children who take cold readily. Massage is useful for the benefit which it affords to the chronic constipation which is so frequently a symptom of malnutrition.

Of the tonics, iron, arsenic, and cod-liver oil are required in most cases, and the amount and combination may be varied from time to time, with the season of the year and the condition of the child's digestion. In general, these children require early hours, a simple diet, a quiet, regular life, and very little medicine.

MARASMUS.

Synonyms: Athrepsia, infantile atrophy, simple wasting.

Wasting is a symptom of many conditions in infancy. It occurs in tuberculosis, in infantile syphilis, and also as a result of acute or chronic disease of the stomach and intestines. Cases of wasting dependent upon such causes are not included in this chapter.

Marasmus is the extreme form of malnutrition seen in infancy, occurring, so far as is known, without constitutional or local organic disease. It is a vice of nutrition only.

Etiology.—Marasmus is not very often seen in the country or in private practice; but it is frequent in dispensary practice in all large cities, and is especially common in institutions for young infants. In my own experience in four institutions, more than one half the deaths under one year were directly or indirectly from this cause. Marasmus is a very large factor in the immense infant mortality of large cities in summer. Although the cause of death is usually reported under some other name, the determining factor in the fatal result is the previous marantic condition of the patient. The primary cause may be a congenital weakness of constitution which may depend upon heredity. It is often seen in premature children and in the illegitimate offspring of girls of sixteen or eighteen. In the vast majority of cases, however, it depends upon two factors—the food and the surroundings. Among the poor who live in tenements, infants who are artificially fed almost invariably do badly. This is due to ignorance in regard to the proper methods of infant-feeding and inability to procure what the child requires, especially pure cow's milk. A country infant may be neglected in many respects, and is often badly fed; but it has plenty of pure air, and usually thrives. In the city, as long as an infant has a plentiful supply of good breast-milk it continues to do well in most instances, in spite of the fact that its surroundings are bad. When there are not only bad feeding and unhealthful surroundings, but also an inherited constitutional vice, we have all the factors required to produce marasmus in its most marked form. The odds are so against the infant that its feeble spark of vitality flickers for a few months only and gradually goes out.

Another prominent factor in the production of marasmus is the overcrowding of infants in institutions. Even though artificially fed after the most approved methods, I have seen scores of infants who were plump and healthy on admission lose little by little, until at the end of three or four months they had become wasted to skeletons—hopeless cases of marasmus, dying of some mild acute illness, such as an attack of indigestion or bronchitis, the essential cause, however, being marasmus. The common mistake is that of placing too many children in one ward, with no chance of obtaining a proper amount of fresh air. No house-plant is more delicate or sensitive to its surroundings than an infant during the first few months of life.

Lesions.—The post-mortem findings in cases of marasmus are exceedingly unsatisfactory, and throw little if any light upon the disease. Every now and then general tuberculosis is discovered in patients dying apparently of marasmus, the existence of which was not previously suspected. An occasional lesion is fatty liver. This may lead to such enlargement of the organ that its weight is increased by one half. Both to the naked eye and under the microscope the usual changes of fatty infiltration are present, often to an extreme degree. In the past too much has doubtless been made of this condition of the liver in marasmus. From figures given elsewhere (see article on Fatty Liver), it will be observed that the lesion is not more frequent in this condition than in infants dying from other diseases. The most marked examples are seen in cases of marasmus which have lasted for seven or eight months. Its exact relation to the condition of wasting has not yet been determined.

With these exceptions the autopsies show nothing striking, and I have had the opportunity to make at least two hundred of them. The lesions usually found are the following: The brain is commonly anæmic, with dark fluid blood in the sinuses, marantic thrombi being rare. A strip of hypostatic pneumonia, from one to two inches wide, may be seen along the posterior border of both lungs, involving the lung to the depth of half an inch, or less. In the younger infants there are frequently areas of atelectasis in the lower lobes. The pleura is almost invariably normal. The heart is pale, with perhaps a slight increase in the pericardial fluid. The spleen and kidneys are pale, but otherwise normal. The stomach may be dilated; the mucous membrane is usually pale, often coated with tenacious mucus. The intestines contain undigested food, sometimes mucus. The solitary follicles of the colon and small intestine, and sometimes Peyer's patches, are slightly enlarged, the mucous membrane in other respects being normal. The mesenteric glands are often slightly enlarged. In addition to the above, there may be evidence of some recent infection, which has been the cause of death; there may be acute bronchitis, broncho-pneumonia, or intestinal catarrh.

The above lesions represent what has been found in the great majority of the cases, and very disappointing they are to one who sees them for the first time. Nor does the microscopical examination of the organs throw any light upon these cases. I have personally examined with care the stomach and intestines of more than a dozen cases, several of them in which autopsies were made only two or three hours after death, without finding anything of pathological importance. The theory advanced by certain German writers, that atrophy of the intestinal tubules is the explanation of marasmus, has found no support in my observations, nor in those of other American writers.

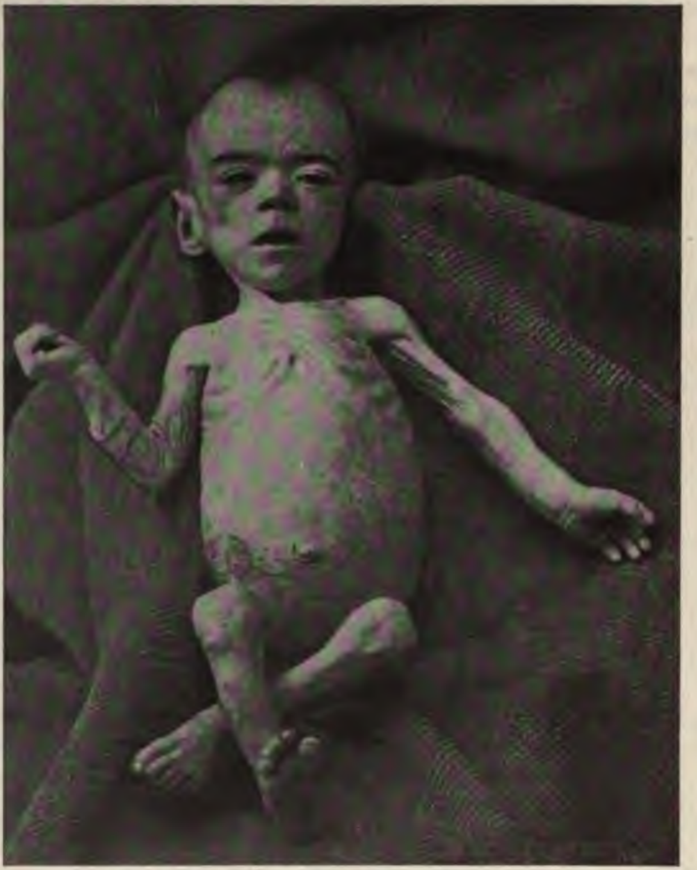

FIG. 41.—Marasmus; a patient in the Babies' Hospital, ten months old, weight six pounds. Weight at birth reported to have been nine pounds.

The true pathology of marasmus seems to me to be a failure of assimilation, owing to imperfect digestion, improper food, unhygienic surroundings, or feeble constitution. As a result, there is a progressive loss in weight, feeble circulation, imperfect lung expansion, imperfect oxidation of the blood, lowered body temperature, and, finally, a deterioration of the blood itself. Each of these effects becomes in turn a cause aggravating all the others, continuing until a condition is reached which is incompatible with life, for resistance becomes so feeble that the slightest functional disturbance proves fatal.

Symptoms.—The general history of these cases is strikingly uniform. The following is the story most frequently told at the hospital: "At birth the baby was plump and well nourished, and continued to thrive for a month or six weeks while the mother was nursing it; at the end of that period, circumstances made weaning necessary. From that time the child ceased to thrive. It began to lose weight and strength, at first slowly, then rapidly, in spite of the fact that every known form of infant-food was tried." As a last resort the child, wasted to a skeleton, is brought to the hospital.

The most constant symptom is a steady loss in weight. The general appearance of these patients is characteristic. They have an old look; the skin is wrinkled, has lost its tone, and hangs in folds upon the extremities (Fig. 41). The legs are like drumsticks; the abdomen is prominent; the temples are hollow; the fontanel is sunken; the eyes large; the features sharp; and the hands resemble bird-claws. Often the children are reduced literally to skin and bones. Anæmia is a very marked and almost a constant symptom, the amount of hæmoglobin being frequently reduced to 30 per cent, and in one of my cases to 18 per cent. Anæmic heart-murmurs are frequently heard. The body temperature is usually subnormal, unless artificial heat is used. A rectal temperature of 95° or 96° F. is very common, and one of 93° or 94° F. is occasionally seen. In addition to the pallor of the face, there may be a leaden hue due to congenital or acquired atelectasis. A frequent symptom is general œdema, depending upon the abnormal condition of the blood or blood-vessels. The first thing which calls attention to this is often an unexpected gain in weight. The œdema may increase until the cellular tissue of the whole body is affected. I have never, however, seen effusions into the large cavities. Œdema is usually associated with marked anæmia, and is generally a grave symptom. The stools are sometimes normal, but usually contain undigested food, and are large in proportion to the amount of food taken. No matter how carefully fed, these patients are easily upset. Now and then mucus is seen in the stools, but this is not a constant nor a marked feature. Vomiting is excited from the slightest cause, and often food is regurgitated almost as soon as swallowed. The appetite, in a severe case, is almost entirely lost; children refuse to take food from the bottle or spoon, and unless fed by gavage they die of inanition. In the earlier cases there may be an unnatural hunger, so that the children cry much of the time, and are relieved only when the bottle is given.

The complications are thrush, erythema of the buttocks, and bedsores, sometimes over the sacrum and heels, but most frequently upon the occiput. Occasionally there is seen a reflex spasm of the muscles of the neck, producing a marked opisthotonus, which may last for several days or weeks.

The course of the disease in most cases is steadily downward. It may be cut short at any time by acute disease. Frequently these infants die suddenly when apparently they have been as well as for several weeks. In many instances the autopsy reveals no explanation of the sudden death; but in other cases it may be due to the regurgitation of food, and its aspiration into the larynx, the patient being too weak to cough. Rarely, death occurs from convulsions. In summer, these children wilt with the first days of very hot weather, and die often in a few hours from a slight functional derangement of the stomach and bowels.

Diagnosis.—No sharp line can be drawn between marasmus and malnutrition. In the wasting which follows chronic disease of the stomach and intestines there is usually a history of an antecedent acute attack. The chief difficulty in the diagnosis of marasmus is to exclude tuberculosis. In some cases a differential diagnosis is impossible during life. Not infrequently tuberculosis is found at autopsy, even in infants of a few months, in whom there have been no symptoms except those of marasmus. Even when signs in the lungs are present, if situated posteriorly, they may be due either to tuberculosis or to the hypostatic pneumonia which is present. Signs in front are more significant; and consolidation anteriorly makes tuberculosis almost certain. In simple wasting there is often a history that the child was in splendid condition at birth, and continued so until it was weaned, from which date it had gone down steadily. In tuberculosis no such definite cause may be present; the children are often very delicate from birth. Simple wasting is so much more common that the chances are always in its favour.

Prognosis.—This depends on the age of the infant and the extent and duration of the disease. If the child is over eight months old, the chances of recovery are much better than in one under four months, for the fact that it has lived so long is generally evidence of pretty strong vitality. Very young infants are always difficult subjects to deal with. They go down more rapidly, and build up more slowly than those who are older. In most other circumstances the prognosis is much worse in cases of long duration. In a given case much depends upon whether everything possible can be done for the child: whether a wet-nurse can be secured or artificial feeding done in the best manner, and whether the patient can have the benefit of the best surroundings, in the country in summer and in winter a warm climate where it can be kept out of doors the greater part of the time. In institutions cases under four months old are usually hopeless. Of those over eight months quite a proportion can be saved by proper treatment, even though the body-weight is reduced to eight or nine pounds. When recovery occurs it may be complete, and the child at three years may be as vigorous as any child of its age. All these statements refer only to cases of simple marasmus. The presence of organic disease puts the case into another category.

Treatment.—The most important is that which relates to prophylaxis. This, for large cities, may be summed up in a single sentence: Give the poor the opportunity to obtain pure cow's milk and teach them how to feed it to young infants, and at the same time give ample opportunities for obtaining fresh air. In institutions the most important thing is to give adequate air-space for each child. Often only four or five hundred cubic feet are allowed, when one thousand are necessary, even with the best ventilation. Children should be changed from one apartment to another and opportunity given for thorough airing, and there should be perfect ventilation, not only in the daytime but at night.

As far as possible, wet-nurses should be obtained if the infants are under four months old. For these very young patients success by artificial feeding is generally impossible. With those of six months or over, good artificial feeding is very frequently successful. In modifying cow's milk for these cases the formulas most likely to agree are those with low fat, low proteids—partially peptonized in many cases—and relatively high sugar. Further suggestions will be found in the chapter on Feeding in Difficult Cases. In institutions we seldom succeed without wet-nurses.

For very young infants, with a temperature which is habitually subnormal, the incubator may be used. If this is impossible, children should be rubbed with oil, rolled in cotton, and surrounded with hot-water bags or bottles. The general management should be much the same as described in the chapter on Malnutrition. At least once every day—by means of spanking, mild flagellation, or, better, by the alternate use of the hot and cold baths—children should be made to cry vigorously, in order to insure proper expansion of the lungs. They require no drugs, but a great deal of careful nursing.

CHAPTER VI.

DISEASES DUE TO FAULTY NUTRITION.

THE diseases due to faulty nutrition are numerous. There are two, however, which have been so clearly shown to originate in this way that they may be put in a class by themselves. These are scorbutus and rickets. The prevailing opinion of the medical profession is that both of these are essentially "food-diseases." The purpose of considering them in connection with the disturbances of nutrition is to emphasize this relationship.

SCORBUTUS (SCURVY).

Scorbutus is a constitutional disease, due to some prolonged error in diet. It is characterized by spongy, bleeding gums, swellings and ecchymoses about the joints, especially the knee and ankle, hæmorrhages from the nose, and occasionally from other mucous membranes, extreme hyperæsthesia, and often pseudo-paralysis of the lower extremities. Added to these local symptoms there is usually a general cachexia with marked anæmia. While scorbutus and rickets are very frequently associated, they are not necessarily connected, and can hardly be considered as different forms of the same disease; although cases of scorbutus have been described in older writings under the title of Acute Rickets. In Germany it is known as Barlow's disease.

For the statistical matter here presented I am indebted to the report of the American Pædiatric Society's Collective Investigation of Infantile Scurvy in 1898, embracing 379 cases, reported by 138 observers. Of these, 31 cases were from my own practice.

Etiology.—Age is an important factor; more than four-fifths of the cases occur between the sixth and the fifteenth months, and half of them between the seventh and the tenth months. Scurvy has been seen in infants under a month old. The great majority of the cases reported have been observed in private practice, often in the best surroundings. Previous disease is not a factor of much importance. Most of the children attacked have been in good health up to the development of scurvy. In about one-fourth of the number some previous derangement of the digestive tract has existed.

The only etiological factor yet known to bear any constant relation to the production of scurvy is diet. The important facts regarding the previous diet brought out by the Society's investigation are as follows:

Previous food	Breast-milk	in	12 cases;	alone in 10.
	Raw cow's milk........	"	5 "	" " 4.
	Pasteurized milk.......	"	20 "	" " 16.
	Condensed milk.......	"	60 "	" " 32.
	Sterilized milk........	"	107 "	" " 68.
	Proprietary infant-foods	"	214 cases.	

This table shows that while scurvy may occasionally develop with almost any variety of food, three stand out prominently—viz., proprietary infant-foods, condensed milk, and sterilized milk. In all of these it would appear that something needed for normal healthy nutrition is wanting. Scurvy is not likely to follow unless an improper diet is continued for a long period, usually several months. In some instances where it developed in nursing infants, the nurse's milk has been examined and found totally inadequate to the needs of nutrition, many of the children having exhibited serious disturbances of nutrition before any signs of scurvy appeared.

In several of the cases reported as occurring with a diet of raw or pasteurized milk it is certain that the milk formula used was at fault, the most common condition being low proteids. Several cases have come under my personal observation where children had been kept for four or five months upon percentages which should have been continued only a few weeks. However, I have seen at least three cases of scurvy which developed while taking pasteurized milk where no such explanation was possible, and the heating (167° F. for thirty minutes) seemed to be the cause. The number of cases occurring while upon a diet of sterilized milk (usually heated to 212° F. for one hour) is so large that we are driven to the conclusion that the heating alone was the cause, especially since prompt recovery has frequently followed when no other change was made than to discontinue the heating. These facts show that sterilized milk should always be prescribed with caution, its effects watched, and patients warned of its possible danger; it should not be continued as the sole diet for long periods.

No one fact in the etiology of scurvy is better established than its development after the prolonged use of condensed milk or the proprietary infant-foods. In this respect, as with reference to sterilized milk, my personal experience, including now upward of sixty cases of scurvy, coincides with the findings of the Society's report.

While it may be regarded as established that the cause of scurvy is dietetic, no single dietetic error can be held responsible for the disease. At present it seems impossible to go further than to say that something necessary to normal nutrition is lacking in the food. None of the theories yet advanced in explanation of how diet causes scurvy is wholly satisfactory.

Lesions.—The most marked effects of scurvy are seen in the bones, blood-vessels, and the blood. The number of recorded autopsies is not yet large, only six being included in the Society's report. I have myself had the opportunity of making examinations in three cases. The findings are remarkably uniform, but represent, of course, the extreme results of the disease. The most striking lesion is subperiosteal hæmorrhage, which is practically constant and may occur almost anywhere in the body, but affects chiefly the bones of the lower extremities; it is often very extensive, and may reach from the knee to the great trochanter, or from the ankle nearly to the knee. Extravasations may also be found between the muscles, and blood may infiltrate the cellular tissue in the neighbourhood of the joints. Besides these lesions resulting from hæmorrhagic periostitis the bone itself may be affected. Separation of the epiphyses from the shaft of some of the long bones, generally at the lower end of the femur or lower end of the tibia, is found in most of the fatal cases. Notwithstanding the serious lesions near the large joints, the joints themselves are usually normal.

The minute bone changes are very similar to those of rickets. But there are also differences of importance. The disposition to hæmorrhage, which is altogether the most characteristic feature of scurvy, is entirely wanting in rickets. The visceral lesions are inconstant. Those most frequently found are small hæmorrhages beneath the pleura, pericardium, and peritonæum, sometimes into the various organs, also bronchopneumonia, and nephritis. There may be small extravasations found upon the surface of any of the mucous membranes. The alterations in the blood-vessels are undoubtedly an important factor in bringing about the disposition to hæmorrhage, but as yet they have been very imperfectly studied. The changes in the blood, in the gums, and the lesions of the skin will be considered with the symptoms.

Symptoms.—In most cases a period of indisposition, fretfulness, pallor, and failing nutrition precedes the local symptoms, but usually tenderness of the legs is the first symptom noticed. In the beginning this is occasional and so slight as to cause the infant to cry only upon handling. Later it becomes almost constant and is very acute. At first this soreness is not very definitely localized, but is generally more marked about the knees and ankles. Some swelling may be noticed, often just above the ankle-joints. Coincident with these may be seen the changes in the mouth. The gums are of a deep purplish colour, swollen, particularly about the upper central incisors, and may quite cover the teeth. They bleed from the slightest rubbing, and sometimes spontaneously. The child becomes fretful and cross, sleeps badly, loses colour, weight, and appetite. It may become quite cachectic in appearance. All these symptoms come on gradually, often with periods of a few days in which apparent improvement is seen. Sometimes they may continue for several weeks without making any perceptible impression upon the child's previously good condition.

If the disease is recognised, and proper treatment instituted, rapid improvement follows, with complete and permanent recovery. If not recognised, and the faulty diet is continued, the disease advances to the more severe form. The tenderness of the legs becomes exquisite, so that any movement or even the slightest touch causes the child to scream with pain or apprehension. The legs often lie motionless, and no voluntary movement can be excited by any means. Paralysis is often suspected. The disability is chiefly owing to the extreme pain which motion provokes, but may depend upon epiphyseal separation. Small ecchymoses are frequently seen about any of the large joints, resembling the ordinary "black-and-blue" spots, and these often confirm the opinion previously formed that the child has met with some accident. The swelling near the joints, particularly the knee, may be so great that the limb is nearly twice the size of its fellow. The mouth symptoms are usually striking. In addition to spongy, swollen, bleeding gums, dark purplish bags may be seen over teeth not yet through. There may be bleeding from the roof of the mouth or from the pharynx. The pain is sometimes so severe as seriously to interfere with taking food; there is moderate though rarely extreme salivation. Blood may be vomited or passed with the fæces or the urine. In the severe cases the stools are rarely normal, more or less catarrhal colitis usually being present. The general condition is one of grave anæmia, accompanied by a marked cachexia and progressive wasting. The child cries almost constantly, sleeps little, and is truly a pitiable object. Slight fever is often present during the last few weeks. Unless recognised and the cause removed, the condition grows steadily worse, the symptoms continuing until death occurs either by a slow asthenia, suddenly from heart failure, or from some intercurrent disease, such as broncho-pneumonia or acute gastro-enteritis. The duration of the illness in the fatal cases is from two to four months.

The onset is gradual in the great majority of the cases, the earliest symptoms noticed in the order of frequency being pain and tenderness of the legs, soreness and sponginess of the gums, disability, anæmia, cutaneous hæmorrhages, and very rarely hæmaturia.

Pain and tenderness are very prominent, being noted in 95 per cent of the Society's cases; in the majority they were present only on motion or handling. The location of the pain and tenderness in 184 cases was as follows: Lower extremities alone, 133; upper extremities alone, 2; lower and upper, 42; lower and trunk, 7. In all but two cases, therefore, the lower extremities were affected, the lower part of the thigh and the leg just above the ankle being the usual seat.

Disability, or pseudo-paralysis, is a very common symptom, and in all severe cases a constant one. It exists in varying degrees from the slight disinclination to use the limb to complete helplessness. In many cases it is more marked than the pain, and has led to a diagnosis of poliomyelitis.

Swellings are associated with pain and tenderness in most of the severe cases. They are most marked near the joints, but may extend for some distance along the shafts of the bones. In nearly all cases the location is the lower part of the thigh or the lower part of the leg, and usually of both sides. Swellings are occasionally seen near the wrists, elbows, shoulders, and hip-joints; in rare cases, over the ribs, scapula, or ilium. Redness is not generally present, but the parts may have a dark purplish colour. It is to the hæmorrhage that both the swellings and the discoloration are chiefly due.

Protrusion of the eyeball is present in about 10 per cent of the cases; an extreme exophthalmus is sometimes seen, and is due to orbital hæmorrhage.

The gums are affected in nearly all cases, the exceptions being those recognised and treated early. Hæmorrhage occurs in about one-half the cases, and frequently there is ulceration not unlike that of a mercurial stomatitis. It is rather curious that, though the lower teeth are cut first, the upper gum is almost always most affected, and in the milder cases usually alone involved. Of 45 cases in which no teeth had been cut, the gums were affected in 24 and normal in 21. This is sufficient to disprove the old opinion that the gums are affected only when teeth have appeared. The severe inflammation and ulceration sometimes seen seem to be the result of secondary infection.

Hæmorrhages beneath the skin are present in about half the cases. They are rarely extensive, usually multiple, and their location is no doubt often determined by a slight traumatism. Hæmorrhages from the mucous membranes are not quite so frequent. There may be bleeding from the gums, nose, bowels, kidneys, and rarely from the stomach. Hæmorrhages in most cases are frequently repeated, but seldom profuse.

Epiphyseal separation is seen only in very severe cases. It is nearly always either of the lower epiphysis of the femur or the tibia, and is often bilateral. The separation is usually caused by some slight injury, the condition of the bone predisposing to this occurrence. In a case of my own which recovered, rapid union occurred under anti-scorbutic treatment.

Anæmia is slight in the early stage, but steadily increases as the disease progresses. Blood examinations show great reduction of the hæmoglobin, sometimes to 35 or 40 per cent; also in nearly all cases a proportionate reduction of the red cells. Leucocytosis and poikilocytosis may be present.

The urine contains albumin in one-fourth of the cases; in nearly half of those containing albumin casts also are found. In rare cases hæmaturia has been the first symptom noticed; usually, however, it occurs later, and is seen in about 5 per cent of the patients.

Evidences of general malnutrition are present in all advanced cases, varying, of course, greatly in degree. In a few infants under my own observation the weight, colour, and general appearance of health have continued in spite of very decided local symptoms. In most of them the impaired nutrition is shown by loss of appetite, occasional attacks of vomiting, and still more frequently by derangements of the bowels, which vary from slight indigestion to a serious catarrhal condition of both small and large intestine. It is with the latter that the discharge of blood is usually seen.

Association with Rickets.—In the Society's investigation great pains were taken to obtain definite and accurate data regarding this. Of the cases, 340 in number, in which this point was noted, symptoms of rickets were present in 152, or 45 per cent; these symptoms were recorded as slight in 72; marked in 64; and not specified in 16. In the remainder of the cases, 55 per cent, it is definitely stated that symptoms of rickets were absent. It is also stated that in 50 of the patients which were rachitic, the rickets antedated the development of the scurvy. From these facts it would seem to be pretty well established that though rickets and scurvy have points of resemblance, such as the age when they are seen, bony changes, dependence on defective nutrition, etc., they can not be regarded as different forms of the same disease. The two most striking characteristics of scurvy—viz., tendency to hæmorrhages and prompt curability by fresh food and fruit juices—have no counterpart in rickets. However, their coexistence in the same patient is of common occurrence.

Diagnosis.—The disease with which infantile scurvy is most frequently confounded is rheumatism. In fully four-fifths of the cases which have come to my own notice this has been the previous diagnosis. The extreme rarity of rheumatism under one year should always make one cautious; pain and tenderness of the legs only, should, in an infant, invariably suggest scurvy rather than rheumatism. The extreme disability has often led to a diagnosis of poliomyelitis, but here again the acute tenderness should set one right. Many cases of scurvy come into the hands of the orthopædic surgeon with a diagnosis of joint or spinal disease. Where the swelling was mainly of one limb I have twice known a diagnosis of malignant disease to be made, from the cachexia, the shape of the swelling, the discoloration, and the pain. I have known two cases to be operated upon by eminent surgeons, once with a diagnosis of sarcoma and once of ostitis of both tibiæ. Not until the subperiosteal hæmorrhages and epiphyseal separation were discovered was the nature of the trouble suspected.

The diagnosis of scurvy seldom presents any difficulties to one who has once seen a case. No one need err if the essential features of the disease are kept in mind: the extreme soreness of the legs, spongy, swollen gums, swelling near the large joints, a tendency to hæmorrhages, and usually a history of the prolonged use of some proprietary infant-food, of sterilized or condensed milk. If any doubt exists, this will be removed by the prompt improvement and generally rapid cure following an anti-scorbutic diet.

Prognosis.—This is invariably good if the disease is recognised early. No patients with symptoms so serious improve with such marvellous rapidity as do the great majority of those with scurvy under proper management. The figures of the Society's report on this point are interesting. The average duration of the disease before treatment was begun in over three hundred cases was somewhat over three weeks. In 80 per cent striking improvement was noticed during the first week of treatment, and in 40 per cent within three days. Over two-thirds of these cases were well within three weeks, and nearly one-third within one week, after the beginning of treatment.

It is only when the disease is of long standing, when the malnutrition is severe, or when serious complications, usually involving the digestive tract, are present that the symptoms persist and the issue becomes doubtful. It is difficult to tell what the exact mortality of scurvy is. Any case allowed to go on may result fatally. The younger the infant the more likely is this to occur. I have seen three deaths in about sixty cases. Barlow's early article included thirty-one cases with seven deaths. It is rare that scurvy leaves any permanent effects. Recovery is not only rapid but complete. Relapses are extremely rare and have been observed only in one or two cases, where chronic indigestion existed of so extreme a character that proper feeding was impossible. The after-effects are usually the result of prolonged malnutrition, of which the attack of scurvy was only one manifestation.

Treatment.—This is remarkably simple—viz., to discontinue all proprietary foods, condensed milk or sterilized milk, and to substitute a diet of fresh cow's milk, modified to suit the child's digestion. With this treatment alone improvement will soon begin and complete recovery follow. However, the addition of fresh fruit juice is of the greatest value, and when it is given improvement is much more rapid. Hence it should always be combined with the change in diet. Orange juice is possibly to be preferred, but the juice of any fresh ripe fruit will answer the purpose. From half an ounce to four ounces a day may be given, best in divided doses, given about one hour before the milk-feeding. The only really difficult cases to manage are those in which the general condition approaches one of marasmus, or when scurvy is accompanied by marked gastric or intestinal disturbance. When an intestinal catarrh is present, with the bowels moving five or six times a day, one may hesitate to give the fruit juice for fear of increasing these symptoms. In a number of instances I have seen intestinal symptoms, which had resisted ordinary measures, immediately improved by the fruit juice, thus establishing their intimate connection with the scorbutic condition.

Other things of value are fresh beef juice, and for older children fresh vegetables, especially potato. The anæmia and malnutrition call for iron, cod-liver oil, and other tonics, which should be given after active symptoms of the disease have disappeared. Infants with scurvy should be handled as little as possible, and should be particularly protected against exposure in their extremely susceptible condition.

RICKETS (RACHITIS).

Rickets is a chronic disease of nutrition. While the only important anatomical changes are found in the bones, it is not to be regarded as a bone disease; but as a very complex pathological process which affects the bones, muscles, ligaments, mucous membranes, and nearly all the organs of the body, particularly those of the nervous system. It occurs especially between the ages of six months and two years. It is not common in the country, but is exceedingly frequent in most large cities. While not a fatal disease *per se*, rickets adds very greatly to the danger from all acute diseases in infancy, and evên to some degree also to those of later life. Under proper conditions of diet and hygiene it tends to spontaneous recovery.

Etiology.—The essential cause of rickets is dietetic, although hygienic influences play a very important rôle in its production. While it seems to be demonstrated that diet alone may produce rickets, nevertheless this condition is much more easily produced when there are also unfavourable hygienic surroundings. Rickets is not common in nursing children unless lactation be unduly prolonged,* as, for example, where nursing is continued for fifteen to eighteen months without other food. Artificially-fed children are much more prone to the disease, especially those who are badly fed. The diet in these cases is usually very deficient in fat, and often at the same time in proteids, while it contains an excess of carbohydrates. It is somewhat difficult to separate the effects which these different conditions produce. It appears, however, that the most important factor is a great deficiency in fat. Rickets is exceedingly common in children reared upon the proprietary foods, nearly all of which are very low in fat and contain an excess of carbohydrates. It is also common in children who are reared upon sweetened condensed milk, and for precisely the same reason. When both fat and proteids are low, rickets is more liable to result than when only the fat is deficient.

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* An exception to this statement must be made in the case of Italian and Negro children. In this class as observed in New York it is very common to see marked rickets in those getting nothing but the breast.

Hygienic surroundings are next in importance to diet. Although, as previously stated, rickets is essentially a disease of cities, being principally seen in children living in crowded tenements where the effects of improper food are most strikingly shown, yet even here the disease is rare in those who get a plentiful supply of good breast milk.

Animal experiments.—Bland-Sutton experimented, in the Zoölogical Gardens, London, upon lion whelps. Those which were weaned early and fed solely upon raw meat invariably became extremely rachitic. Two young cubs, fed upon rice, biscuits, and raw meat, died from rickets. Two young monkeys, upon an exclusively vegetable diet, became rachitic. To the young lions who had developed rickets, milk, cod-liver oil, and pounded bones were given in addition to the meat, and in three months, although the hygienic condition of the animals remained unchanged, all signs of rickets had disappeared. Guerin produced typical rickets in puppies which were kept upon a meat diet for four or five months, while others of the same litter, which were suckled, remained in good health. Other animal experiments by various observers with different articles of food have given results that were not uniform. It seems, however, to be pretty positively established, that withholding milk from young animals and putting them upon a diet of meat, vegetables, or starches is sufficient to produce rickets, and that the earlier this is done the more certain is the result. This may occur apart from any change in the hygienic surroundings. These animal experiments strengthen the opinion above given, that the essential cause of rickets is improper food, and that the element most uniformly lacking is fat.

Distribution of rickets.—According to Palm, the disease is almost unknown in the extreme north—Greenland, Iceland, Norway, and Denmark. It is also very rare in China, Japan, Greece, Turkey, and the southern portions of Italy and Spain. Its greatest frequency is in the temperate zone. The general immunity of children in southern latitudes appears to be due to the out-of-door life, and the almost universal custom of maternal nursing. In the cities of America no race is exempt from the disease. In New York the greatest susceptibility is among the Negroes and the Italians. Extreme cases of rickets are almost invariably in one of these nationalities. It is exceptional to see in a dispensary or hospital a child of either of these races who does not show, to a greater or less degree, the signs of rickets. These two southern races seem to bear very badly the climate and the confined life of the northern cities. So far as my observations are concerned, there is no peculiarity in the food of these people which explains the prevalence of rickets among them, and this must be attributed to a race peculiarity. In the country, the immunity from rickets is due partly to the more prevalent custom of maternal nursing, and partly to the better surroundings; the increased resistance of the children rendering them much less susceptible to the influences of bad feeding than those of the cities. In New York among dispensary and hospital patients, rickets is exceedingly common, and is seen in all nationalities, although chiefly in the foreign elements of the population.

Heredity.—There is no evidence that rickets is a hereditary disease. Any cachexia in the parents, such as syphilis, tuberculosis, or alcoholism, may, however, by diminishing the child's resistance, be a predisposing cause of rickets. The later children in a family are more likely to be affected than the earlier ones, especially when the interval between the pregnancies has been short, or where anything else has caused a deterioration in the general health of the mother.

Previous disease.—Rickets not infrequently develops in syphilitic children; the connection, however, seems to be no closer than with any other cachexia. The relation of rickets to other diseases, particularly to those of the digestive tract, is very much less intimate than one would expect. Acute diseases of the stomach and intestines are very frequently followed by marasmus, but only exceptionally by marked rickets. There is no sufficient ground for believing that rickets exerts any protective influence against tuberculosis, as has been asserted. In fact the thoracic deformity of rickets may be a predisposing cause to tuberculosis.

Rickets affects both sexes with equal frequency. The symptoms usually manifest themselves between the sixth and fifteenth months. Congenital and late rickets will be considered separately.

Rickets is therefore a complex disease of nutrition, whose exact pathology has not yet been definitely settled. It is more difficult to believe that the general nutritive disturbances are the result of the bone changes, than to regard both as having a common origin. Kassowitz regards the bone changes as inflammatory, excited by the presence of some irritant. The irritant has been believed by many to be lactic acid, originating in the digestive tract; but the evidence in support of this theory is not conclusive. It is very doubtful whether the process is as simple as the formation of lactic acid in the intestine and its circulation in the blood. It is, however, clear that it is something which interferes with the assimilation of the lime salts. At the present time, the disposition is to regard rickets as a disease of nutrition, which may be produced in animals by certain dietetic changes. In infants, it seems to be settled that it may be produced by similar changes in diet, aided very greatly, however, by unhygienic surroundings. The effect of these abnormal conditions is shown upon the whole organism, but the only constant and regular anatomical changes are in the bones. These osseous lesions resemble those of chronic inflammation. Precisely how the dietetic and other causes produce the bone changes is still a matter of speculation. The constancy of bone changes in rickets gives it a place as an essential disease, and not merely a form of malnutrition.

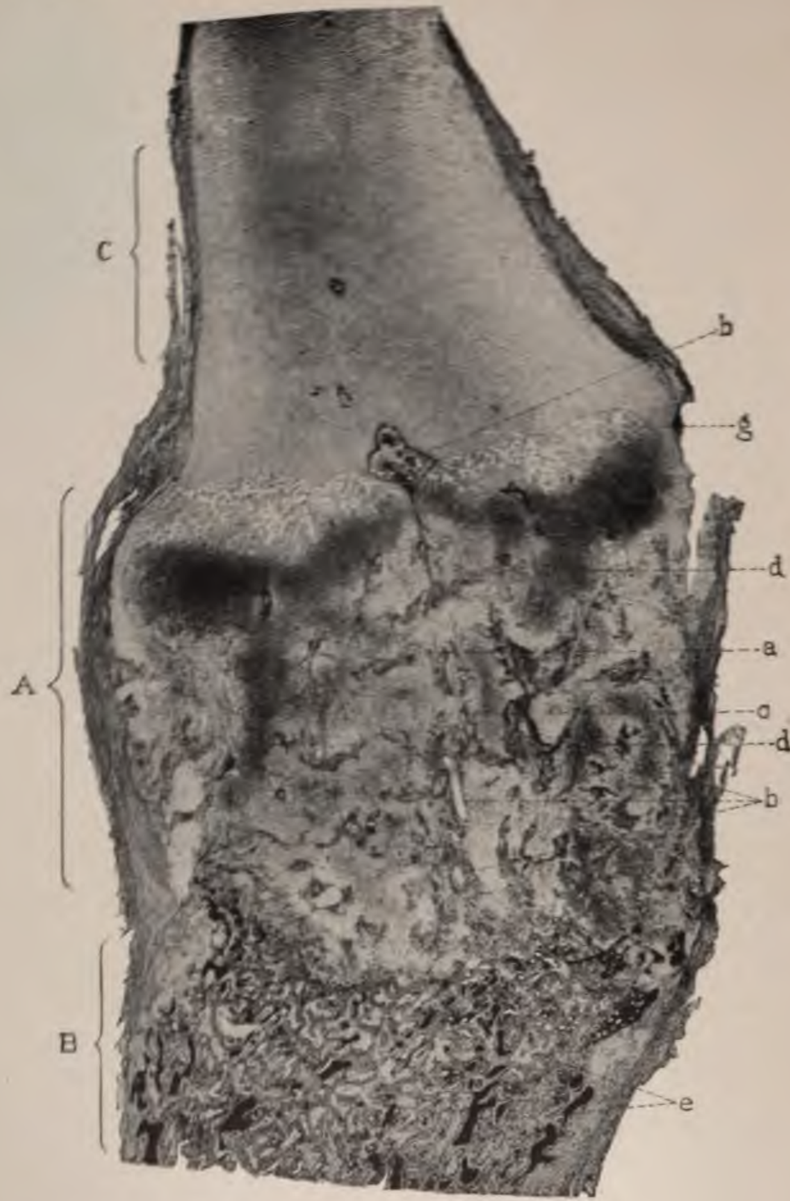
Lesions.—The only constant and characteristic lesions of rickets are found in the bones. It is still a matter of dispute whether these bony changes are to be considered as inflammatory, or simply as the result of disordered nutrition. Disordered nutrition and chronic inflammation are closely allied, and it really makes but little difference which view is taken. Occurring at a time when the growth of bone is so rapid, the effects of rickets are very striking and very serious.

In order to appreciate how the bones are affected by rickets, it must be remembered that the long bones grow in length by the production of bone in the cartilage between the epiphysis and the shaft; that the shaft grows in thickness by the production of bone beneath the inner layer of the periosteum; and that the medullary canal is continually increasing in size by the absorption of the inner layers of the bone. In rickets there is an exaggerated production of cartilage at the epiphysis, and excessive cell-growth beneath the periosteum, while the process of ossification in these tissues goes forward slowly and imperfectly, or is entirely arrested. At the same time the absorption of the medullary layers may be even more rapid than normal. In health the growth of bone in length is much more rapid than its increase in diameter, owing to the greater activity of the changes taking place at the epiphysis; so, in rickets, it is at the extremities of the long bones that the most marked changes are seen.

One of the most striking features of rachitic bones is their unnatural flexibility. This is due to deficient ossification in the superficial layers of the shaft of the long bones, and also at their extremities. Normally, bone contains about one third organic and two thirds inorganic matter. In marked rickets the proportions are reversed, the bones often containing twice as much organic as inorganic matter. Changes are seen in all the long bones, but all are not affected to the same degree. Sometimes those most affected will be the bones of the leg, sometimes those of the forearm, and sometimes the ribs. The extent varies with the severity of the process.

There are characteristic changes in form. The most constant is enlargement of the epiphyses of all the long bones. This is most strikingly seen in the lower extremities of the radius and tibia. The enlargement may be so marked that the width of the epiphysis is increased by one half. All the sharp angles, borders, and prominences of the bones are rounded off. The curvatures of rachitic bones are more fully described under the head of Symptoms. They may be due to a variety of causes. Some are simply an exaggeration of the normal curves, much increased by the swelling of the epiphyses; others are due to muscular action, to atmospheric pressure, to some unnatural posture, such as the cross-legged position, to the weight of the limbs, or to the weight of the body. The principal change in the form of the flat bones consists in the production of large bosses or prominences due to thickening of the bone, usually about the centre of ossification. These bosses are soft and spongy. Frac-

PLATE IV.



BONE IN RICKETS.

Longitudinal section of a rib at the junction of the costal cartilage, in a severe case of rickets (slightly magnified). C = costal cartilage. B = bone, A = proliferating cartilage-zone, which is much widened. Between the hypertrophied cartilage cell-columns (*a*) making up this proliferating zone, are seen medullary spaces (*b*) containing blood-vessels. In this zone lie masses of bone (*c*) not calcified. The calcification zone is almost wanting, only scattered islands (*d*) of calcified cartilage-cells being seen.

Beyond this proliferating zone (A) is a layer of bony tissue (B) made up of small bands of which only a few have a nucleus containing lime (*e*). These nuclei appear black. The bony bands differ both in form and arrangement from those of normal ossification. Between the bony masses are medullary spaces which appear light in the illustration. At (*g*) the beginning of cartilage proliferation is seen. Above this zone the cartilage is normal. (From Karg and Schmorl.)

tures are not uncommon. The bones most frequently broken are the radius and ulna; next, the clavicle or the ribs. The fractures are usually of the green-stick variety. There is a bending of the outer and a fracture of the inner layers of the shaft of a long bone. This results in more or less impaction, and is usually followed by the production of considerable callus. The epiphyseal changes result in arrested growth in length, rachitic bones being usually much shorter than normal. Increased vascularity is seen in the bosses upon the flat bones, at the extremities of the long bones and upon stripping the periosteum from the shaft.

In a longitudinal section of one of the long bones, the principal change seen at the extremity is that the cartilaginous layer which unites the epiphysis and the shaft is very much enlarged, both in width and thickness, the latter being sometimes four or five times the normal. This cartilaginous area is of a bluish colour, rather softer than normal cartilage. On one side it blends with the cartilage of the epiphysis, on the other it presents an irregular dentated border, and in it the calcified areas are irregular and scattered. The epiphyseal centres of ossification are enlarged, softer, and more vascular than normal, thus increasing the size of the extremity of the bone. In the shaft, the outer layers of bone are thickened and soft, like decalcified bone, the deeper parts being firmer, while the deepest layers may be completely ossified. The medullary canal is much more vascular than normal, its contents resembling granulation tissue. Toward the extremities the trabecular spaces are much increased in size, so that the bone appears unnaturally porous. On vertical section of one of the flat bones—e. g., one of the bosses upon the skull—there is found a great increase in the size of the trabecular spaces. The bosses are made up of large spongy masses, so soft as to be easily indented with the finger, and on pressure there oozes blood and serum in a considerable quantity.

Microscopical changes.—At the junction of bone and cartilage at the extremity of one of the long bones, there are readily traced in normal bone (Fig. 42) several distinct zones. Next to the hyaline cartilage (*a*) there is a proliferating zone (*b*), made up of cartilage cells and matrix, the cells having no orderly arrangement. Next to this is a columnar zone (*c*, *d*), in which the cartilage cells are arranged in regular rows or columns. Adjoining this is the zone of calcification (*e*); and, finally, there is the zone of ossification (*f*, *g*), where true bone is formed.

In rickets (Plate IV and Fig. 43), the principal changes are seen in the proliferating and columnar zones. The proliferating zone (Fig. 43, *b*) is increased chiefly by the multiplication of new cells; it is also more vascular than normal. The columnar zone (*c*) is affected in a similar way and to a much greater degree. It is less regular in its formation, and, instead of containing but few vessels, it shows large vascular channels, sometimes surrounded by medullary spaces (*e*). The ossification zone, instead of being narrow and sharply outlined, is broad and very irregular.

Calcified areas (*f*) may be seen in the midst of regions which are cartilaginous, while masses of cartilage (*h*) occupy areas which should be completely calcified. In some places there appears to be a transformation of cartilage into bone-tissue of an inferior sort by a direct or metaplastic process. In the shaft there is seen more or less thickening, and an increased vascularity of the periosteum. Beneath the inner layer there is excessive cell-proliferation, while calcification of this new tissue is imperfect or absent, and instead of hard, compact bone, we find irregular, spongy masses. In the spongy bone there is considerable thickening, with an erosion of bony trabeculæ, which results in the formation of large medullary spaces filled with blood-vessels and connective tissue rich in cells.

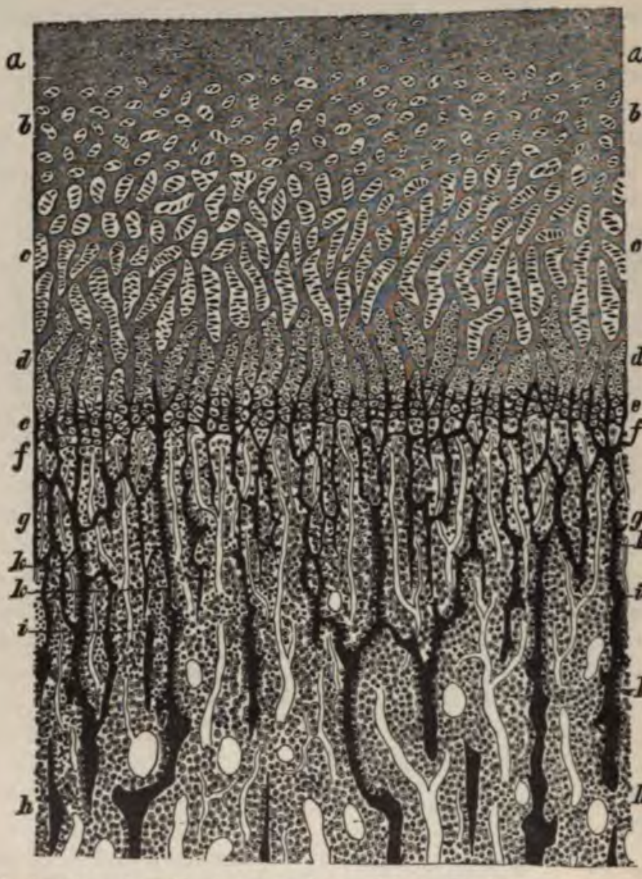


FIG. 42.—Section through ossification zone of normal bone (Ziegler). *a*, hyaline cartilage; *b*, zone of beginning cartilage proliferation; *c*, columns of cartilage cells; *d*, columns of hypertrophic cartilage; *e*, zone of temporary calcification; *f*, zone of primary medullary spaces; *g*, zone of primary bone formation; *h*, fully developed spongy bone; *i*, blood-vessels; *k*, layer of osteoblasts.

Termination of the rachitic process.—After a variable time, usually from three to fifteen months, the active proliferative process going on in the cartilage and beneath the periosteum ceases, and is gradually replaced by ossification. The bone becomes less vascular, and a rapid formation of bone takes place in the normal way. In addition, there is in some places a direct transformation of cartilage into bone. Condensation and

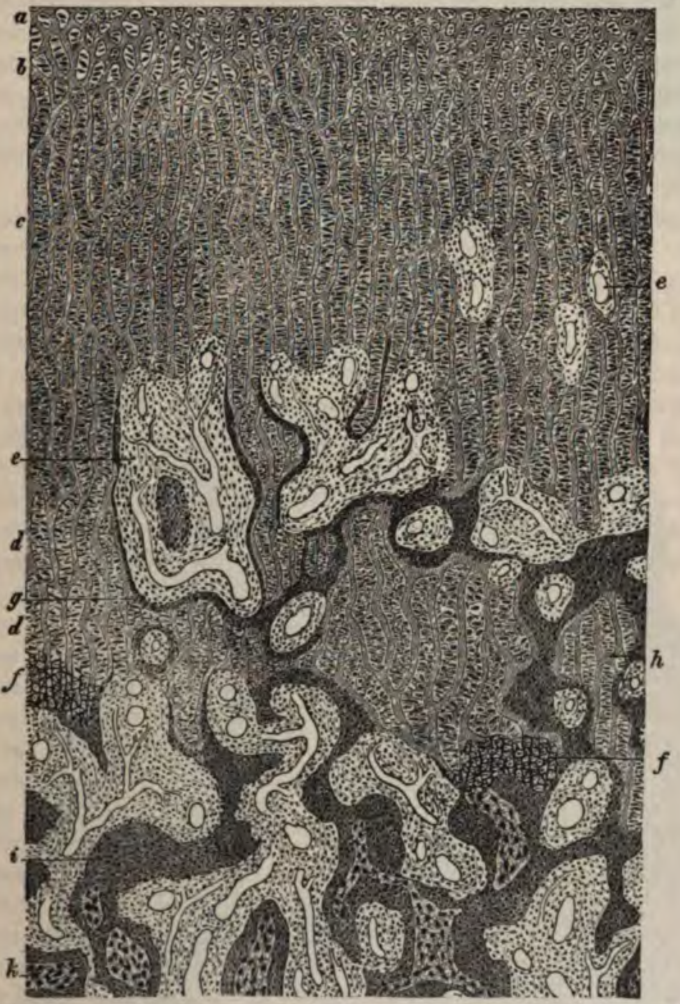


FIG. 43.—Rachitic bone (Ziegler). Longitudinal section through ossification zone of the upper diaphysis of the femur of a moderately rachitic child one year old (highly magnified). *a*, unchanged hyaline cartilage; *b*, beginning cartilage proliferation; *c*, columns of proliferated cartilage cells; *d*, columns of proliferated hypertrophic cells; *e*, medullary spaces containing blood-vessels lying within the cartilage; *f*, calcified cartilage; *g*, bony tissue; *h*, remains of cartilage within the bony tissue; *i*, point of uncalcified bony tissue; *k*, calcified bony tissue.

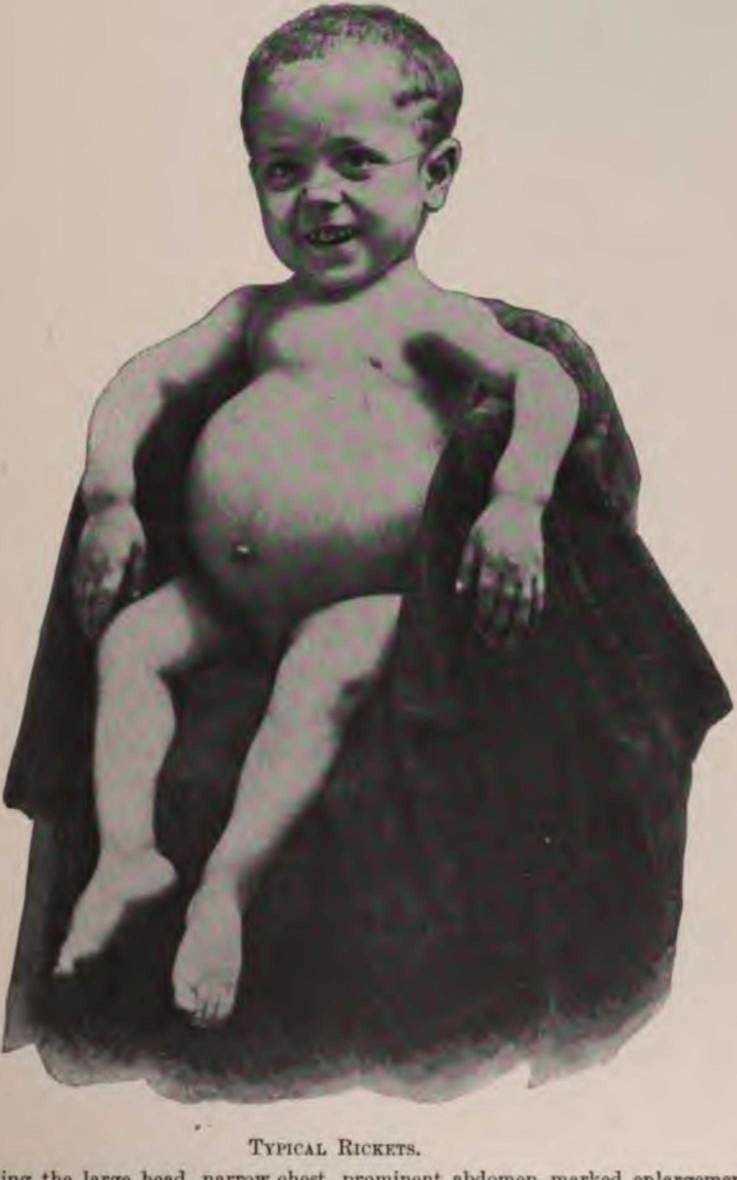
contraction take place in the spongy masses of bone. As the result of this, the affected bone may become even harder than normal; often it is ivory-like. Its structure, however, is never quite like that of healthy bone.

In the long bones the epiphyseal swellings slowly diminish, and may quite disappear; the slighter curvatures may be entirely overcome, and the greater ones much lessened. The beading of the ribs becomes almost imperceptible; the bosses upon the skull shrink very markedly, and may leave scarcely a trace of their existence. In most cases the active process in rickets has come to an end by the time the child is two and a half years old, often at two years.

Visceral lesions.—These are not infrequent, but are not essential to rickets. In the lungs they are due to deformities of the chest wall and to complications. Beneath the deep lateral furrows which are so common, there is found a part of the lung in a state of more or less complete collapse. This is accompanied by emphysema of the portion just anterior to it. Acute and chronic bronchitis and broncho-pneumonia are exceedingly frequent. A low grade of chronic catarrhal inflammation of the stomach and intestines is common, and is often associated with dilatation of these organs. The spleen is enlarged in most cases during the period of active symptoms. This is usually moderate in degree, although marked enlargement is not at all rare. The swelling of the spleen is due to simple hyperplasia, and not to amyloid degeneration. Enlargement of the liver is less frequent, and may occur with or without that of the spleen. There are no constant changes in the structure of these organs. The lymph nodes (lymphatic glands) are frequently enlarged. Rachitic patients are more prone to these swellings than are other children. They are due to simple hyperplasia, and have no close connection with rickets. Cerebral changes are rare, and those described are rather of accidental occurrence than dependent upon the rachitic process. As stated under Symptoms, enlargement of the head is usually due to thickening of the cranial bones. Although hydrocephalus is occasionally seen, it is extremely doubtful whether it is more frequent than in patients not rachitic. Hypertrophy of the brain has been described in connection with rickets, but as yet this does not seem to be established by sufficient pathological evidence. The muscles are flabby from imperfect nutrition, and sometimes atrophied from disuse, but no essential anatomical changes have been demonstrated in them.

Symptoms.—A well-marked case of rickets makes a striking picture (Plate V), and one not easily mistaken. There are seen the large head, beaded ribs, narrow chest, prominent abdomen, symmetrical swellings of the epiphyses of the wrists and ankles, and curvatures of the extremities. The beginning of symptoms is nearly always insidious, and the patient does not usually come under observation until they have existed for several weeks, often several months.

PLATE V.



TYPICAL RICKETS.

Showing the large head, narrow chest, prominent abdomen, marked enlargement of the epiphyses at the wrists and ankles. There are also curvatures of the forearms and legs which are not so well shown.

The patient a child two and a half years old.

Early Symptoms.—The most constant early symptoms are sweating of the head, extreme restlessness at night, constipation, beading of the ribs, and cranio-tabes. The head-sweating is rarely absent, and may continue for several months. It is especially profuse during sleep, the perspiration standing out in large drops upon the forehead, often being sufficient to wet the pillow. This is one of the causes of the nasal and bronchial catarrhs so common in rachitic infants. There is marked restlessness during sleep: the children tossing about the crib, kicking off the clothes, and never having the quiet, natural slumber of healthy infants. This may be due to many causes, but when persistent and associated with marked perspiration of the head, rickets should be suspected. Constipation is frequently seen as an early symptom, although it is more marked in the later stages of the disease.

The beading of the ribs is almost invariably the first appreciable change in the bones, and it is well-nigh constant. This forms the so-called "rachitic rosary," consisting of nodules at the line of junction of the costal cartilages and the ribs. It may be slight, or there may be a row of knobs as large as small marbles. In many cases with marked thoracic deformity, little or no beading of the ribs is seen externally, although at autopsy it is found to be very marked upon the internal surface of the chest (Plate VI). Beading of the ribs was noted in all but two of one hundred and forty-four successive cases of rickets, at the time of the first examination. In infants under six months there may be found soft spots in the cranium, usually over the occipital or posterior portions of the parietal bones. These are from one fourth to one inch in diameter, and there are usually several of them present. By pressure with the finger they give a sort of parchment-crackling sensation. This condition is known as cranio-tabes. Cranio-tabes is believed to be more frequent when syphilis is associated with rickets, and it is seen also in syphilitic cases which are not rachitic. A rachitic cachexia is not usually present until the symptoms have existed for several months, and in many cases it is not seen at all.

Deformities.—The deformities of rickets are almost invariably symmetrical in character, and usually numerous. In extreme cases almost every bone in the body is affected.

Head.—This usually appears to be too large, and although it may not be greater in circumference than that of a healthy child of the same age, it is out of proportion to the rest of the body. In marked cases the increase in circumference may be one or two inches. The enlargement is chiefly due to thickening of the cranial bones. In one case with marked deformity, I found the skull over the parietal bones half an inch in thickness (Fig. 44). This thickening diminishes with recovery, but in most cases the head remains throughout life larger than it should be. The shape of the rachitic head is somewhat square (Fig. 45), owing to the formation of large bosses over the parietal and frontal eminences. It is flattened at the occiput from pressure, and flattened also at the vertex. In extreme cases, the prominences upon the frontal and parietal bones may be so great as to produce quite a marked furrow along the line of the sagittal and frontal sutures, and one at right angles to this along the coronal suture (Fig. 46). This condition gives unusual prominence to the forehead. Marked deformity of the head has been observed in thirty-three per cent of my cases. The sutures may remain open for an unnatural time, occasionally until the end of the first year. The fontanel is late in closing, being frequently found open at two and a half, and sometimes even at three years. Often at eighteen or twenty months the fontanel is two inches in diameter. The veins of the scalp are often prominent, and the hair is frequently worn from the occiput, owing to restlessness during sleep. Occasionally rickets and hydrocephalus are associated, but the latter is the least frequent of all causes of the enlargement of the head.

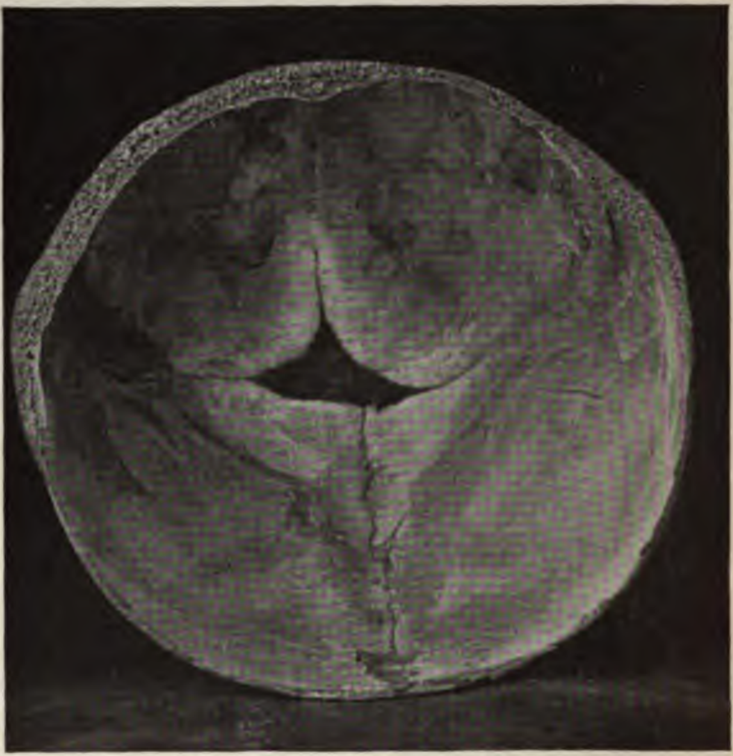
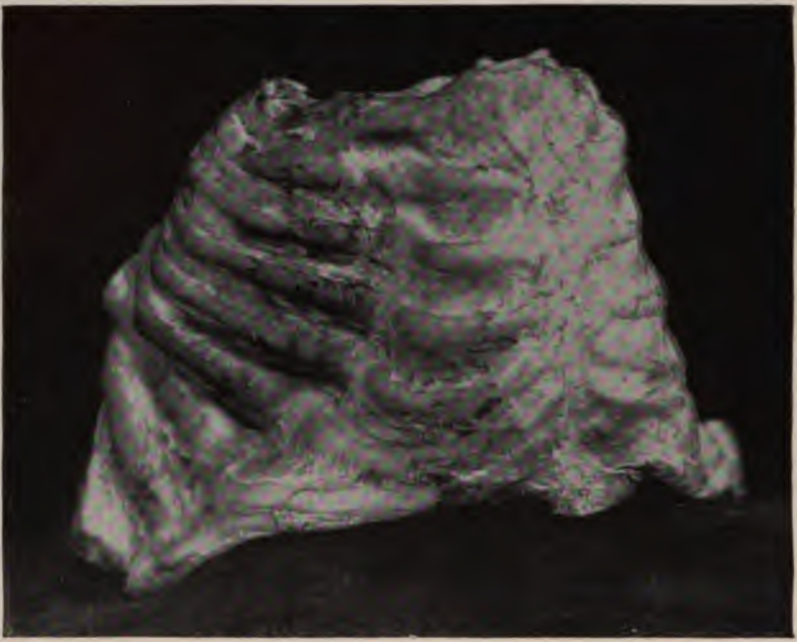
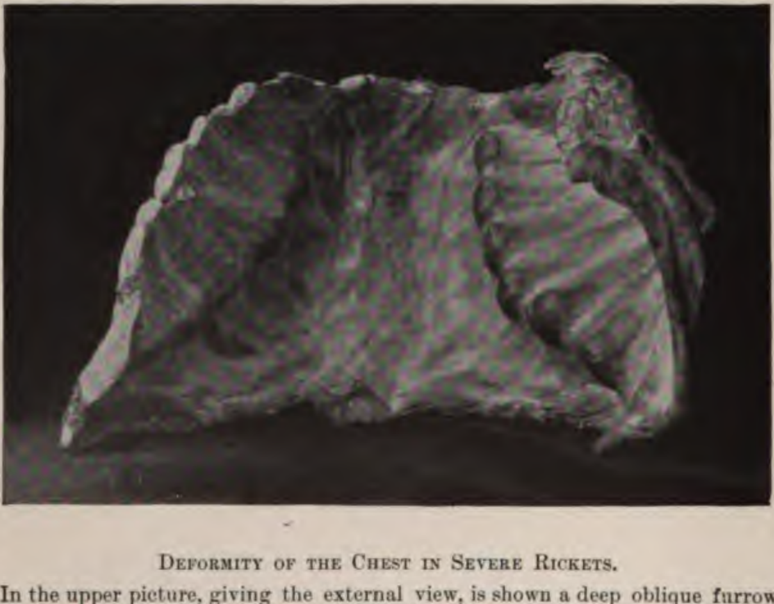


FIG. 44.—Rachitic skull from colored child two years old, horizontal section, inner surface showing thickening of the bones, especially the frontal, and open fontanel.

PLATE VI.





Deformity of the Chest in Severe Rickets.

In the upper picture, giving the external view, is shown a deep oblique furrow at the junction of the ribs and costal cartilages, these meeting at an acute angle.

In the lower picture the ribs have been separated from the spine and spread open, showing the same deformity as it appears from within, looking forwards.

From a coloured child ten months old.

Chest.—Beading of the ribs has already been mentioned. This is the most characteristic feature, but in the majority of cases there are, in addition, lateral depressions over the lower third of the chest, at the line of junction of the cartilages with the ribs, with eversion of the lower borders of the ribs. In severe cases these depressions or furrows are so great as to cause serious deformity (Plate VI). Usually there is a great diminution in the transverse and an increase in the antero-posterior diameter of the chest. Fig. 47 shows the outline of the chest of a rachitic child of two years, compared with that of a healthy child of the same age. Another frequent deformity is the "rachitic girdle," which consists in a transverse depression about two inches broad, extending from one side of the chest to the other, just above its lower border. A less frequent deformity is the "funnel chest," a deep central depression over the ensiform cartilage. This is sometimes nearly an inch and a half in depth. Marked thoracic deformity was seen in twenty per cent of my cases, but in only a small proportion was the chest normal.

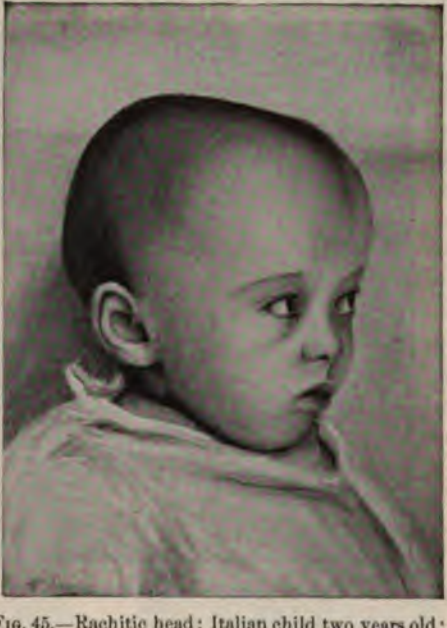


FIG. 45.—Rachitic head; Italian child two years old; square, prominent forehead and flat vertex.

The factors in the production of the thoracic deformity are atmospheric pressure and soft chest walls, these sinking in at the point where they have least resistance, viz., at the junction of the costal cartilages and the ribs. When there is any obstruction to the entrance of air, as with bronchitis, hypertrophied tonsils, or adenoid growths of the pharynx, the thoracic deformities are exaggerated. Irregular chest deformities depend upon the coexistence of pathological conditions in the lungs. Pigeon-breast is occasionally seen, but it is doubtful if this depends upon rickets alone.

Spine.—In very many of the milder cases this is normal. The most characteristic deformity consists in a posterior curve (kyphosis), which is a general one, usually extending from the mid-dorsal to the sacral region. This existed in forty-six per cent of my cases. In the early part of the disease it disappears entirely on suspending the child, or making extension upon the extremities; but in cases of long standing it may not disappear entirely by these tests. Very much less frequently there is seen a rotary curvature. This, in my experience, has been more frequently to the left side than to the right—the opposite of the common form of lateral curvature seen in young girls. Marked lateral curvature in children under three years is usually rachitic.

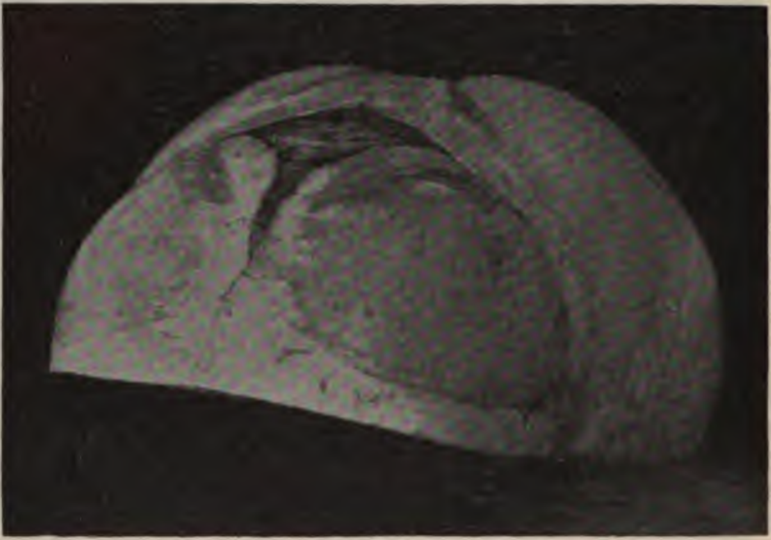


FIG. 46.—Rachitic skull from child one year old, showing frontal and parietal bosses and wide fontanel.

The clavicle is affected only in severe cases. The usual deformity consists in an exaggeration of the anterior curve at the inner third of the bone, which is somewhat shortened and its extremities enlarged. It is not infrequently the seat of green-stick fracture.

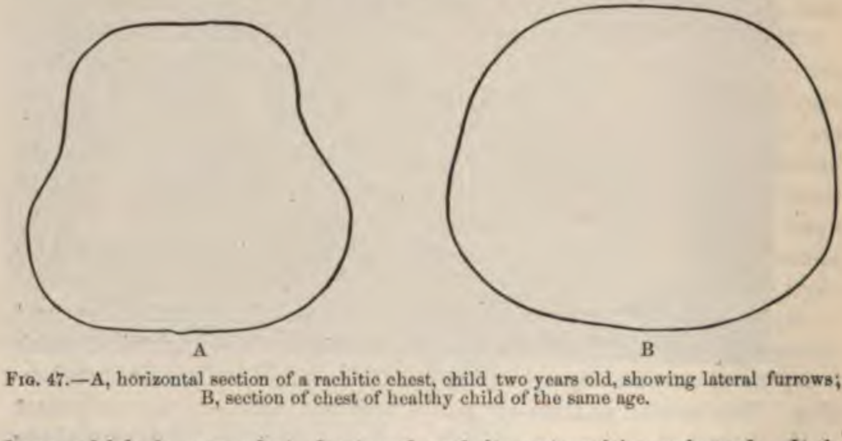


FIG. 47.—A, horizontal section of a rachitic chest, child two years old, showing lateral furrows; B, section of chest of healthy child of the same age.

Deformities of the pelvis belong to obstetrics rather than to pædiatrics. The most common rachitic change is a diminution of the antero-posterior diameter and a narrowing of the subpubic arch. Irregular deformities, sometimes described as "crumpling of the pelvis," are not infrequent.

Extremities.—Deformities of the upper extremities are usually symmetrical. The humerus is affected only in severe cases. It has a forward and outward curve, although rarely a very marked one. Both the epiphyses are enlarged, although the upper one can not often be made out unless the child is very thin. The radius and ulna are frequently affected. They present a convexity upon their extensor surfaces (Plate V), which in some cases is very marked, particularly in children who have been creeping about. Green-stick fractures here are quite frequent. Rachitic changes at the epiphyses are more common than in the shaft, enlargement of the epiphyses at the wrist being one of the most constant bony deformities of rickets (Plate V). It was present in ninety-five per cent of my cases. Less frequently similar swellings are seen at the elbow. Enlargement of the ends of the metacarpal bones or the phalanges I have seen in but two or three extreme cases.

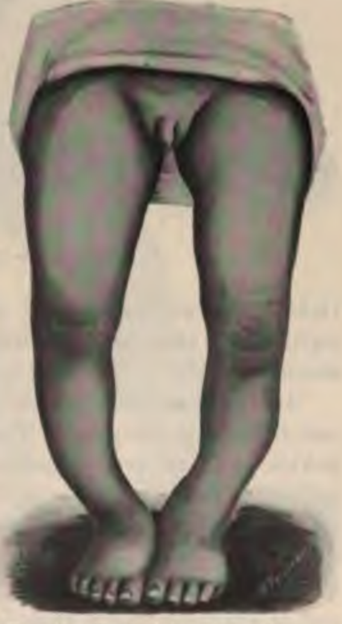


Fig. 48.—Typical bow-legs of severe form.

The lower extremities are rather more frequently affected than the upper, but in a similar way. The femur is involved only in severe cases; it commonly presents a general forward and outward curve, which is mainly due to the weight of the legs as the child sits. Occasionally there is also an outward rotation of the femur, where children have been allowed to sit much in a cross-legged posture. When such children begin to walk, the toes are turned very far outward. The principal deformities of the lower extremity are bow-legs (Fig. 48) and knock-knees (Fig. 49). Knock-knees are more common in females, and are believed to be due to an overgrowth of the inner condyles of the femur. Enlargement of both condyles can be demonstrated in most of the marked cases of rickets. The cases of slight bow-legs may be due simply to swelling of the epiphyses, the shaft of the bone being quite normal. This point I have verified by post-mortem observations. Such are probably most of the deformities which disappear spontaneously. The most severe cases of bow-legs are often associated with some degree of antero-posterior curvature, and the latter may be the principal deformity. An exaggerated case of this kind is shown in Fig. 50. Enlargement of the epiphyses at the ankle is usually present when it is seen at the wrists, and nearly to the same degree. Enlargement of the upper epiphyses of the tibia and the fibula is seen only in severe cases. The cause of the deformities of the leg is not, primarily at least, walking too early, since they are common in children who have never walked; slight deformities, however, may be aggravated by early walking. A change which has not been sufficiently emphasized is the arrested growth of the long bones; this is one of the most characteristic features of rickets. A rachitic child of three years often measures in height six or eight inches less than a healthy child of the same age, the difference being almost entirely in the lower extremities.

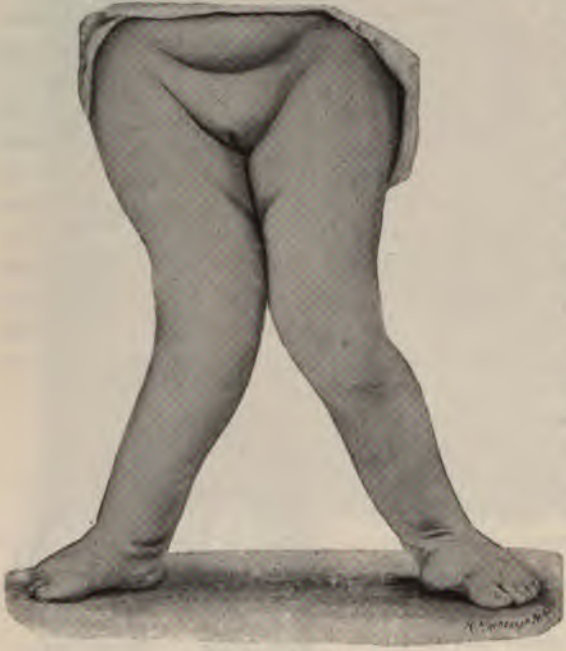


FIG. 49.—Knock-knees.

All the *ligaments*, but particularly those about the large joints, are lax and frequently elongated. This may lead to the deformity known as weak ankles, or to an over-extension at the knee (*genu recurvatum*); also to unnatural mobility at the hips, shoulders, elbows, and wrists. The condition of the ligaments plays an important part in the production of spinal deformities.

Muscles.—The muscular symptoms of rickets are almost as constant and as characteristic as those of the bones. The muscles are small, very flabby, and poorly developed; hence rachitic children are unable to sit erect, or to stand or walk at the proper age. Of one hundred and fifty-one cases in which the date of walking alone was investigated, only twenty-seven, or eighteen per cent, walked before the fifteenth month; forty-seven per cent were not walking at the eighteenth month; twenty per cent not at two years; and ten per cent not at two and a half years. Late walking is one of the most common symptoms for which advice is sought by parents with rachitic children. The muscular power in the extremities is sometimes so feeble as to suggest paralysis. I have seen a number of cases in which the symptoms so resembled paralysis, that even expert diagnosticians were unable to differentiate rickets from poliomyelitis except by the electrical reactions, those in rickets being usually normal or exaggerated. In other cases the symptoms may suggest cerebral palsy of the flaccid type. The muscular symptoms may be marked when the bony changes are slight, and conversely. As no lesions of the muscles have been demonstrated, the symptoms are probably due to imperfect nutrition. Two other symptoms depend chiefly upon the condition of the muscles, viz., pot-belly and constipation.

Pot-belly is quite an early symptom, and in most cases a very marked one (Plate V). It was noted in sixty per cent of my cases. The enlargement of the abdomen is uniform. It is everywhere tympanitic, and it may be as tense as a drumhead. It is due to a loss of tone in the abdominal muscles, and in the muscular walls of the stomach and intestine. It is aggravated by chronic indigestion and consequent intestinal putrefaction. The enlargement is thus mainly from tympanites. There may be a marked degree of dilatation both of the stomach and the colon. To a very small degree only, does the large abdomen depend upon swelling of the liver or spleen.

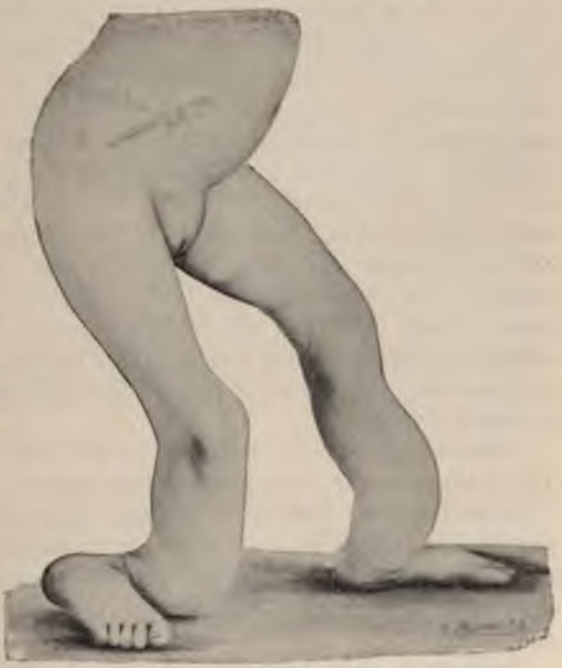


Fig. 50.—Extreme rachitic deformities of the legs.

The constipation of rickets, as already suggested, depends upon the loss of tone in the muscular walls of the intestines. It may alternate with diarrhœa. It rarely happens that a rachitic child has habitually normal evacuations from the bowels. Hard, dry, constipated stools frequently set up a condition of chronic catarrh of the colon in which large masses of mucus are discharged.

During the most active part of the disease—viz., from the third to the ninth month—*tenderness* may sometimes be elicited by pressure upon the epiphyses. This, however, is not a constant symptom, and a very unreliable one for diagnosis. In my own experience it has been present in but a very small proportion of the cases. Acute tenderness should always suggest scurvy rather than rickets.

Fever.—According to some observers there is a febrile movement which belongs to the active stage of rickets, but I have never been able to satisfy myself of the truth of this observation.

Dentition.—As a rule, dentition is late and apt to be difficult—i. e., it is associated with attacks of indigestion or other disturbances which may be serious. Individual cases, however, present great variations in regard to this symptom. A study of the progress of dentition in one hundred and fifty rachitic children gave the following results: in fifty per cent the first teeth were cut on or before the eighth month, and in thirteen per cent on or before the fifth month; however, twenty per cent of the cases had no teeth at twelve months, and in eight per cent none had appeared at fifteen months. Even though the first teeth come at the usual time, the progress of dentition is often arrested by the development of rickets, and no advance made for five or six months. The difference in the cases appears to depend very much upon the age of the child when rickets begins. Those who give no evidence of it until nine or ten months old often have a nearly normal dentition, while the cases developing early show a marked retardation of this process. The order in which the teeth appear may be very irregular, but there is no rule in this respect. The character of the teeth in rickets, in the great majority of cases, is good. This was true in eighty-four per cent of one hundred and twenty-six cases examined with reference to this point. This is in striking contrast to hereditary syphilis, where the tendency to early decay is so constantly seen.

General appearance.—Rachitic patients are almost always anæmic. The blood is low in hæmoglobin, often down to thirty or forty per cent. In some few cases there is in addition quite marked leucocytosis. The number of red globules is not often nor uniformly affected. The majority of rachitic patients are fat and flabby. The tissues are soft and have but little resistance. Rarely, they may be thin, like patients suffering from marasmus.

Rachitic patients are very prone to suffer from hypertrophied tonsils, adenoid growths of the pharynx, and enlargements of the lymph nodes of the neck. In all forms of acute illness the feeble resistance of these patients is very evident. This is especially true of acute disease of the lungs.

The *mucous membranes* are very vulnerable in all rachitic patients. From the slightest indiscretion in diet an attack of acute indigestion or diarrhœa is brought on, and from a very insignificant exposure, catarrhal inflammation of the upper or lower air passages is excited. In rachitic patients all such attacks are prone to run a protracted course. Inflammation of the trachea and larger bronchi is liable to extend to the smaller bronchi and the lungs.

The downward displacement of the *liver* and *spleen* from contraction of the chest should not be mistaken for enlargement of these organs. Moderate enlargement of the spleen is very common during the stage of most active symptoms—i. e., from the sixth to the twelfth month. Great enlargement of either liver or spleen is infrequent.

Blood.—From a study of the blood in twenty cases of rickets, Morse (Boston) concludes that anæmia is present in most cases, its intensity varying with the severity of the rachitic process. All the usual forms of anæmia are seen. Leucocytosis may or may not be present; it is more marked in cases attended by an enlarged spleen. All or any of the white cells may be increased.

Nervous symptoms are among the most frequent manifestations of rickets. Restlessness at night has already been mentioned as a prominent early symptom. Pain and tenderness are rare. A disposition to muscular spasm is seen in many cases. There may be laryngismus stridulus, tetany, or general convulsions. The first two are rare except in rachitic patients. All of these probably depend upon defective nutrition of the nervous centres. While in all infants, owing to the irritability of the nervous centres, convulsions are easily excited from relatively slight causes, in those who are rachitic this susceptibility is greatly intensified. As a predisposing cause of convulsions in infancy, rickets takes the first place. The younger the child and the more active the rachitic process, the more frequently do convulsions occur. They belong especially to the first year, being most frequent between the third and ninth months. The exciting cause of convulsions in these cases is usually to be found in the stomach or intestine.

Course and termination.—Rickets is essentially a chronic disease, and its course is measured by months. The active symptoms in most cases continue from three to fifteen months, although they occasionally last a much longer time. The duration of the symptoms depends chiefly upon the duration of the exciting cause. That active symptoms cease when a child reaches the age of eighteen months or two years, is no doubt due largely to the fact that at this age the diet is more general, and is more likely to furnish what the child needs, and that more fresh air is likely to be secured than at an earlier age.

The earliest symptoms of improvement are a diminution in the nervous symptoms, especially in the restlessness at night; increased muscular power, as shown by a disposition to stand or walk; diminution in the head-sweats; disappearance of the cranio-tabes; and improvement in the anæmia. The changes in the deformities are very slow, and from month to month almost imperceptible. When improvement once begins, however, it usually goes steadily forward, relapses being exceedingly rare.

Congenital rickets.—Infants may present at birth the characteristic deformities of rickets, and there may be found even the minute bone changes of the disease. Such cases are reported to be common in Vienna and other large cities of Europe, where mothers during pregnancy have lived under unfavourable conditions. In America, however, congenital rickets is a very rare disease. Single cases have been reported by several writers; but it must be remembered that cretinism and achondroplasia have often been improperly included under this head.

Late rickets.—Rare instances have been reported of bony deformities in all respects like those of rickets, developing in children from six to twelve years old. A number of such cases have been observed in England. I have not seen this disease, nor has a case been seen during the past twenty years at the Hospital for Ruptured and Crippled, New York, where more deformities come under observation than anywhere else in this country.

Acute rickets.—Although from time to time cases have been reported with this title, from a study of the histories it is clear that the great majority, if not all of them, were cases of infantile scurvy. It is doubtful whether, strictly speaking, there is such a thing as acute rickets.

Diagnosis.—The diagnosis of rickets is not usually difficult, and after carefully examining a case one can not often be in doubt. It is the mild cases and the early stages of the disease that are most likely to be overlooked. The most important early symptoms for diagnosis are sweating of the head, cranio-tabes, great restlessness at night, delayed dentition, and enlarged fontanel. All these, taken separately, may mean something else, but collectively they can mean nothing but rickets. In the later stages some of the characteristic deformities are usually present; the most constant are beading of the ribs, enlargement of the epiphyses of the wrists and ankles, and bow-legs.

Special symptoms, when unusually prominent, may give rise to difficulty in diagnosis. The enlargement of the head may be mistaken for hydrocephalus. The delayed dentition and large fontanel of the cretin may be mistaken for rickets. Muscular weakness may be so great, especially when affecting the legs, as to make it easy to mistake a rachitic pseudo-paralysis for actual paralysis due to a cerebral or spinal lesion. When walking is much delayed, rickets may be passed over as simple backwardness. In nearly all of the last-mentioned group of cases the diagnosis may be cleared up by a careful search for the bony changes, and by the fact that in rickets there is only a general weakness of all the muscles, and not actual paralysis of any limb or group of muscles. The greatest difficulty is usually found where the muscular symptoms are marked and the bony changes slight, as is not infrequently the case. Here the question is, whether rickets is sufficient to explain all the symptoms, or whether in addition some other condition is present. The electrical reactions will decide the question of poliomyelitis, while the presence of cerebral symptoms, exaggerated knee-jerks, and rigidity of the legs, will usually mark a cerebral birth-palsy. The bony enlargements of syphilis are not likely to be confounded with rickets, if it is remembered that the early lesions of syphilis are more like boggy infiltrations over the bones than actual swelling of the bone itself, and that when the bone is affected it is not at the extremity, but at the junction of the epiphysis and the shaft; the bone changes of late syphilis affect the shaft rather than the extremities of the long bones; where the bone is enlarged near the joint it is usually upon one side only. In syphilis there may be necrosis, while in rickets breaking down of bone is never seen. From scurvy, rickets is differentiated by the absence of marked hyperæsthesia, ecchymoses, and other hæmorrhages, the changes in the gums, and most of all by the fact that anti-scorbutic diet produces no immediate change in the symptoms. The diagnosis of rachitic curvature of the spine from vertebral caries will be considered in connection with the latter disease.

Prognosis.—Rickets *per se* is never a fatal disease. It is, however, a large factor in the mortality of the first two years, as the cachexia which it produces predisposes strongly to every form of acute disease. It is an important etiological factor in certain serious nervous conditions, especially convulsions. According to Gowers, ten per cent of the cases of epilepsy are in children who previously suffered from rickets. Rickets adds very greatly to the danger from all acute diseases of infancy, particularly those of the respiratory tract. This depends partly upon the feeble muscular power and partly upon the thoracic deformities. The encroachment upon the capacity of the lungs by a marked thoracic deformity, may in itself be enough to keep a child in a delicate condition and retard its growth. At the same time such a condition is a constant invitation to acute attacks of bronchitis or pneumonia. The effect of rickets upon the future health of the child, depends chiefly upon the presence and extent of the thoracic deformity. When this is absent, as a rule no serious after-effects are seen, and although children may remain somewhat dwarfed on account of their short legs, in other respects they may be as well as if they had never been the subjects of rickets.

Prophylaxis.—As rickets is primarily due to improper food or feeding, and secondarily to bad surroundings, it may be prevented by the observance of proper rules of feeding as laid down elsewhere, and by removing children from their faulty surroundings. Especial care should be given to the later children of a family where the earlier ones have shown even the mildest symptoms of rickets, as the predisposition is sure to increase with each successive child.

Treatment.—In considering the treatment of rickets, the natural course of the disease is to be kept in mind, viz., that active symptoms frequently continue only until the tenth or twelfth, rarely longer than the eighteenth month, and that after this time the patient suffers more from the results of the disease than from the disease itself. The most important period for treatment, therefore, and the one in which it is most effective, is from the sixth to the fifteenth month. The earlier the treatment is begun the better will be its results. Constitutional treatment after the fifteenth or eighteenth month, has very little effect upon the disease, for by this time most of the harm has been done. The course of the disease when untreated is toward spontaneous recovery, from the changes in diet and life which are usually made when children have reached the latter half of the second year. Most of the cases seen in private practice are of a mild type and recover without special treatment, often no diagnosis being made until later in life, when the bony deformities or stunted growth indicate the previous existence of rickets. The first step in treatment is to remove the cause, and is therefore to be directed to the diet and hygiene of the patient. The results will depend upon how completely these causes can be removed.

Diet.—Carbohydrates, including sugars, proprietary infant-foods, and all farinaceous substances, should be reduced to the minimum, and in some cases prohibited. So far as possible the diet should consist of nitrogenous food and fats, especially milk, cream, eggs, red meat and fresh fruit. These articles are to be given according to the rules laid down in the chapters on Infant Feeding. In addition, cod-liver oil—which in these cases may be considered quite as much a food as a medicine—should be administered as soon as the stomach will tolerate it.

Hygiene.—This is the most difficult part of the treatment. In large cities it is almost impossible to secure for rachitic patients the surroundings they require. Whenever possible, such children should be sent to the country; but where this is out of the question, much may be accomplished by frequent excursions upon the water or into the country, by keeping children as much as possible in the parks and open squares of the city, and securing plenty of fresh air in sleeping rooms. Mothers are often very much afraid of fresh air, on account of the tendency of these children to take cold. If cold sponge-baths are given every morning, much can be done to lessen this susceptibility. Sunshine, though difficult to obtain in large cities, is a most efficient therapeutic agent. The establishment of suburban hospitals and homes for these cases would do more than anything else to lessen the mortality from rickets.

In a disease which tends so uniformly to recovery when causal conditions are removed, it is difficult to estimate the real value of medicinal treatment. No one thinks of relying upon drugs alone in the treatment of rickets, and where they are used in conjunction with other means it is illogical to attribute all the improvement to the drugs employed. Those most used are cod-liver oil, phosphorus, and various preparations of lime. Regarding the value of cod-liver oil, there can be no question. While it can not be ranked as a specific in rickets, it should be given in every case unless contra-indicated by the condition of the stomach, except possibly during very hot summer weather. Phosphorus has been popularized in the treatment of rickets by Kassowitz, who regards it as a specific for the disease. I have been unable to satisfy myself, after several years' trial, that in the great majority of the cases it had any decided influence upon the course of the disease. The best results from phosphorus are obtained in the early cases, where there are cranio-tabes and marked nervous symptoms. But even here I have not seen the striking benefit reported by others. In the later stages of rickets, it has been difficult to see any special result from its use. Phosphorus may be administered either in the form of the officinal oil of phosphorus diluted with olive oil, or as Thompson's solution. The dose is gr. $\frac{1}{200}$ three times a day, given after meals; it should be continued for several months. In such doses I have never seen it cause unpleasant symptoms.

The absence of lime in rachitic bones has led to the use of various preparations of lime as remedies. Those most employed are the phosphate, the lactophosphate, and the hypophosphite. While these may be beneficial as tonics, they are not in any sense to be classed as specifics. It is probable that when lime is given in excess of the amount furnished by ordinary breast-milk or cow's milk, this excess passes through the bowels unabsorbed. Arsenic and iron are valuable in the treatment of rickets, the special indication for their use being the presence of marked anæmia. Profuse sweating may be relieved by small doses of atropine—i. e., gr. $\frac{1}{800}$, three or four times a day, to a child of six months.

Treatment of the rachitic deformities.—The deformities of the chest are less amenable to treatment than most of the others. After the third year something can be done by gymnastics to develop the chest muscles and to increase the pulmonary expansion. The employment of the pneumatic cabinet, in which it is sought to overcome these deformities by the use of rarefied air; has never been given the trial which it deserves. From the very meagre reports published, this appears to be of considerable value.

The deformity of the spine (kyphosis) may usually be overcome by postural treatment. The patient should lie upon a hard bed; no pillow should be allowed under the head, but in severe cases one should be placed beneath the back, so that the head and buttocks are slightly lower than the lumbar spine. While sitting, the shoulders should be kept back and the trunk supported. For a few minutes each day the child should be placed upon the face, and the deformity overcome by raising the buttocks while pressure is made upon the spine. In severe cases, an apparatus for giving spinal support, either by a steel brace or a plaster-of-Paris jacket, may be worn a few hours each day when the child is sitting up. Other means should be employed, especially friction and massage, to develop the spinal muscles.

In very many cases slight deformities of the extremities are outgrown when the general treatment can be properly carried out. Where these exist, the physician should take the curve of the limbs by seating the child upon a flat surface and tracing their outline with a pencil held perpendicularly (see Fig. 51). A fresh tracing should be taken once a month. If the deformity is not very great and no increase takes place, it is safe to continue with general treatment only. If the deformity is marked or if it increases in spite of the constitutional treatment, braces should be applied. Something may be done toward straightening the bones by intelligent manipulation. Walking should be discouraged until the bones are quite firm. Friction of the extremities, and even the use of electricity, will do very much to increase muscular development. The habit of sitting

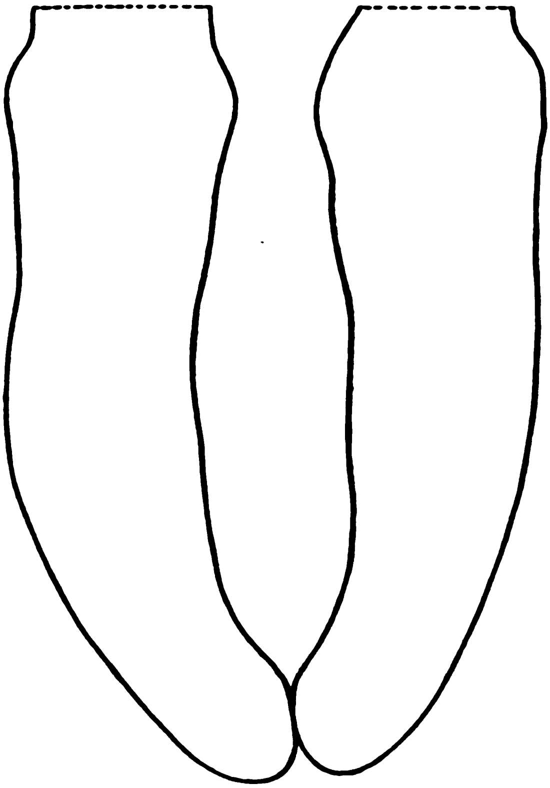


FIG. 51.—Tracing, showing the curve in a case of bow-legs.

cross-legged—a very common one in rachitic children—should be prevented, and in fact any other habitual posture, on account of the danger of increasing certain deformities. But little is to be expected from the use of apparatus for the correction of rachitic deformities after the child is two and a half years old; since at this time, and often even at two years, the bones are so firm that no amount of pressure from a steel brace will have any effect.

Without going fully into the question of the surgical treatment of rachitic deformities, for which the reader is referred to text-books of general and orthopædic surgery, I will only state that osteotomy seems to me to offer decided advantages over the other means of treating severe deformities. A vast amount of time and patience is wasted in the vain attempt to overcome very marked deformities by apparatus. The best results in osteotomy are obtained when the operation is delayed until the fourth or fifth year, by which time the bones are sufficiently firm and solid. Operations in the second year are generally unsatisfactory, and those in the third year often so, because of the bending of the bones which takes place subsequently. The deformities which require operation are bow-legs and knock-knees, less frequently the curvatures of the femur or of the bones of the forearm.

SECTION III.

DISEASES OF THE DIGESTIVE SYSTEM.

CHAPTER I.

DISEASES OF THE LIPS, TONGUE, AND MOUTH.

MALFORMATIONS.

Harelip.—This is one of the most frequent congenital deformities. It is caused by an incomplete fusion of the central process with one or both of the lateral processes from which the upper half of the face is developed. This deformity may be single or double; the fissure is never in the median line, but usually just beneath the centre of the nostril. There may be simply a slight indentation in the lip, or the fissure may extend to the nostril. Both single and double harelip—more frequently the latter—may be complicated by fissure of the palate. Double harelip is usually accompanied by a fissure between the intermaxillary and the superior maxillary bone of each side.

Cleft Palate.—This is second in frequency to harelip. It may involve the soft palate only, or the fissure may extend into the hard palate, producing a wide gap in the roof of the mouth. The most frequent form is that in which only the soft palate is affected.

For the surgical treatment of both these deformities the reader is referred to text-books upon surgery. As to the time of operation, in cases of harelip it is wisest to defer interference until the child is well started in its growth—usually the second month—and in cleft palate during the second year. The medical treatment of these cases consists in the care of the mouth and in the nutrition of the patient. The mouth in all cases must be kept scrupulously clean, but the greatest care is necessary not to injure the epithelium. A camel's-hair brush and plain lukewarm water, or a weak alkaline solution, are to be recommended. Both these deformities are exceedingly likely to be complicated by thrush. This is a serious menace to the success of any operation, and even to the life of the patient. The nutrition is always a matter of much difficulty, and a very large number of these cases die of inanition or marasmus. In cases of harelip, if the fissure is so great as to interfere with nursing, the child may be fed with a spoon or a medicine dropper until the operation can be done. In cleft palate there may be attached to the rubber nipple of the nursing bottle a flap of thin sheet rubber in such a way that it closes the fissure in the mouth when once the nipple is in place. This flap should be shaped like a leaf, one extremity being sewed to the neck of the rubber nipple and the other end left free. In many cases, both before and immediately after operation, gavage (page 64) may be resorted to with the greatest benefit and with very little inconvenience.

Congenital Hypertrophy of the Tongue.—This is usually due to disease of the lymphatics, and is to be regarded as a lymphangioma. In a few cases hypertrophy of the muscular fibres has been present. The tongue may reach an enormous size, so that it is impossible for it to be contained within the cavity of the mouth, and it may thus interfere with nursing, deglutition, and even with respiration. The treatment is surgical. Cases like the above are to be distinguished from those of enlargement of the tongue seen in sporadic cretinism. In this disease the tongue is considerably enlarged and may protrude slightly from the mouth, but it is rarely, if ever, large enough to cause other symptoms. It diminishes notably under treatment with the thyroid extract.

Bifid Tongue.—These cases are extremely rare. Brothers has reported to the New York Pathological Society a case of cleft tongue in a child of one month. There was, in addition, a fissure of the soft palate.

Tongue-Tie.—This deformity is due to such a shortening of the frenum that it is impossible to protrude the tongue to a normal extent. It differs considerably in degree in different cases. In some, the tongue can not be advanced beyond the gums. Tongue-tie may interfere with articulation, and even with sucking. The treatment consists in liberating the tongue by dividing the frenum with scissors and completing the operation with the finger nail. This should be done in every case unless the child is a bleeder. In many cases the mother may think the tongue tied when the frenum is of normal length.

Bifid Uvula.—This is not very uncommon. It usually occurs in connection with cleft palate, but is occasionally seen when there is no other deformity present. It may be complete or partial, and it does not of itself require treatment.

DISEASES OF THE LIPS.

Herpes.—Herpes labialis is an exceedingly common affection in children, occurring in acute febrile diseases, particularly pneumonia, and sometimes alone. It is the familiar "fever sore" or "cold sore" of domestic medicine. The appearance is similar to herpes in other parts of the body. There is first a group of vesicles, then rupture and the formation of crusts. It is often quite difficult to cure on account of the disposition of children to pick the lip with the fingers. Although it heals without treatment, recovery is facilitated by the use of some antiseptic lotion, such as dilute boric acid, followed by a dusting powder of zinc oxide and boric acid. This treatment is generally more successful than the use of ointments. Young children should wear mittens at night, to prevent picking at the crusts.

Eczema of the Lip.—This is an exceedingly common condition, and a very troublesome one. The vermilion border is dry and rough, and prone to deep cracks or fissures. These are usually seen at the angles of the mouth or in the median line. When severe they are exceedingly painful, bleed freely, and are the cause of very great discomfort, especially in the cold season. The lips should be covered at night by simple ointment, and this should be used as much as possible during the day. Where deep fissures form, they should be touched with burnt alum, or with the solid stick of nitrate of silver. Syphilitic fissures are considered with the symptoms of that disease.

Perlèche (French, *perlècher = to lick*).—This name was first given by Lemaistre, in 1886, to a form of ulceration occurring usually at the angle of the mouth. It begins in most cases as a small fissure, which, by constant licking and irritation, to which there is usually added infection, may produce an intractable ulcer of considerable size. It often resembles the mucous patch of hereditary syphilis. The ulcer is of a grayish colour, is quite painful, and is associated with considerable swelling of the lip. It lasts from two to four weeks. The treatment is the same as in simple fissure—viz., the use of burnt alum or nitrate of silver, and covering the part with bismuth or oxide of zinc.

DISEASES OF THE TONGUE.

Epithelial Desquamation.—This is a disease of the lingual epithelium, which is characterized by the appearance upon the dorsum or margin of the tongue, of circular, elliptical, or crescentic red patches, with gray margins which are slightly elevated. It is sometimes improperly called psoriasis of the tongue. It is quite a common condition.

The beginning of the disease is not often seen. It is stated first to appear as a white or gray patch, like thickening of the epithelium. These patches enlarge quite rapidly, and are followed by detachment of the epithelium and the formation of bright red areas, which are the parts denuded of epithelium. As usually seen, there exists upon the tongue from two to half a dozen of these red patches surrounded by a gray border, which is about one twelfth of an inch wide, and slightly elevated. The outline of the patch is nearly always crescentic (see Fig. 52). From day to day the configuration of the patches changes; the gray lines advance across the tongue from side to side, or from base to tip, disappearing as they reach the border or the extremity. They are followed by the red patches, and as the old ones fade away new ones form and run the same course. The white border seems to be made up entirely of epithelium.

The red patches are of a bright colour nearest the border, gradually shading off into the normal colour of the tongue. Only the epithelium is involved, the deeper structures being unaffected. The duration of the disease is indefinite; it usually lasts for months, and often for years. Guinon reports several cases which recovered during an intercurrent attack of measles or scarlet fever.

The cause is unknown. The condition occurs rather more frequently in females than in males, and Gubler has reported an instance of several members of the same family being affected. Most of the cases are seen in infancy and early childhood. The condition has been thought to depend upon nearly every disease of this period. Parrot believed that it was always syphilitic, but this view has been effectually disproved by subsequent observation. The disease is not accompanied by pain, salivation, or by other symptoms of stomatitis, and it is of little practical importance. Its symptoms are so characteristic that it can hardly be mistaken for any other condition. Treatment is unnecessary.

Two other forms of epithelial desquamation have been observed, both much more rare than that described. In one of these the red denuded portion occupies the margin of the tongue, while the centre is gray or white; the irregular wavy outline which separates the two suggests strongly an outline map, and the condition is sometimes called the "geographical tongue." In another variety nearly the whole organ may be uniformly red, from loss of the epithelium, there being no borders or patches. Both these varieties are of much shorter duration than the more common form, usually lasting only a few weeks.*

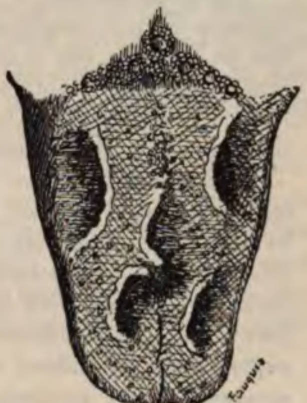


Fig. 52.—Epithelial desquamation of the tongue. (Guinon.)

Glossitis.—Inflammation of the tongue is not very common in children. It is usually of traumatic origin. The injury may be due to biting the tongue in a fall or in an epileptic seizure. Glossitis is sometimes excited by the irritation of a sharp tooth, causing a wound which may be the avenue of infection; or it may result from taking into the mouth irritant or caustic poisons. In a small number of cases no cause can be found. The symptoms are marked swelling of the tongue, so that it may protrude from the mouth; and it may even be so great as to cause severe dyspnœa. There are also profuse salivation, difficulty in swallowing and in articulation, and often considerable local pain. There may be a rise of temperature to 102° or 103° F. The treatment consists in the use of fluid food, which in severe cases may be introduced through the nose by means of a catheter. Ice may be used externally, or, better still, pieces of ice should be kept in the mouth continually. If there is obstruction to respiration, and in all severe cases, scarification should be done on the dorsum of the tongue along the side of the raphé.

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* For a fuller description and literature of the subject, see Guinon, Revue Mensuelle des Maladies de l'Enfance, 1887, p. 385; and Gautier, Revue Médicale de la Suisse, Romande, October and November, 1881.

The acute swelling of the tongue and lips occurring in some cases of urticaria may be mentioned in this connection. This is a rare condition in children, but it may develop rapidly and to such a degree as to cause alarming symptoms. The treatment consists in the use of ice locally, free purgation by salines, and in extreme cases needle punctures to relieve the œdema.

Tongue-swallowing.—This term is used to describe a rare condition seen in infants, in which the tongue is turned backward into the pharynx, so as to obstruct respiration. It may be drawn quite into the œsophagus. Several marked cases have been collected by Hennig.* While most frequently occurring with paroxysms of pertussis, tongue-swallowing has been seen in other diseases. This should not be forgotten as one of the explanations of sudden asphyxia in a young infant. The conditions necessary to its production are a somewhat relaxed organ or a long frenum. In none of the fatal cases reported, however, had the frenum been divided. In some weak infants, falling back of the tongue, so that its base partly covers the epiglottis, produces asphyxia, precisely as it occurs in adult life under full anæsthesia. The recognition of the condition is a very easy one, and its treatment is to relieve the obstruction by drawing the tongue forward by the finger or forceps.

Ulcer of the Frenum.—The friction against the sharp edges of the lower central incisors frequently causes an ulcer of the frenum in infants. I have never seen it in older children. It usually occurs in pertussis, but is seen in other conditions. In some it appears to be produced by friction of the teeth during nursing from the breast or bottle. It is more often seen in children who are delicate or cachectic than in those who are healthy and well nourished. The ulcer may be confined to the frenum, or it may extend quite deeply into the tongue. It is usually about one fourth of an inch in diameter, and of a yellowish-gray colour. When not readily cured by touching with alum or nitrate of silver, the child may be fed by gavage for several days, or the teeth may be covered by a bit of absorbent cotton.

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* Jahrbuch für Kinderheilkunde, xi, 299.

ALVEOLAR ABSCESS.

This is common in children, especially among the class of hospital and dispensary patients, in whom little or no attention is given to the care of the teeth. It causes severe pain and acute swelling, which may be limited to the gum, or it may involve to a considerable extent the periosteum of the jaw, and even cause swelling of the whole side of the face. If there is retention of pus, there may be quite severe constitutional symptoms, such as a chill and high temperature; but in most of the cases these are wanting. The abscess usually opens spontaneously into the mouth, but it may open externally if the molar teeth are the ones affected. It may even lead to necrosis of the jaw. If its site is the upper jaw, the pus may find its way into the nasal cavity or into the maxillary sinus.

The treatment is, in the first place, prophylactic. This requires attention to the teeth to prevent decay, and the removal of old carious fangs, which are a constant menace to the health of the child in more ways than one. The free use of the toothbrush and some antiseptic mouth-wash will, in the great majority of cases, prevent the occurrence of this disease. It is important that the abscess be opened early and free drainage secured. If there is a carious tooth it should be drawn.

DIFFICULT DENTITION.

The place of dentition as an etiological factor in the diseases of infancy is one which has given rise to much discussion. From a very early period the view has descended, that a large number of the diseases occurring between the ages of six months and two years are due to difficult dentition. The list of such diseases is a long one, but year by year it has been shortened as one after another has been shown to depend upon other causes, dentition being only a coincidence.

At the present time many good observers deny that dentition is ever a cause of symptoms in children; some even going so far as to say that the growth of the teeth causes no more symptoms than the growth of the hair. Without doubt the usual mistake made in practice is in overlooking serious disease of the brain, kidneys, lungs, stomach, and intestines, because of the firm belief that the child was "only teething." The physician who starts out with the idea that in infancy dentition may produce all symptoms usually gets no further than this in his etiological investigations. Although I strongly believe that the importance of dentition as an etiological factor in disease has been in the past greatly exaggerated, and although I once held the opinion that simple dentition never produced symptoms, I have been compelled by clinical observations to change my opinion upon this subject; and I am now willing to admit that, particularly in delicate, highly nervous children, dentition may produce many reflex symptoms, some even of quite an alarming character.

Speaking from general impressions, not from statistics, I should say that in my experience about one half of the healthy children cut their teeth without any visible symptoms, local or general; in the remainder some disturbance is usually seen, and though in most cases it is slight and of short duration, it may last for several days or even a week. The symptoms most commonly seen are disturbed sleep, or wakefulness at night and fretfulness by day, so that children often sleep only one half the usual time. There is loss of appetite, and much less food than usual is taken. There is often, but not always, an increase in the salivary secretion, a slight amount of catarrhal stomatitis, and a constant disposition on the part of the child to stuff the fingers into the mouth. The bowels are often constipated or there may be slight diarrhœa. The thermometer may show a slight elevation of temperature to 100° to 101·5° F. The weight may remain stationary for a week or two, and there may even be a loss of a few ounces. The duration of these symptoms in most cases is but a few days, and they require no special treatment. If the food is forced beyond the child's inclination, attacks of indigestion with vomiting and diarrhœa are easily excited.

Symptoms more severe than the above are rare in healthy children, but are not infrequent in those who are delicate or rachitic. In such susceptible children, even so slight a thing as dentition may be the cause, or at least the exciting cause, of quite serious symptoms. Often there is some other factor in the case, such as bad feeding or feeble digestion. In delicate or rachitic children there may be seen the symptoms already mentioned as occurring in healthy infants, but in greater severity; and in addition there may be severe attacks of acute indigestion. Occasionally there is an elevation of temperature to 102° or 103° F., lasting usually only two or three days, and accompanied by no symptoms except almost complete anorexia. Convulsions which could fairly be attributed to dentition I have seen but once; they are more apt to occur in rachitic children. There are certain cases of eczema in which the symptoms undergo a distinct exacerbation with the eruption of each group of teeth. As regards almost all the other diseases which are commonly attributed to dentition, I believe that it is a delusion to trace them to this cause.

The physician should watch a child carefully, and examine it frequently, to be sure that he is not overlooking some serious local or constitutional disease before he allows himself to make the diagnosis of difficult dentition. Probably in ninety-five per cent of the cases in which symptoms are present, they are due to some cause other than dentition. When, however, symptoms such as any of those mentioned disappear immediately when the teeth come through, and when we see them repeated four or five times in the same child with the eruption of each group of teeth, and accompanied by red and swollen gums, I think we can not escape the conclusion that dentition is a factor in their production, though perhaps not the only one.

In the treatment of this condition drugs occupy but a small place. It should be remembered that infants are at this time in a peculiarly susceptible condition as regards the digestive tract, and attacks of indigestion, and even severe diarrhœa, are readily excited from slight causes, especially from overfeeding. Special care should be exercised in this respect. The strength of the food should be reduced, as well as the amount given. The poor appetite indicates a feeble digestion, which should not be overtaxed. As attacks of bronchitis and acute nasal catarrh are readily induced, even slight exposure should be guarded against. The nervous symptoms, when severe, may be relieved by the use of moderate doses of the bromides and phenacetine, better than by opiates. All soothing syrups should be discountenanced. All the various devices for making dentition easy are a delusion. In a small number of cases lancing the gums is of decided value. I have myself seen marked and undoubted relief given by it. This is likely to be the case where the gums are tense, swollen, and very red, with the teeth just beneath the mucous membrane. To press a tooth through the gum by simply rubbing gently with the finger covered with sterile gauze is frequently much more effective than an incision. It is seldom, however, that the relief expected is seen from any of these measures.

CATARRHAL STOMATITIS.

This is characterized by redness and swelling of the mucous membrane, and by increased secretion of the salivary and the muciparous glands of the mouth. It usually involves a large part of the mucous membrane.

Etiology.—Catarrhal stomatitis may result from traumatism. This injury may be mechanical, or due to heat or any irritant accidentally taken into the mouth. It frequently occurs at the time of the eruption of a tooth. It complicates measles, scarlet fever, diphtheria, influenza, and many other infectious diseases. In these cases, and in many others, the disease is probably due to direct infection.

Lesions.—The lesions are essentially the same as in catarrhal inflammations of other mucous membranes. There are congestion with desquamation of epithelial cells, and sometimes the formation of superficial ulcers. The process may be a very superficial one, or it may extend to the submucous tissue.

Symptoms.—The mucous membrane is intensely injected, all the capillaries are dilated, and small hæmorrhages easily excited. The mucous membrane is swollen, this being most apparent over the gums or about the teeth. There may be some swelling of the lips. The mouth seems hot, and the local temperature is certainly increased. There is considerable pain, as shown by fretfulness, but particularly by the disinclination to take food; infants, though evidently hungry, either refusing the breast or bottle altogether, or dropping it after a few moments. The increase in secretion is sometimes marked, so that the saliva pours from the mouth, irritating the lips and face and drenching the clothing. In other cases the saliva is swallowed. On close inspection there may be seen swelling of the muciparous follicles, and even the formation of tiny cysts from the accumulation of secretion within them (Forchheimer). The tongue is usually coated, the edges reddened, and the papillæ prominent. In febrile diseases, such as typhoid, etc., we may get an accumulation of dead epithelium with the formation of cracks and fissures of the tongue, and the lips may present a similar condition. The neighboring lymphatic glands are slightly enlarged and tender. The constitutional symptoms accompanying simple stomatitis are not severe, but some disturbance is almost always present. There may be derangement of digestion with vomiting, and even a mild attack of diarrhœa. In the majority of cases the disease runs a short course, recovery taking place in a few days when the primary cause is removed. In very delicate children it may be prolonged, and from the interference with nutrition may even lead to serious consequences.

Treatment.—The mouth and teeth should be kept clean. Food is more acceptable if given cold. In very severe cases, where food is refused, gavage may be resorted to three or four times daily. In all cases children may be given ice to suck. This is refreshing, both on account of the cold and from the relief to the thirst. The mouth should be kept clean with a solution of boric acid, ten grains to the ounce, or an alkaline solution, such as Dobell's, diluted with an equal amount of cold boiled water; or simply water may be used. In the severe forms, where there is much swelling and slight catarrhal ulceration, astringents are required. In my experience alum is the best; this may be applied in the form of the powdered burnt alum mixed with an equal amount of bismuth, or in solution, ten grains to the ounce, with a swab or brush. Where ulcers are slow in healing and very painful the powdered burnt alum may be applied directly.

HERPETIC STOMATITIS.

Synonyms: Aphthous, vesicular, follicular stomatitis.

In this form of stomatitis we have the appearance first of small yellowish-white isolated spots and subsequently the formation of superficial ulcers. These ulcers are first discrete, but may coalesce and form others of considerable size. It is a self-limited disease, usually running its course in from five days to two weeks.

Etiology.—Very little is as yet positively known regarding the cause of herpetic stomatitis. It is not common in the first year, but after that is very frequently seen throughout childhood. It occurs in the strong as well as in the delicate. It is often associated with some disturbance of the stomach, and occasionally with dentition. I have adopted the term *herpetic* because the condition is analogous to herpes of the lips and face, the difference in appearance being due chiefly to location. It is apparently caused by something which acts upon terminal nerve filaments.

Lesions.—The generally accepted opinion is that there is first a vesicle, followed by a death of epithelial cells covering it, and then a superficial ulcer. The white appearance is due to the fact that the ulcers, being on a mucous membrane, are always moist. These ulcers may extend superficially, but never deeply; they heal quickly with the formation of new epithelial cells, leaving no cicatrices. Herpetic stomatitis is always associated with more or less catarrhal inflammation.

Symptoms.—The disease is characterized by local and general symptoms. The former are quite indefinite—general indisposition, loss of appetite, and slight fever. The local symptoms consist in the development of small, shallow, circular ulcers, usually coming in successive crops. While most frequent at the border of the tongue and the inside of the lips, they may be found upon any part of the mucous membrane of the mouth or the pharynx. There may be only half a dozen present, or the mouth may be filled with them. They are first of a yellowish colour, and on an average about one-eighth of an inch in diameter. By the coalescence of several smaller ones there may form patches of considerable size, sometimes nearly covering the lips. The older ulcers are apt to have a dirty grayish colour, and in places may look not unlike a diphtheritic membrane. The smaller ones are surrounded by a red areola, and when healing the margin is of a bright-red colour. Their appearance is often more like that of an exudation upon the mucous membrane than an excavation into it. The other symptoms are much the same as in catarrhal stomatitis, but usually of greater severity. The pain is particularly intense, it being often difficult to induce children to take anything in the form of food. The tongue is frequently coated, but there is never the foul breath of ulcerative stomatitis. The duration of the disease is from one to two weeks, and, if the child is in good condition, complete recovery takes place even without any special treatment. In badly nourished children the disease may last for two or three weeks; relapses may occur, and the condition may interfere very seriously with the child's nutrition.

Treatment.—This is the same as in catarrhal stomatitis, with the addition that to each one of the ulcers finely powdered burnt alum should be applied with a camel's-hair brush. If this is not effective, the solid stick of nitrate of silver may be used. The ulcers will usually yield rapidly to this treatment. In my experience, drugs given with the purpose of affecting the lesion in the mouth have been without benefit.

ULCERATIVE STOMATITIS.

Ulcerative stomatitis is believed to occur only when teeth are present. It is characterized by an ulcerative process, beginning at the junction of the teeth and the gum, and extending along the teeth; it occasionally involves other parts of the mouth, but never spreads beyond the buccal cavity.

Etiology.—A form of ulcerative stomatitis is produced by certain metallic poisons, especially mercury, lead, and phosphorus; but all these are now rare. Ulcerative stomatitis also occurs in scurvy; and it seems probable that an allied disturbance of nutrition, with spongy, swollen gums, precedes some other forms of ulcerative stomatitis. Bad surroundings and improper food act as predisposing causes; for the disease is quite common in hospital and dispensary patients, although rare in private practice. Local causes of some importance are want of cleanliness of the mouth and teeth and the presence of carious teeth. Conditions which produce a lowered vitality of the gums act as a predisposing cause, and infection as an exciting cause of the disease. The constant clinical features of ulcerative stomatitis and the occasional occurrence of epidemics indicate a specific cause.*

Lesions.—The disease may begin at any part of the mouth, but most frequently upon the outer surface of the gum along the lower incisor teeth. From this point it extends behind the teeth, and from the incisors to the canines and molars, usually of one side only; but it may involve the entire gum of both jaws. From the gums the process may spread to the lips, affecting the fold of mucous membrane between the gum and the lip, and also to the inner surface of the cheek, especially opposite the molar teeth, where large ulcers often form. In neglected cases the disease may extend into the alveolar sockets, the teeth loosening and falling out. The periosteum of the alveolar process may be involved, and even superficial necrosis of the jaw may occur, as has happened in several cases that came under my observation.

Ulcers similar in appearance may also be present in other parts of the mouth—i. e., on the soft palate or the tonsils, sometimes even when the gums are not involved.

Symptoms.—The first things noticed are the very offensive breath and the profuse salivation. It is usually for one of these symptoms that the patient is brought for treatment. On inspection of the mouth, there are seen in the mild cases, swollen, spongy gums of a deep-red or purplish colour, which bleed at the slightest touch. There is a line of ulceration, usually along the incisor teeth, most marked in front, which may extend to any or to all of the teeth; sometimes it affects only the gum along the molar teeth, the incisors escaping. At the junction of the teeth and gum is seen a dirty, yellowish deposit, on the removal of which free bleeding takes place. The diseased parts are very painful, and the child cries and resists any attempt at examination. In the more severe cases and in those of longer duration the teeth are loosened, sometimes being so loose that they can be picked from the gum. There may be necrosis of the jaw, and even a loose sequestrum may be found. In these cases the ulceration along the gums is deeper, and there may be ulcers in the cheek opposite the molar teeth, or inside the lip. The swelling may be so great that the teeth are almost covered; this is seen particularly in the scorbutic form. The saliva pours from the mouth, adding greatly to the discomfort of the patient. Beneath the jaw are felt the large, swollen lymphatic glands, which are painful and tender to the touch, but show no tendency to suppurate. The tongue is somewhat swollen, and shows at the edges the imprint of the teeth; it has a thick, dirty coating.

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* The most important bacteriological investigations of this disease are those of Bernheim and Pospischill (Jahrbuch für Kinderheilk., xlvi, 434). Of thirty cases studied, in all but two, both mild ones, they found two micro-organisms associated; sometimes one and sometimes the other predominated. One was a fusiform bacillus often bent, with sharp ends, somewhat resembling the diphtheria bacillus but larger; it was stained by methyl blue and decolourized by Gram. The other was a spiral form. It is interesting to note that similar bacteria were found by Miller in carious teeth, and by Vincent in ulcero-membranous tonsillitis (see page 308).

The disease is attended by little or no fever or other constitutional symptoms. The general condition of these patients is often poor, and there may be quite a marked cachexia. Other forms of stomatitis may be associated, and it should not be forgotten that the gangrenous form may follow.

When not recognised or not properly treated, ulcerative stomatitis may last for months. When properly treated it tends in all recent cases to rapid recovery, usually in a few days. No deformity of the mouth is left, the only untoward results being shrinking of the gum, sometimes loss of some of the incisor teeth, and more rarely a superficial necrosis of the alveolar process. All these are quite uncommon. Ulcerative stomatitis can hardly be confounded with any other form, and not only should a diagnosis of the lesion be made, but the condition upon which it depends should, if possible, be discovered; scorbutus, particularly, should not be overlooked.

Treatment.—The first thing to be done is to remove the cause. When dependent upon metallic poisoning the source should be discovered. Scorbutic cases should have the usual anti-scorbutic diet. Cleanliness of the mouth is of great importance, and this may best be accomplished by the use of peroxide of hydrogen diluted with from one to four parts of water. It should be followed by plain water, and repeated several times a day. In other cases an astringent solution of alum, five grains to the ounce, or a mouth-wash of chlorate of potash, three grains to the ounce, may be employed. The only objection to the last mentioned is the pain which it usually produces.

The specific remedy for ulcerative stomatitis is chlorate of potash. The best method of administration is to give two grains, or one-half teaspoonful of a saturated solution, largely diluted, every hour during the day for the first twenty-four hours and subsequently every two hours; when improvement occurs the dose may be still further reduced. Marked benefit is usually seen in one or two days even in cases that have lasted for several weeks. If the case does not yield readily to this treatment there is probably disease at the roots of the teeth, and when loose these should be removed, and the jaw examined to see if there is necrosis. Occasionally when there is no disposition to heal, the shreds of necrotic tissue should be carefully removed, and burnt alum or nitrate of silver applied.

The constitutional and dietetic treatment in all these cases should be the same as that employed in scurvy—i. e., plenty of fruit, fresh vegetables, and sometimes the internal administration of mineral acids, especially aromatic sulphuric acid. Iron is indicated in most of the cases.

Ulceration of the Hard Palate.—This is usually seen in the first few weeks of life, but may occur in any child suffering from marasmus. The primary cause may be the injury inflicted in cleansing the mouth. In other cases it is due to the friction of the rubber nipple, or something else which the child is allowed to suck. In still others it is apparently produced by the habit of tongue-sucking frequently observed in these young infants. The appearances are quite characteristic: there is found, rather far back upon the hard palate, usually in the middle line, a superficial ulcer, from a fourth to a half inch in diameter. There are no signs of acute inflammation. Thrush may coexist, but it has no relation to the production of the disease. Spontaneous recovery usually occurs in from one to three weeks, provided the cause can be removed. In children suffering from marasmus these ulcers are very intractable, and in many instances their cure is practically impossible. It is therefore especially important to prevent, if possible, their formation by care in cleansing the mouth, and in avoiding the other causes referred to. When ulcers have appeared they should be treated as cases of herpetic stomatitis.

THRUSH.

Synonyms: Sprue; German, Soor; French, Muguet.

Thrush is a parasitic form of stomatitis characterized by the appearance upon the mucous membrane, usually of the tongue or of the cheeks, of small white flakes or larger patches. It is common in infants of the first two or three months, and in all the protracted exhausting diseases of early life.

Etiology.—The exact class to which the vegetable parasite which produces thrush belongs has not yet been definitely settled. Robin's opinion was long accepted that it was the *oïdium albicans*; the view of Grawitz, that it is the *saccharomyces albicans*, is now more generally adopted. If a little of the exudate from the mouth is placed upon a slide and a drop of liquor potassæ added, the structure of the fungus is readily seen. With the low power of the microscope there can be made out fine threads (the mycelium) and small oval bodies (the spores). With a high power the threads can be seen to be made up of a number of shorter rods, at the ends of which the spore formation takes place (Fig. 53). The mycelium is produced from the spores. The spores of this fungus are of very common occurrence in the atmosphere. It is difficult or impossible for thrush to develop upon a healthy mucous membrane. Its growth is favoured by slight abrasions, such as are often produced by rough methods of cleansing the mouth; also by catarrhal stomatitis, a scanty salivary secretion and want of cleanliness. The fungus may grow in a medium of any reaction, but best in one which is slightly alkaline or neutral. The nature of the process which it produces is in all probability a sugar fermentation, the acid reaction of the mouth being the result of the growth rather than its cause. Infection may come from another patient by means of a rubber nipple or a cloth which has been used for the infected mouth, from the nipple of the nurse, or directly from the air. The disease is an exceedingly common one in foundling asylums, in all places where many young infants are crowded together, and where cleanliness of mouths, bottles, etc., is neglected. It is especially frequent in children suffering from malnutrition, marasmus, or other wasting diseases, and in those who have harelip, or any deformity of the mouth.

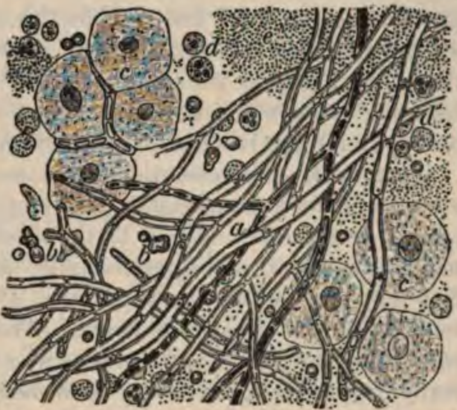


FIG. 53.—Thrush fungus (highly magnified). *a*, mycelium; *b*, spores; *c*, epithelial cells from the mouth; *d*, leucocytes; *e*, detritus. (Jaksch.)

Lesions.—According to Forchheimer, the spores lodge between the epithelial cells and gradually separate the different layers. This occurs before the formation of the white pellicle. Later the disease spreads on the surface of the mucous membrane, and also penetrates the deeper structures. It may invade the blood-vessels and cause thrombosis or even be carried to distant parts. Although the saccharomyces albicans is commonly found upon flat epithelium, its growth is not confined to it. It usually begins at many distinct points upon the mucous membrane, and gradually spreads until coalescence takes place; a continuous membrane may be thus formed. No pus is produced by the process.

The usual seat is the tongue, the inside of the cheeks, and the hard palate, but not infrequently it involves the lips, the tonsils, the pillars of the fauces, and the pharynx. Further extension in the digestive tract than this is rare, although the stomach, and even the intestines, may be invaded. I have seen it but once or twice in the œsophagus and never in the stomach, and I know of but two reported cases in this country in which thrush has been found there. Cases involving the œsophagus and the stomach appear from reports to be much more common in Europe. In three cases in the Babies' Hospital the saccharomyces albicans has been found in the lungs of infants suffering from broncho-pneumonia.

Symptoms.—The essential symptoms of thrush are the appearance upon the mucous membrane of the mouth—usually beginning upon the tongue or the inner surface of the cheek—of small white flakes which resemble desposits of coagulated milk, but which differ from them in the fact that they can not be wiped off. If forcibly removed, they usually leave a number of bleeding points. There may be only a few scattered patches, or the mouth and pharynx may be covered. The mouth is generally dry, the tongue coated; food may be refused on account of pain, and there may be some difficulty in swallowing. The other symptoms depend upon the conditions with which the thrush is associated.

Diagnosis.—This is rarely difficult. The deposit may be mistaken for coagulated milk, but is distinguished by the features just mentioned. When existing upon the pharynx and fauces it has been confounded with diphtheria, although this mistake can hardly be made if all the facts of the case are taken into consideration—the age of the patient, the involvement of the lips and tongue, the dry mouth, the absence of glandular enlargement, etc. In any case of doubt the examination of the deposit under the microscope at once reveals its true nature.

Prognosis.—Thrush is not in itself a dangerous disease, except in the very rare instances where it may obstruct the œsophagus, and this can hardly occur except in a condition of exhaustion which is necessarily fatal. In a feeble and delicate infant, thrush may be a serious complication by interfering with the taking of sufficient nourishment. With proper treatment most of the cases involving only the mouth are readily cured.

Treatment.—Thrush may be prevented in almost every case by due attention to cleanliness of the mouth, rubber nipples, bottles, cloths, etc. All rubber nipples should be kept in a solution of borax or salicylate of soda, and the child's mouth should be cleansed several times a day. On no account should a feeding-bottle be passed from one child to another.

In the treatment of the disease the essential things are cleanliness, and the use of some mild antiseptic mouth-wash. The best routine treatment is to cleanse the mouth carefully after every feeding or nursing with a solution of borax or bicarbonate of soda, and to apply twice a day a 1-per-cent solution of formalin. Occasionally better results follow the use of nitrate of silver, a 3-per-cent solution applied twice daily. All application should be carefully made, so as not to injure the epithelium. The best method of cleansing is by the finger wrapped in absorbent cotton, or by a swab. Applications to be especially avoided are those mixed with honey or any syrup. In several hospital cases the disease seemed to be prolonged by the irritation of the rubber nipple of the feeding-bottle. In such it has been our practice to feed by gavage for two or three days, as all cases improved much more rapidly when this was done.

GONORRHŒAL STOMATITIS.

There has been described by Dohrn and Rosinski a form of stomatitis in the newly born, due to a gonorrhœal infection. This is not likely to take place unless the epithelium has been removed. The infection in all cases occurred from the mother. The lesion consists in the formation of yellowish-white patches upon the tongue or hard palate—regions in which the epithelium is liable to be injured by rough attempts at cleansing the mouth. There may be other evidences of gonorrhœal infection, especially ophthalmia. The diagnosis rests upon the discovery of the gonococcus in the exudate. In all the cases cited the general health was not affected, and recovery followed in the course of a week or ten days.

The treatment consists in thorough cleanliness and in the application of a saturated solution of boric acid, as in thrush.

SYPHILITIC STOMATITIS.

The buccal symptoms of hereditary syphilis are important both from a diagnostic and therapeutic standpoint. The most frequent lesions are fissures, ulcers, and mucous patches. Fissures are found upon the lips, most frequently at the angle of the mouth, and are usually multiple. They may be quite deep and cause frequent hæmorrhages. Mucous patches are superficial ulcers developing from papules which form upon the mucous or muco-cutaneous surfaces. In cases of acquired syphilis in children the primary sore may be seen upon the tongue, the lip, or the tonsil. All these symptoms are more fully considered in the chapter on Syphilis.

DIPHTHERITIC STOMATITIS.

In severe cases of diphtheria the membrane is found not only upon the pharynx and tonsils, but it may appear anywhere upon the buccal mucous membrane or the lips. It is questionable whether the diphtheritic process ever begins in the mucous membrane of the mouth, or is ever limited to this part. In my own experience diphtheritic stomatitis has always been associated with deposits upon the tonsils and pharynx. It is seen only in the severest cases, and in those which, from other conditions present, are almost necessarily fatal. Bearing in mind the above points, it can hardly be mistaken for any other variety of stomatitis, although not infrequently the mistake is made of regarding as diphtheritic, cases of herpetic stomatitis in which the ulcers have coalesced. The treatment, so far as the mouth is concerned, consists in cleanliness by frequent gargling or syringing with a saturated solution of boric acid Forcible removal of the membrane is not to be advised.

GANGRENOUS STOMATITIS—NOMA.

Synonym: Cancrum oris.

The term noma is used to designate all forms of spontaneous gangrene occurring in children, which involve mucous membranes or mucocutaneous orifices. The most frequent situation being the mouth, noma and gangrenous stomatitis are often used synonymously. Noma may, however, affect the nose, external auditory canal, vulva, prepuce, or anus. It is a rare disease, and usually terminates fatally.

Etiology.—Noma is seldom seen outside of institutions for children, where small epidemics are not uncommon. It is usually secondary to some of the infectious diseases, most frequently following measles, and next to this scarlet fever, typhoid, or whooping-cough. While it may occur at any age, most of the cases are in children under five years, and in those of poor general condition. Noma seldom attacks parts previously healthy. In the mouth it may be preceded by catarrhal, or more often by ulcerative stomatitis; in the auditory canal, by a chronic otitis media. There seems little doubt that the disease is contagious. In 1899 I saw five cases in a single ward, all beginning in the auditory canal, which were apparently produced by the use of the same syringe to clean the ears without proper disinfection. All these children were suffering from whooping-cough at the time.

The results of bacteriological studies of noma are not uniform nor as yet conclusive. In the gangrenous tissue pyogenic cocci and putrefactive bacteria are usually abundant. In the border zone, and extending into the adjacent healthy tissue, bacilli have been found which are regarded by Babes, Bartels, Schmidt, and others as the specific organism of the disease, although they do not altogether agree in their descriptions. In cases reported by Freymuth, Petruschky, and in one of my own, bacilli closely resembling, if not identical with, diphtheria bacilli were found. Others have ascribed the disease to streptococci. It is not improbable that more than one micro-organism, or even other agents, may under certain conditions have the power of causing this form of gangrene.

Lesions.—The process is one of slowly spreading gangrene. In most of the cases there are thrown out inflammatory products in quite large amount, but there is little or no tendency to limitation of the disease. This usually advances steadily until death occurs. In a small number of cases a line of demarcation finally forms, and the slough separates, leaving a large area to be partially filled in by granulation and cicatrization. Other infectious processes are liable to accompany the disease, particularly broncho-pneumonia.

Symptoms.—The constitutional symptoms are not usually severe until the local disease has existed for several days. Then those of marked prostration and sepsis develop, sometimes quite rapidly. The temperature is usually elevated to 102° or 103° F., and sometimes to 104° or 105° F. There are dulness, apathy, feeble pulse, muscular relaxation, and very often diarrhœa. Before death the temperature may be subnormal.

Of the local symptoms, often the first to attract attention is the odour of the breath; sometimes it is the dusky spot on the cheek or lip. On examination of the mouth, there usually is found upon the gum or inside of the cheek a dark, greenish-black necrotic mass, surrounded by tissues which are swollen and œdematous, so that the cheek or lips may be two or three times their normal thickness. Externally the parts are tense and brawny from the swelling, this infiltration always extending for some distance beyond the gangrenous part. As the process extends, the teeth loosen and fall out; there may be necrosis of the alveolar process of the jaw and perforation of one or both cheeks or lower lip. Extensive sloughing of the face may take place, usually upon one side, sometimes upon both, giving the patient a horrible appearance, as shown in Fig. 54. In this patient the process began in the right cheek, subsequently involving the left; perforation occurred in both cheeks, and before death a large part of the face was gangrenous. The odour from a severe case is very offensive, and, in spite of all efforts at disinfection, it may fill the ward or even the house. Pain is rarely severe, and in many cases it is absent.. Extensive hæmorrhages are rare.

I have notes of seven cases in which noma affected the ear, being preceded by chronic otitis media in every instance. The disease began in the deeper structures of the canal, the first symptom noticed usually being a nodular swelling just beneath the ear, crowding the lobe upward. Shortly afterward there appeared the dirty brown discharge with a gangrenous odour; later, the gangrenous circle surrounding the meatus. This gradually extended, until in some cases the whole side of the face and head were involved. A probe could readily be passed into the cranial cavity. All these cases ended fatally.

The usual duration of the disease is from five to ten days. If recovery takes place, there is first seen a line of demarcation; then the slough is thrown off, and granulation and cicatrization begin, but require a long time, usually leaving an unsightly deformity.

The prognosis is grave, about three-fourths of the cases proving fatal. The results depend not only upon the disease itself, but upon the condition of the patient with which it is associated.

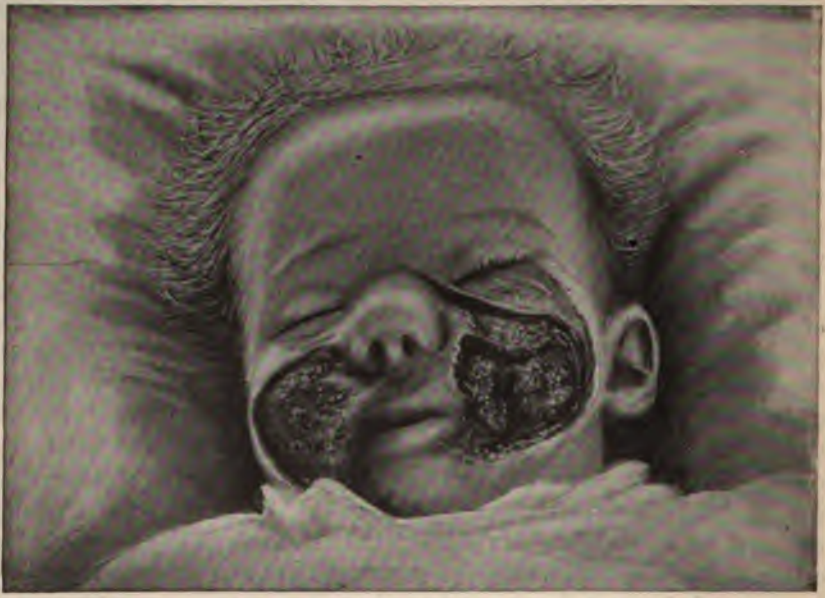


FIG. 54.—Gangrenous stomatitis, following measles. (From a photograph lent by Dr. Henry Moffat.)

Gangrenous stomatitis can hardly be mistaken for any other form of disease occurring in the mouth, and early recognition is of great importance, since only early treatment is likely to be successful.

Treatment.—Much can be done to prevent the disease by careful attention to all the milder forms of stomatitis, particularly to the ulcerative variety. Frequent and thorough cleansing of the mouth in all acute infectious diseases is a part of the treatment which is too often neglected. This should be a matter of routine in every severe illness in a young child. Recognising the malignant nature of gangrenous stomatitis, its treatment should be radical from the very outset. Of the measures which have been proposed, that which seems to offer the best chance of arresting the process is excision with cauterization. This should be done under anæsthesia. In excising, one should go some distance into tissues apparently healthy, for the reason that the process has always advanced farther in the subcutaneous tissues than in the skin. The edges of the wound should then be thoroughly cauterized, best by the Paquelin cautery. Of the other means employed, the use of strong carbolic acid immediately followed by alcohol is probably the best. This is to be used after excising, or curetting the necrotic tissue. Cases have been reported in which the use of anti-streptococcus serum, and also the diphtheria antitoxin, have appeared to arrest the disease. The mouth should be kept as clean as possible by the use of peroxide of hydrogen. The general treatment should be supporting and stimulating. As the possibility of contagion exists, every case should be isolated.

CHAPTER II.

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

ACUTE pharyngitis may exist as a primary disease, or with any of the infectious diseases, particularly scarlet fever, measles, diphtheria, or influenza. Secondary pharyngitis will be considered in connection with these different diseases.

Certain children have a constitutional predisposition to attacks of acute pharyngitis, and contract it upon the slightest provocation. In some of them there is a strongly marked rheumatic diathesis. Attacks of acute pharyngitis often follow exposure. In many cases they are associated with acute disturbances of digestion. All of the above causes probably act by producing local and general conditions favourable to the development of micro-organisms already present in the mouth. They are cases of auto-infection. The bacteria most frequently associated with severe attacks are streptococci, less frequently staphylococci and pneumococci.

In acute catarrhal pharyngitis the inflammation may involve the entire mucous membrane of the tonsils, fauces, uvula, posterior and lateral pharyngeal walls, or any part of it. It may exist alone, or in connection with a similar inflammation in the rhino-pharynx or in the larynx. In the beginning there is seen an acute erythematous blush, usually involving the entire pharynx. This may entirely subside after twenty-four hours, or it may be followed by the usual changes of acute catarrhal inflammation—dryness, swelling, and œdema. Later there is increased secretion of mucus, and finally there may be muco-pus. Occasionally slight hæmorrhages are present.

There is pain at the angle of the jaws, which is increased by swallowing, also a sensation of dryness and roughness in the pharynx, and often an irritating cough. There may be slight swelling of the neighbouring lymphatic glands. The constitutional symptoms in young children are often severe. Not infrequently there is a sudden onset with vomiting, and a rise of temperature to 102° or even 104° F. These symptoms are usually of short duration, frequently less than twenty-four hours, and in two or three days the patient may be entirely well. In other cases the pharyngitis may be accompanied or followed by laryngitis.

Acute primary pharyngitis is to be distinguished from scarlet fever, measles, and influenza. A positive diagnosis from scarlet fever is impossible until a sufficient time has elapsed for the eruption to appear, and the patient should be closely watched for the first sign of this. If scarlet fever is prevalent, a child with the symptoms of severe pharyngitis should at once be isolated while waiting for the diagnosis to be settled. There is commonly less difficulty in excluding measles because of the presence of Koplik's sign on the buccal mucous membrane, and the accompanying catarrh of the eyes and nose. Influenza is recognised only by the greater severity of the constitutional symptoms and the prevalence of an epidemic.

The first step in the treatment of acute pharyngitis is to open the bowels freely by means of calomel, castor oil, or magnesia. The child should be kept in bed, and the diet should be fluid, or, in the case of infants, the amount of food should be much reduced. Pieces of ice may be swallowed frequently for the relief of pain and thirst. Internally there may be given two grains of phenacetine every four hours to a child of three years. It is important at the outset to induce free perspiration. The disease is not serious, and the indications are to make the child as comfortable as possible during the short attack. I have seen but little benefit from the use of aconite, although for years I saw it used as a routine treatment.

UVULITIS.

Acute inflammation of the uvula, with swelling and œdema, occurs as a part of the lesion in acute pharyngitis. In rare instances the uvula may be the principal or the only seat of inflammation. Huber (New York) has reported two cases, one of which is unique. An infant ten months old was apparently well until two hours before it was seen, when there was noticed a constant irritating cough, accompanied by considerable gagging. Later there could be seen in the mouth a prominent red mass, the enlarged and elongated uvula. It was accompanied by paroxysms of cough, which interfered both with nursing and deglutition. The general symptoms were quite alarming. The uvula was found to be fully one inch long and half an inch wide, red and œdematous; in other respects the throat was normal. The symptoms were relieved by multiple needle punctures and the use of ice. In such conditions the greatest relief is often afforded by the application of adrenalin, or its use as a spray or gargle.

ELONGATED UVULA.

Probably this is primarily a congenital condition. It is increased by repeated attacks of acute or subacute inflammation. The degree of elongation varies in different cases; in some it may reach an inch in length. According to Bosworth, only the mucous membrane is involved in the elongation. The symptoms are those of local irritation, especially a cough upon lying down, and the sensation of a foreign body in the pharynx. In some cases it may be a reflex cause of asthma, or, more frequently, of catarrhal spasm of the larynx. The diagnosis is very easily made by inspecting the throat. The treatment consists in grasping the tip of the uvula with forceps and cutting off the excess with the scissors, or a uvulatome. Care should be taken not to cut off too much of the uvula, or severe hæmorrhage may occur.

RETRO-PHARYNGEAL ABSCESS.

Two distinct varieties are seen: (1) the so-called idiopathic abscesses which belong to infancy, and (2) abscesses secondary to caries of the cervical vertebræ.

RETRO-PHARYNGEAL ABSCESS OF INFANCY.—All of the later investigations regarding this disease go to show that primarily it is not a cellulitis, but a suppurative inflammation of the lymph nodes (lymphatic glands) with a surrounding cellulitis. Jules Simon has described the retro-pharyngeal lymph nodes as forming a chain on either side of the median line between the pharyngeal and the prevertebral muscles. These nodes are said to undergo atrophy after the third year, and in some cases to disappear entirely. Retro-pharyngeal abscess—or more properly retro-pharyngeal lymphadenitis, since the process does not invariably go on to suppuration—is probably never primary, but secondary to infectious catarrhs of the pharynx, and is set up by the entrance of pyogenic bacteria, usually the streptococcus. Its pathology is the same as the more frequent suppurative inflammation of the external cervical lymph nodes, with which it is sometimes associated. Usually only a single node is involved, but sometimes two or three are affected, and these may be situated upon opposite sides. I have seen retro-pharyngeal lymphadenitis so severe as to give rise to marked local symptoms, although it did not go on to suppuration. This is rare; Kormann's observations, however, show that swelling of these glands in diseases of the mouth and throat is very much more common than is generally supposed. Similar abscesses from suppurative inflammation of other lymph nodes in the neighbourhood of the pharynx may occur. I have seen one situated between the epiglottis and the base of the tongue.

Etiology.—These cases almost invariably occur in infancy. Fully three-fourths of those that have come under my observation have been in patients under one year. Bokai (Buda-Pesth) reports that of sixty cases observed, forty-two occurred during the first year, eleven during the second year, and only seven at a later period. The primary disease is usually a severe rhino-pharyngitis, or an attack of epidemic influenza, but rarely it occurs as a sequel of scarlet fever or measles. In six hundred and sixty-four cases of scarlet fever, Bokai noted retro-pharyngeal abscess in seven cases. After measles it is even more rare. Retro-pharyngeal abscess usually occurs in winter or spring, on account of the prevalence of the diseases upon which it depends. It is seen in children previously robust, but more often in those who are delicate and who in consequence are prone to severe catarrhal affections.

Symptoms.—The early symptoms in most cases are merely those of an ordinary rhino-pharyngeal catarrh. After this has subsided the temperature may remain slightly elevated, often for a week or more, before local symptoms are noticeable. Sometimes, without any distinct history of previous catarrh, there are seen quite high temperature, from 102° to 104° F., loss of flesh, and prostration. A careful examination may be required, and sometimes observation for a day or two, before the explanation of these constitutional symptoms is discovered. In other cases the early constitutional symptoms are so slight as to escape notice, and the physician is summoned on account of the local symptoms, usually the dyspnœa, which in a short time may assume an alarming character. The duration of the inflammatory process before abscess forms is generally five or six days, but it may be several weeks. The temperature is invariably elevated, usually from 100° to 103° F.; occasionally it may be 104° or 105° F., with symptoms of prostration seemingly out of all proportion to the local disease, but which are to be explained by the tender age and feeble resistance of the patient.

The first local symptom may be a sudden attack of dyspnœa severe enough to cause asphyxia. This is due to the pressure forward of the abscess which encroaches upon the opening of the larynx. Usually before it occurs the breathing is noisy, especially during sleep, and on account of the obstruction to nasal respiration the patient breathes with the mouth open. The mouth may be dry, or there may be a copious secretion of pharyngeal mucus. The dyspnœa is in most cases greater on inspiration, and in some it is noticed only then, expiration being normal. The dyspnœa is sometimes increased by attempts at swallowing. The degree to which deglutition is interfered with depends upon the size and the position of the tumour. It is more difficult when the tumour is low down. The child may find it impossible to swallow, and in consequence may refuse to nurse; or the difficulty in nursing may depend upon the nasal obstruction. Sometimes there is regurgitation of food through the nose or mouth. The voice is usually nasal. Generally there is no hoarseness, but a peculiar short cry which is quite characteristic. There may be complete aphonia; often there is a short, dry cough. In many of the cases a tumour is to be seen externally, just below the angle of the jaw and in front of the sterno-mastoid muscle; exceptionally this may be more prominent than the internal swelling. The head is thrown back in order to relieve the pressure upon the larynx, and is held somewhat rigidly. In one or two cases I have noticed torticollis as an early symptom.

A positive diagnosis is made by an examination of the throat. On inspection there is seen a distinct bulging of the lateral wall of the pharynx, usually a little above the base of the tongue. The swelling may be so great as to crowd the uvula to one side and nearly fill the pharynx. It is rarely if ever in the median line. There is usually redness of the mucous membrane and œdema of the uvula and of the adjacent parts. On digital examination the swelling is made out even better than by inspection. It may be situated so low down as not to be visible at all. In the early stage there may be felt only a localized induration or a somewhat diffuse swelling, but by the time the swelling is large enough to produce marked symptoms, fluctuation can generally be discovered.

Prognosis.—When left to itself the abscess may open into the pharynx, the pus being swallowed or expectorated. The cavity may close rapidly by granulation, and in a few days the patient be entirely well; or the abscess may refill. It is rare for much burrowing to occur. In young or very delicate infants the constitutional symptoms may be so severe that the child continues to fail even after the evacuation of the abscess, and, gradually sinking, dies usually from broncho-pneumonia. In other children a fatal result is generally due to the fact that the disease was not recognised.

Death may occur from asphyxia due to pressure upon the larynx, to œdema of the glottis, or from rupture of the abscess into the air passages, especially if this occurs during sleep. Carmichael, Bokai, and others have reported deaths from ulceration into the carotid artery, or one of its large branches. Carmichael's patient was only five weeks old. The general mortality is from five to ten per cent; many deaths are owing to a failure to make the diagnosis. Gautier has collected ninety-five cases, with forty-one deaths. In my experience death has most frequently resulted from late broncho-pneomonia; in one case it was due to a secondary retro-œsophageal abscess.

Diagnosis.—Retro-pharyngeal abscess is to be suspected if in an infant there is difficulty in swallowing, noisy dyspnœa, mouth-breathing, and the head drawn backward. A positive diagnosis is possible only by a digital examination of the pharynx. The mistake most often made is, that the physician, called to a young child suffering from great dyspnœa, has jumped at a diagnosis of laryngeal stenosis, and forthwith performed tracheotomy or intubation, without taking the trouble to get the history or to make a careful examination of the pharynx.

Many such cases are reported in which the child has died during the operation or immediately afterward, the autopsy first revealing the nature of the disease. A sudden attack of dyspnœa like that caused by the rupture of an abscess might be produced by the lodgment of a foreign body in the pharynx or larynx. A digital examination would aid in the diagnosis. I once saw in an infant a sarcoma of the pharyngeal lymph glands which gave an external and internal tumour exactly like that of a retro-pharyngeal abscess.

Treatment.—Before the abscess has pointed, hot applications should be made to the throat to relieve the symptoms and to hasten the formation of pus, since resolution is not to be expected. Spontaneous opening should never be waited for, on account of the danger of the rapid development of serious symptoms from pressure or œdema, or of suffocation from an opening into the air passages, especially during sleep.

As soon as the diagnosis is made the case should be carefully watched, and as soon as well-marked fluctuation is detected, the pus should be evacuated. External incision has its advocates, and in a few cases, when the tumour is chiefly external, it offers some advantages; but as a routine operation the internal opening is, to my mind, much to be preferred. In opening through the mouth the patient should be seated in an upright position and the head firmly held. The introduction of a mouth-gag may cause asphyxia; but a tongue depressor may be used, and a bistoury which has been guarded to its point plunged into the abscess at its thinnest portion and the incision made toward the median line. The head should then be bent forward, to allow the pus to escape through the mouth. It is well to insert the finger into the cavity and break down any septa; for after a simple puncture the abscess may refill. Incision, although usually easy, in some cases may be quite difficult on account of the swelling and the small pharynx of the infant. For the past few years I have adopted the plan of opening these abscesses with the finger nail, a procedure simple, efficient, and free from danger. While the patient is held as above described, the wall of the abscess is perforated where it points, by the nail of the forefinger which has been sharpened to a cutting point. I have seldom seen a case in which this was difficult. The amount of pus evacuated is from one drachm to half an ounce. In the majority of cases no after-treatment is required. The relief of the dyspnœa and dysphagia is immediate, and recovery rapid.

Retro-pharyngeal Abscess from Pott's Disease.—This form is rare in comparison with that just described, and under three years of age it is extremely so. These abscesses are usually larger, and the amount of pus contained may be from four to eight ounces. They form very much more slowly, often lasting for months, and as with other secondary abscesses, the constitutional symptoms are seldom severe. The swelling is frequently in the median line, and is not so circumscribed as in the idiopathic cases. The pus often burrows along the spine for several inches.

The symptoms of Pott's disease of the cervical region are usually present for several months before the appearance of the abscess. Sometimes the abscess precedes the deformity, and it may be the first intimation of the existence of bone disease. The local symptoms resemble those of the idiopathic cases, but they develop more slowly, and sudden attacks of fatal asphyxia are very rare. External swelling is usually seen, and it may be quite large, extending almost from one ear to the other, forming a distinct collar. On digital exploration there may be found an irregularity of the anterior surface of the cervical vertebræ, and occasionally a marked angular prominence.

When left to themselves these abscesses may open externally in front of the sterno-mastoid muscle just below the jaw, sometimes nearly as low as the clavicle; they may rupture internally into the pharynx, the œsophagus, or the air passages; or they may burrow a long distance in front of the spine. Death may result from pressure upon the larynx, or from rupture into the larynx, trachea, or pleura; all these, however, are rare. The abscesses not infrequently refill after they are evacuated, and occasionally a discharging sinus is left for many months.

Treatment.—These abscesses should be opened as soon as they are large enough to give rise to local symptoms. The external incision just in front of the sterno-mastoid muscle is generally to be preferred to opening through the mouth, since it gives better drainage, and the after-treatment is more easily carried on; and a sinus opening externally is less objectionable than one opening into the pharynx.

ADENOID VEGETATIONS OF THE VAULT OF THE PHARYNX.

This is a very common condition and one much neglected by the general practitioner. It is the source of more discomfort and the origin of more minor ailments than almost any other pathological condition of childhood.

There is a mass of lymphoid tissue situated at the vault of the pharynx which in structure closely resembles the tonsils. It is often spoken of as the "pharyngeal tonsil." Like the faucial tonsils, this may become greatly hypertrophied, so as to form a tumour large enough to fill the rhino-pharynx completely. These tumors have a broad attachment which is sometimes more to the roof, and sometimes more to the posterior wall of the pharynx. The term *adenoid vegetations* was given to them by Meyer, who first described them in 1868. In infancy these growths are soft, vascular, and spongy; in older children they become firm, dense, and more fibrous. Their appearance is well shown in Fig. 55. Adenoid vegetations are associated with hypertrophy of the faucial tonsils in about one-third the cases. Growths large enough to cause decided nasal obstruction may in time produce changes in the facial bones amounting to positive deformity. The bony palate is dome-shaped or even acutely arched; the dental arch of the upper jaw becomes almost V-shaped. Deformities of the thorax also occur, which will be described with the Symptoms.

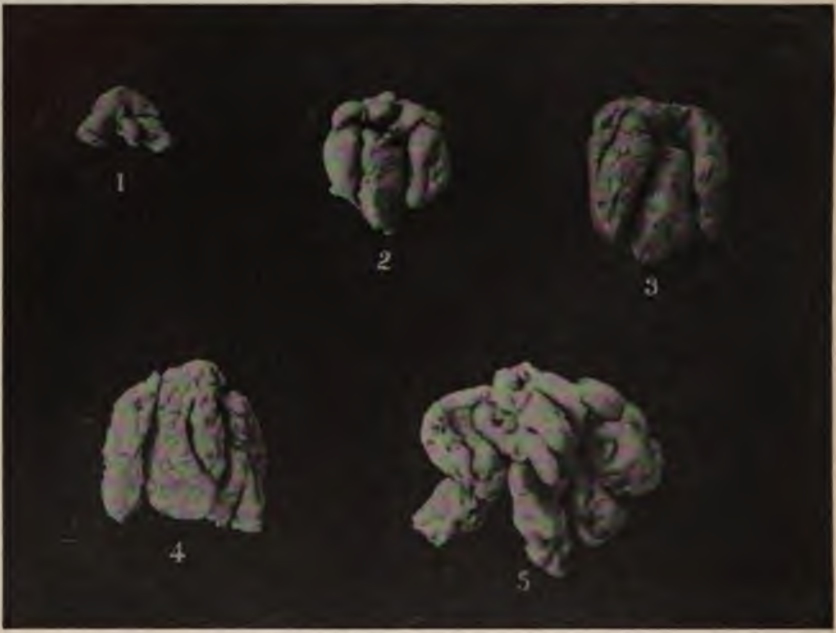


FIG. 55.—Adenoid vegetations, natural size.

(1) From child eight months old; (2) from child twenty-two months old; (3) from child two and one half years old; (4) from child two and one half years old; (5) from child three years old. With the exception of (5) all were removed with a single sweep of the curette.

Although the growths represented are somewhat larger than the average for the ages mentioned, just such ones are constantly met with in practice.

Etiology.—The constitutional condition described elsewhere as "lymphatism," sometimes called the *status lymphaticus*, is the one with which adenoid growths are very frequently associated. Very often, however, they are the most marked manifestation of the condition. I have frequently known every one of a large family of children to be affected, and often the parents have suffered from the same disease. There can be no doubt regarding the influence of heredity in the production of adenoids. In many cases they are congenital. Rachitic children are somewhat oftener affected than others, but no connection with syphilis has been traced. Much interest has lately been awakened regarding the relation of adenoid growths to tuberculosis. Of 945 cases collected by

Lewin in which specimens of adenoids were examined, tuberculosis was present in 5 per cent. Though this proportion is no doubt much higher than will be found in private practice, the fact is an important one; for it is highly probable that this is the channel of infection in not a few cases of tuberculous meningitis. Adenoids are most common in damp, changeable climates. Their first symptoms often follow an attack of measles, scarlet fever, or diphtheria. The repeated head colds are more often a result than a cause of the condition.

Symptoms.—The symptoms of adenoid growths are usually first noticed when children are from eighteen months to three years old; but they may be present almost from birth. I have in several instances seen them to a marked degree in infants only a few months old. The symptoms generally increase in severity as age advances, being always better in summer and worse in winter, until the age of six or seven is reached. The chief symptoms are those which relate to (1) chronic rhino-pharyngeal catarrh, (2) mechanical obstruction, (3) deafness, (4) general malnutrition and anæmia, (5) reflex nervous phenomena.

The rhino-pharyngeal catarrh shows itself by a persistent nasal discharge, frequently recurring acute attacks, or head colds, during the entire winter season. In susceptible children these attacks are often the beginning of a bronchitis, which may keep a young child indoors almost the entire winter.

The obstructive symptoms are inability to blow the nose, mouth-breathing constantly or only during sleep, and a nasal voice. The difficulty in breathing is increased when the child lies upon the back. In consequence of this, children sleep in all sorts of positions—lying upon the face, sometimes upon the hands and knees, and often toss restlessly about the crib in the vain endeavour to find some position in which respiration is easy. The attacks of dyspnœa at night may amount almost to asphyxia, and are the explanation of many of the so-called night-terrors from which children suffer. When the obstruction has existed from infancy there are often deformities of the chest; these are most marked in rachitic subjects. The most frequent one consists in deep lateral depressions of the lower part of the chest, with a prominence of the sternum—the familiar pigeon-breast (Fig. 56). The deformity is due to interference with pulmonary expansion.

Some impairment of hearing exists in a large proportion of the cases. Blake (Boston) found this to be true in 39 out of 47 cases examined; in 35 of these marked improvement in the hearing followed removal of the adenoid growths. Deafness may be due to tubal catarrh or to otitis. Often a history is given of several attacks of suppurative otitis.

The reflex symptoms associated with adenoid growths are many. One of the most important is catarrhal spasm of the larynx, or the familiar spasmodic croup. In my experience the majority of young children who are subject to such attacks have adenoids, the removal of which is frequently followed by their complete cessation. The crowing attacks of newly born infants are believed by Eustace Smith always to depend upon adenoids. I have not been able to satisfy myself upon this point. Other respiratory symptoms associated with adenoids are intractable coughs, frequently of a spasmodic character, without bronchial symptoms or signs; and persistent hoarseness, lasting for months or even years, and recurring every cold season. Both these conditions are often cured by the removal of the adenoids after all other treatment has been without effect. To these growths bronchial asthma also is very frequently due. Their relation to incontinence of urine is often an intimate one; the two coexist in a large number of patients, and in a certain number removal of the adenoids cures the incontinence. Headaches are very common; stammering may be present; chorea and even epileptiform seizures have been attributed to adenoids, although I have never seen either.

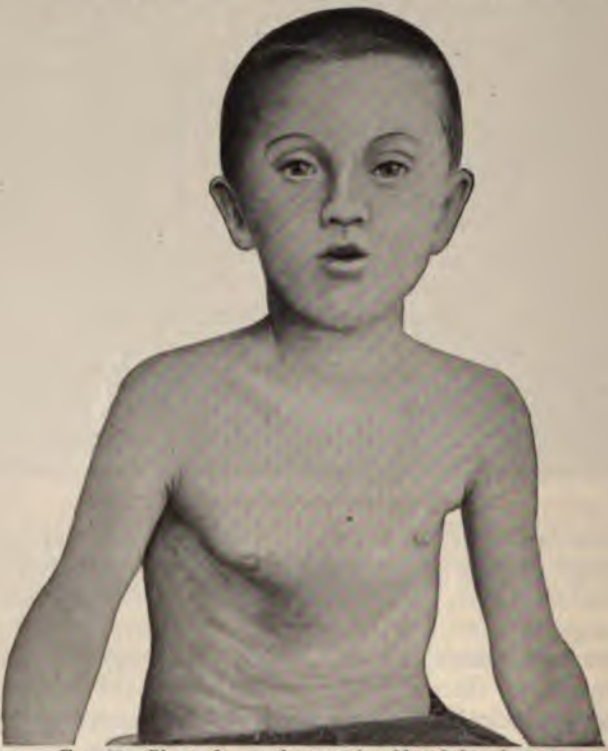


FIG. 56.—Pigeon-breast due to adenoids of the pharynx.

The general health of patients suffering from adenoids may be impaired from lack of oxygen due to obstructed respiration, from loss of sleep, and from confinement to the house, necessitated by attacks of bronchitis or head colds. Marked anæmia is often present. In old and neglected cases of a severe character, children may be stunted in growth, and their facial expression dull and stupid. They are languid, listless, often depressed, and this with their deafness frequently causes them to be regarded in schools as children who are somewhat deficient mentally.

These patients are always better in summer and worse in winter. The natural course of the growths if left to themselves is to increase up to a certain point, and then to remain stationary until puberty, when they usually undergo a certain amount of atrophy. This, with the marked increase in the capacity of the rhino-pharynx which occurs at this time, results in a disappearance of the most aggravated symptoms. A removal to an elevated region with a dry atmosphere will often result in a relief from all the symptoms, and a diminution in the size of the growth, but unless such a change in residence is permanent the symptoms are liable to return. Under ordinary circumstances there is little or no tendency to spontaneous recovery. Children with adenoid growths contract diphtheria and tuberculosis more easily than do others, and in them attacks of diphtheria, scarlet fever, measles, and whooping-cough are all likely to be more severe.

Diagnosis.—In a well-marked case the condition is usually evident from the history, and can scarcely be overlooked. The intractable nasal catarrh, upon which no treatment, local or general, has more than a temporary influence, the mouth-breathing, the disturbed sleep, and the slight deafness—all are characteristic. In some even of the marked cases, attention may be drawn to the larynx, bronchi, or ears as the seat of disease. At other times the patients come for treatment on account of the general symptoms—the nervous depression, the headaches, or the anæmia. In rare cases the leading symptom may be epistaxis. The symptoms do not always depend upon the size of the growth, for in a small throat quite a small growth may cause very marked symptoms.

Although the history is in most cases clear, only an examination can make us certain that an adenoid growth exists. The best method of examination consists in a digital exploration of the pharynx; but this requires a little practice before it is very satisfactory. The head is steadied by one hand, and the forefinger of the other is passed up behind the palate. The growth is ordinarily felt as an irregular, granular, soft, velvety mass, or sometimes as a firm tumour completely blocking the passage; and the finger, when withdrawn, is almost invariably covered with blood. By anterior rhinoscopy, after the use of cocaine, the growth can often be seen.

Treatment.—The disappearance of adenoid growths by absorption is possible only when they are small. This may be aided by the prolonged use of guiaquin, one grain three times a day, or the syrup of the iodide of iron, fifteen drops three times a day; but most of all by removal to a warm, dry climate for the winter season. All possible means should be employed to prevent these patients from taking cold, such as proper clothing, cold sponging, cod-liver oil, etc. With the larger growths these methods may improve the catarrhal symptoms, but can hardly affect the mechanical ones. The reduction of tumours of any considerable size by local applications is, I think, a delusion; every case that has come to my notice has been relieved only by operation.

Removal of adenoid growths is indicated: (1) When the obstructive symptoms—habitual mouth-breathing, disturbed sleep, nasal voice, chest deformities, etc.—are marked; (2) for a chronic nasal discharge, constantly recurring head colds, particularly when these tend to attacks of bronchitis or laryngitis; (3) where there is asthma or repeated attacks of catarrhal spasm of the larynx; (4) with deafness, chronic otitis, or repeated attacks of acute otitis; (5) for certain nervous symptoms—enuresis, stammering, chorea, headaches, night terrors, etc. Although striking improvement is not infrequent, one should be cautious about promising too much from operations where these nervous conditions exist; also in an older child when there is deafness or asthma.

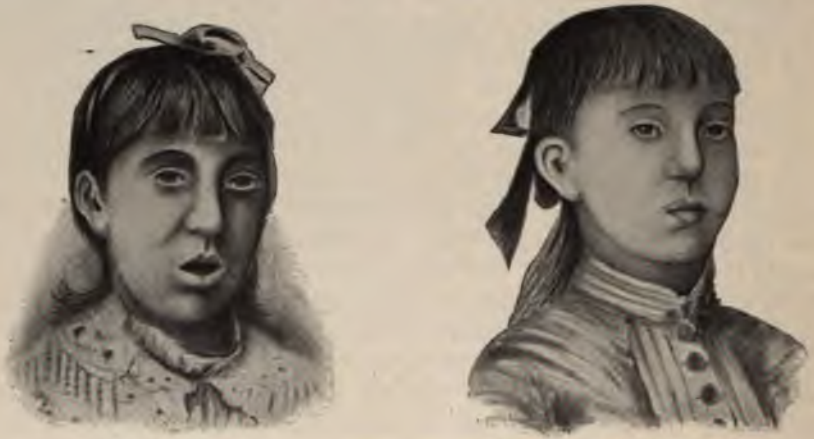
The preferable time for operation is the spring or early summer, in order that during the warm months the mucous membranes may have an opportunity to regain their normal condition; however, operation may be done at any time except during attacks of acute catarrh. Unless the symptoms are very marked, I prefer to defer operation until a child is at least two years old.

Removal of adenoids by scraping with the finger nail is possible only when the growths are very soft; it is at best a very uncertain method, and is not to be advised. Except in the case of children under two or two and a half years old, where the growths are generally small and the patients easily handled, I prefer to operate with general anæsthesia: first, for the sake of thoroughness; secondly, to avoid the fright and pain which so bloody an operation is apt to cause in those who are older, and especially in very nervous children. So many deaths from operations for adenoids or tonsils under chloroform have now been reported (Hinkel in 1898 collected eighteen, and a number have since been added), and so many narrow escapes have occurred that have not been published, that chloroform anæsthesia should, I think, be given up altogether. My preference is for ether; in older children it may with advantage be preceded by nitrous oxide, and sometimes with such patients the nitrous oxide alone may be used, but this is not to be advised with very young children. Deep anæsthesia is not usually necessary, and if the semi-erect position is assumed it increases the danger of the entrance of blood or portions of the growth into the larynx, which might cause fatal asphyxia.

The only instruments required are a mouth-gag, like that used for intubation, and modified Gottstein's curettes, which should be sharp. The physician should have several sizes with different curves to suit the size and attachment of the growth and the capacity of the throat. Many of the instruments used for young children are too large, the smaller ones being more easily manipulated and less liable to do harm.

If no anæsthetic is used, the patient's arms are pinioned to the side by two or three turns of a sheet around the body, the head firmly held by an assistant, upon whose lap the patient sits, as for the operation of intubation. With anæsthesia there is an advantage in using the sheet in the same way. During operation I prefer to have the patient raised to a little more than a half-reclining posture and the head firmly steadied. This position gives the operator a decided advantage over the low-head position, which is necessary when chloroform is used. After the introduction of the gag, the pharynx should be carefully explored with the finger to determine the size and position of the growth. The tongue is then depressed by the left forefinger, while with the right hand the curette is carefully passed high up behind the soft palate until it meets the nasal septum. The handle of the curette is grasped as one holds a pen. The cut is made with a downward movement, depressing the blade and elevating the handle of the curette, it being given a lever-like motion by the action of the wrist. When the curette is grasped with the entire hand, and the full arm used with simply a downward movement, the pharyngeal mucous membrane is often stripped down for some distance below the growth, but not cut off. Care should be taken to keep the blade well against the bony wall of the vault and posterior pharyngeal wall, and the handle in the median line, and not to employ too much force. The majority of the growths encountered in ordinary practice, such as Nos. 1, 2, and 3 in Fig. 55, can be removed with one sweep of the curette, the mass usually coming away in a single piece. Others may require the instrument to be used two or three times. The patient is now turned face downward until most of the hæmorrhage has ceased. Then the cavity should be explored with the finger to ascertain whether the removal has been complete. The forceps (Lowenberg's and various modifications) are quite unnecessary, and in unskilled hands are capable of doing much harm. One unfamiliar with their use may easily tear away pieces of the uvula, soft palate, pharyngeal wall, and even portions of the Eustachian tubes.

The entire operation consumes in most cases less than a minute. Hæmorrhage is always abundant, and seems alarming to one who sees it for the first time. In an average case it amounts to one or two ounces, but generally ceases in a few minutes. A child should not pass from the physician's observation until all bleeding has stopped. It often happens that the patient swallows the growth, a disappointing but not a serious accident. The child should be kept quiet, preferably in bed, for twenty-four hours; and in the house for five or six days, unless the weather is warm. No after-treatment is necessary, or at most a spray of a weak antiseptic solution. Recurrences are extremely rare, except after incomplete operations, such as those performed with the finger nail, etc. The improvement is usually in proportion to the severity of the previous symptoms. It generally begins in a few days, sometimes at once, though the full benefit may not be seen for a month. The breathing becomes freer, the sleep more quiet; the mouth may soon be habitually closed; voice and hearing improve, and the benefit to the general health is soon apparent. The pallor, listlessness, and inattention disappear, and a rapid increase in weight often follows. The entire appearance of the child may in a few months be transformed (Figs. 57, 58).



Before operation. Three months after operation.

Figs. 57 and 58.—Adenoid vegetations of the pharynx; girl twelve years old. (Hooper.)

Dangers and Accidents from Operation.—While it is rare that any accidents of a serious nature are met with, it should not be forgotten that they may occur. Undue laceration of the parts may result from a bungling operation particularly with too large curettes or with the forceps. Hæmorrhage may be excessive or even fatal. In over two hundred operations I have had but one case of serious hæmorrhage. A fatal result is exceedingly rare. Newcomb in 1893 could find but four examples. Hæmorrhage may be continuous after operation, or secondary, in which case it almost invariably occurs within twenty-four hours. It is important, therefore, that the patient be kept under observation for that time. Bleeding is best controlled by injecting into the rhino-pharynx through the nostrils one or two drachms of hydrogen peroxide, full strength, or, this failing, a solution of suprarenal extract may be used in the same manner. As a last resource plugging of the posterior nares may be resorted to. In all cases the patient should be kept absolutely quiet.

Occasionally an acute attack of bronchitis or otitis occurs after operation; and in a few recorded instances acute meningitis, simple or tuberculous, has followed. The danger of asphyxia from the entrance of blood or the tumour into the larynx has already been mentioned.

The danger from chloroform anæsthesia is due not so much to the nature of the operation as to the condition of the patient. It is now well established that all children in whom the condition known as lymphatism is marked, bear chloroform very badly.

CHAPTER III.

DISEASES OF THE TONSILS.

THE tonsils* are lymphoid structures closely resembling Peyer's patches, but, instead of having a flattened surface, the lymphoid tissue in the tonsil is folded upon itself, forming quite deep depressions—the tonsillar crypts. These crypts, like the surface of the tonsils, are lined by epithelial cells. They contain lymphoid cells, desquamated epithelium, particles of food, and bacteria. Under normal conditions the tonsils take no part in absorption from the mouth. When, however, their epithelium is rarefied or removed, the tonsils absorb with very great facility every sort of poison which the mouth may contain. Such poisons are taken up by the lymphatics, and through them reach the general circulation.

Acute inflammation of the tonsils, like that of the pharynx, occurs regularly in diphtheria, scarlet fever, and measles, less frequently in the other infectious diseases. The secondary forms will be considered with the diseases with which they are associated.

Acute catarrhal tonsillitis, or inflammation of the mucous membrane covering the tonsils, occurs as part of the lesion in acute pharyngitis, but very rarely is seen alone.

Croupous Tonsillitis.—This is a more severe form of inflammation than catarrhal tonsillitis. It involves the mucous membrane of the tonsils, the tonsillar crypts, and to a greater or less degree the whole structure of the tonsil. Fibrin is poured out upon the surface in sufficient quantity to form a distinct pseudo-membrane, which usually covers the tonsils, but in primary cases it does not extend beyond them. In most cases both sides are affected. The exudation sometimes begins in isolated dots, like a follicular tonsillitis, which afterward coalesce to form a continuous patch. The membrane is usually of a yellowish gray colour. It can often be completely removed with the swab. The constitutional symptoms are generally marked and resemble those of follicular tonsillitis.

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* See Hodenpyl, American Journal of the Medical Sciences, March, 1891, on Anatomy and Physiology; Packard, Philadelphia Medical Journal, April 21, 1900, on Infection through the Tonsils.

The disease is differentiated with certainty from diphtheria only by means of cultures, which should be made in every case. (See Diagnosis of Diphtheria.) Croupous tonsillitis is nearly always due to the streptococcus. Though never severe when it occurs as a primary affection, it may be very serious when it is secondary to measles or scarlet fever. Its clinical features are more fully considered under the head of Pseudodiphtheria.

Ulcero-membranous Tonsillitis.—This is an inflammation somewhat resembling croupous tonsillitis, but it is often unilateral and associated with superficial ulceration. The tonsil is covered with a dirty yellowish exudation, which may be mistaken for diphtheria. There is superficial necrosis, and when this tissue is wiped away with a swab, bleeding occurs. The disease is further distinguished by the swollen lymph nodes at the angle of the jaw, and by the fact that the constitutional symptoms which accompany other forms of tonsillitis are either very slight or absent altogether. The pathological process is similar to, if not identical with, ulcerative stomatitis (see page 284), with which it is sometimes associated. At such times the breath is foul and there is often profuse salivation.

Ulcero-membranous tonsillitis was first described by Vincent,* and by him attributed to a fusiform bacillus, which he described, although a spirillum was found associated with it. Vincent's observations have since been confirmed by a number of writers.†

The chief interest in ulcero-membranous tonsillitis lies in the diagnosis, although it is not an infrequent disease. It is to be treated, like ulcerative stomatitis, by the internal administration of chlorate of potash, combined with the local use of some antiseptic, such as peroxide of hydrogen or nitrate of silver.

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* La Presse Médicale, March 12, 1896.

† See Sobel and Herrmann, New York Medical Journal, December 7, 1901, for recent literature.

Vincent's bacillus is described as about twice as long as the Klebs-Loeffler bacillus. It is thin, with pointed ends, and sometimes bent; it is negative to Gram, and has not yet been isolated in pure culture, although Vincent was able to make it grow in bouillon with other organisms from the mouth. It is not yet determined whether the disease is due to the fusiform bacillus alone, or that the spirillum plays any part; the spirillum may possibly be merely a morphological variation of the bacillus. The fusiform bacillus is occasionally found alone; the spirillum, never alone. The bacillus is found in smears from an affected tonsil, in making which it is recommended to go deeply into the necrotic tissue, since the superficial parts are crowded with other bacteria.

FOLLICULAR TONSILLITIS.

This is the most frequent and most characteristic form of inflammation of the tonsil. It is essentially an inflammation of the tonsillar crypts, and secondarily of the whole glandular structure.

Etiology.—There is seen in certain children a predisposition to attacks of tonsillitis, so that from very slight exciting causes these occur—sometimes from exposure, sometimes from derangement of the stomach, and sometimes without any evident reason. Children with a rheumatic inheritance appear to be more susceptible than others. One attack predisposes to a second. Patients suffering from chronic hypertrophy of the tonsils are exceedingly prone to acute tonsillitis. It is not very common in infancy, but after this period it is very frequent throughout childhood. The disease, in all probability, begins as an infectious inflammation at the bottom of the crypts, due to the presence of streptococci or staphylococci, which readily enter from the mouth, and excite an attack whenever favourable conditions are present.

Lesions.—As a result of the inflammation, the tonsillar crypts are filled with epithelial cells, pus cells, mucus, and bacteria. These form masses which appear at the mouth of the crypts as small yellow dots, often miscalled ulcers. Sometimes, in addition, fibrin is poured out, and forms, with the other inflammatory products, little plugs which project somewhat from the surface of the mucous membrane, and which can easily be pressed out. Accompanying the changes in the mucous membrane above mentioned, there are acute congestion and swelling of the whole tonsil, with more or less proliferation of the lymphoid tissue. Follicular tonsillitis is always bilateral. Although the pathological process is generally limited to the tonsils, there may be more or less pharyngitis associated.

Symptoms.—The general symptoms usually appear before the local ones, and are often quite severe. The onset is abrupt, with chilly sensations, occasionally a distinct rigour. In infants there is often vomiting, and sometimes diarrhœa. There is pain in the back, in the muscles of the extremities, and in the head. Sometimes there is pain in the lateral cervical muscles. The temperature rises rapidly to 102° or 103° F.; often it touches 104° or 105° F.

The first local symptoms are some swelling of the tonsils and the appearance of isolated yellow spots a little larger than a pin's head. Often these can be wiped off with a swab, or the little plugs can be squeezed out, leaving slight depressions. Later there is acute congestion of the tonsil, with more swelling. Even when the disease is at its height the local pain and discomfort are only moderate, and in many cases scarcely noticeable. The swelling and tenderness of the lymph glands behind the angle of the jaw are not great, and may be absent.

The constitutional symptoms, as a rule, last three days, and are most severe upon the first day. The local symptoms last somewhat longer, but usually by the end of the fourth day the exudate has disappeared, although enlargement of the tonsil may persist for a week or even longer. On account of the connection of tonsillitis with rheumatism, the heart should be watched during attacks, especially in those who are subject to them.

Diagnosis.—Tonsillitis may be confounded at its onset with scarlet fever. Its constitutional symptoms in the beginning closely resemble malaria, influenza, or pneumonia. The great frequency of tonsillitis makes inspection of the throat imperative in every case of acute illness in children. The diagnosis from diphtheria is considered in connection with that disease.

Treatment.—Follicular tonsillitis is a mild disease without danger to life, and one which runs a short, self-limited course. The indications are, therefore, to make the patient as comfortable as possible by the relief of individual symptoms. Older children, particularly those who are rheumatic, should be treated with sodium salicylate, four grains every three hours being given for the first twenty-four hours, and later less frequently. In infants this drug must be given in smaller doses and with care, lest it upset the stomach. The general muscular pains of the first day are best relieved by phenacetine, two grains every four hours to a child three years old. Later it may be used in smaller doses, but enough should be given to make the patient comfortable.

Local treatment is better omitted in infants. Older children may gargle with a solution of boric acid or weak bichloride (1 to 10,000). Benefit often follows painting the tonsils with tincture of iodine or a ten-per-cent solution of silver nitrate. In all doubtful cases the patient should be isolated and the same treatment adopted as in diphtheria.

PHLEGMONOUS TONSILLITIS—PERITONSILLAR ABSCESS—QUINSY.

This is an inflammation of the cellular tissue surrounding the tonsil, sometimes invading the tonsil itself. It may terminate in resolution, but usually goes on to the formation of an abscess. Phlegmonous tonsillitis is much less common in children than in adults, and, compared with the other forms, it is a rare disease in early life. It is the only variety which is regularly unilateral. In most cases the inflammatory process is circumscribed, but in rare instances there is seen a diffuse phlegmonous inflammation of the pharynx.

In certain patients there exists a constitutional predisposition to the disease, which is often associated with rheumatism. The exciting cause may be exposure, or anything which may reduce the patient's general health, to which there is added local infection. Catarrhal pharyngitis predisposes to this disease.

Symptoms.—The onset resembles that of follicular tonsillitis, except that the general symptoms are usually less marked, the temperature is commonly not so high, and the muscular pains and prostration less severe. The local symptoms, however, are more striking. There is very severe pain in the throat, which is increased by deglutition, and finally may be so great that swallowing is almost impossible. It is difficult to open the mouth. There is pain in the lateral muscles of the neck, and often tenderness. In the beginning but little can be seen on inspection, even though the patient complains of a very sore throat. This is always a suspicious circumstance, and should lead one to look out for quinsy. It is due to the fact that the inflammation begins in the deeper tissues, and that the mucous membrane is affected later. After twenty-four or forty-eight hours there is usually quite marked swelling, which is rather more behind the tonsil than elsewhere, pushing it upward and forward; sometimes it is more in front of the tonsil. A little later there is intense inflammation of the mucous membrane covering the tonsil, fauces, and uvula, with marked congestion and œdema; the uvula may be pushed to one side, and the isthmus of the fauces diminished to less than one half its natural size. In one of my own cases marked torticollis was present, and existed for two or three days before the diagnosis of quinsy could be made by the other symptoms.

In most cases the recognition of quinsy is quite easy by attention to the symptoms above mentioned. By inspection of the throat, less information is sometimes obtained than by palpation; by this means a fulness, and later a point of fluctuation, can readily be made out. Acute phlegmonous tonsillitis generally involves no danger to life. In very young infants serious results may follow spontaneous rupture during sleep; and in older children occasionally there may be œdema of the glottis. If not treated, abscess usually forms in from five to seven days, and opens spontaneously.

Treatment.—If an early diagnosis is made an attack of quinsy may occasionally be aborted. For this many drugs have been advocated, but to my mind the best is salol, which should be given in doses of two grains every two hours to a child of five years. In some patients larger doses may be used. This may be combined with small doses (gr. $\frac{1}{4}$) of Dover's powder. Relief may be afforded by very hot or cold applications, according to the sensations of the patient. The holding of ice in the mouth and the application of an ice-bag externally, often give great comfort. In other cases, gargling with very hot water and the application of hot flaxseed poultices externally, will be preferred. As soon as fluctuation is detected an incision should be made with a guarded bistoury. If made too early, only a small amount of pus is evacuated and the abscess may refill. After spontaneous rupture the relief to symptoms is usually immediate.

CHRONIC HYPERTROPHY OF THE TONSILS.—CHRONIC TONSILLITIS.

The condition known as chronic hypertrophy, is a permanent enlargement due to a proliferation of the lymphoid tissue of the tonsils, and an increase in the connective-tissue stroma. If the increase in the connective tissue is slight, the tonsil is soft; if it is great, the tonsil is firm and hard, almost like a fibrous tumour. All degrees are found. Associated with hypertrophy of the tonsils there are frequently found adenoid growths of the pharynx, both of these depending upon similar local and constitutional conditions. There is in nearly all marked cases a chronic pharyngeal catarrh which may involve the Eustachian tubes.

Etiology.—Hypertrophy of the tonsils is an exceedingly common condition in the cities of the seacoast and lake districts of the temperate zone. In a routine examination of 2,000 New York school children, Chappell found enlargement of the tonsils sufficiently marked to be considered pathological, in 270 cases. The causes are constitutional and local. The constitutional causes relate to the conditions described in the chapter upon Lymphatism. This is often found in certain families for several generations. The condition is not connected with tuberculosis. It occurs in children who are in other respects healthy. Hypertrophy of the tonsils is often a congenital condition, increasing slowly during infancy, so as to produce marked symptoms by the time the child is two years old. The most important of the local causes are attacks of acute or subacute pharyngitis. While it is true that attacks of acute inflammation are often the cause of hypertrophy, it is also true that hypertrophy is one of the most frequent predisposing causes of acute attacks, and that it may be seen in children who have never had tonsillitis.

Symptoms.—Hypertrophy of the tonsils is rarely marked enough to cause any decided symptoms before the end of the second year, although I once saw in a younger child enlargement sufficient to bring the two tonsils into contact. The most important local symptoms, formerly ascribed to hypertrophied tonsils, are now known to depend upon adenoid growths of the pharynx. As these conditions are so frequently associated, it is somewhat difficult to determine which symptoms are due to the tonsils alone. In a marked case, the most prominent symptoms are mouth-breathing, disturbed sleep accompanied by snoring, and nasal voice—the patient in some cases talking as though he had food in his mouth. There may be some difficulty in swallowing solid food. Enlarged tonsils may often be felt externally. As a consequence of the obstruction of the Eustachian tubes there may be deafness. Deformities of the chest, such as pigeon-breast, are occasionally seen, but probably depend more upon obstructed respiration by adenoids than by the tonsils.

The soft tonsils may diminish somewhat in size spontaneously. They sometimes shrink very decidedly after an attack of acute tonsillitis, scarlet fever, or diphtheria. As a rule the tonsils become firmer and harder as time passes. They usually increase in size up to a certain point, and then remain nearly stationary until about puberty, when they may diminish considerably. During intercurrent attacks of inflammation, the swelling is much increased, and the symptoms are proportionately aggravated. In cases of marked enlargement very little spontaneous improvement is to be looked for during childhood.

Treatment.—Very large tonsils are a source of continued danger to the patient, and in every case of marked hypertrophy treatment should be advised. The danger may be from Eustachian catarrh and deafness, or from repeated attacks of acute tonsillitis. But quite as important as these is the fact that they increase the liability to contract diphtheria, and add to the dangers both from diphtheria and scarlet fever. If the patient is removed from the locality in which acute tonsillitis is liable to occur, to a dry climate, considerable improvement is likely to result in a young child in whom the tonsils are soft, but not much is to be expected in older children with hard, fibrous tonsils, except, perhaps, a cure of the accompanying pharyngeal catarrh.

The only internal remedy offering much chance of benefit is, in my experience, the syrup of the iodide of iron, which must be given in quite large doses (twenty drops three times a day to a child of five years), and continued for several months. In a small number of cases marked improvement is seen from this treatment, but in the majority but little change occurs. Astringent applications may accomplish something in recent, but practically nothing in old cases. In a marked case, operation is the only thing which can be relied upon to effect a cure. In those in which it is decided not to operate, or in which operation is refused, a faithful trial may be made with the other measures referred to. The question to be decided always is whether or not operation shall be done. For convenience of consideration, the cases may be divided into three groups: (1) those in which the tonsils are nearly or quite in contact; (2) those in which they project not more than one fourth of an inch beyond the faucial pillars; (3) the intermediate cases. All of the first group should unquestionably be operated upon, unless the patient's general condition is such as to forbid operation of any kind. Of the second group, few if any require operation. Whether an operation is done in the third group will depend upon the individual case. If there are frequent attacks of acute tonsillitis, and some deafness, an operation should be performed. If little or no local discomfort is experienced it may be postponed.

Of the various operations proposed, excision with the guillotine is the one which has in children superseded all others in the practice of New York physicians. The risk of hæmorrhage at this age is very slight.

The child is held as for the operation of intubation, except that the head is thrown backward. No after-treatment is required, excepting fluid diet and confinement to the house for two or three days. Excessive hæmorrhage may be controlled by digital pressure, or by the application of styptic cotton upon a swab; in extreme cases, by transfixing the tonsil stump with a hare-lip pin and the application of a ligature. I have more than once seen physicians greatly alarmed at the gray wound on the day following tonsillotomy, the appearance being such as to lead in several cases to the diagnosis of diphtheria. This mistake will not be made if the possibility of it is borne in mind. It is seldom that any but good results follow the operation of tonsillotomy if properly performed. It is too often neglected. Where adenoids of the pharynx are also present, the symptoms may depend more upon them than upon the enlarged tonsils, and little benefit is seen until the adenoid growths also are removed. Both may be operated upon at a single sitting, or at two sittings if preferred.

It is not usually necessary to remove the tonsil to a point even with the faucial pillars, but the more nearly we can come to this the better. The amount of shrinkage from cicatrization after operation has been, in my experience, generally less than was expected. As a rule, enlargement of the tonsil subsequent to an operation is not seen; but one should be careful about promising parents that it will not occur. I have seen it in two or three instances to a striking degree, and think it more likely to occur if children operated on are very young—i. e., under three years.

CHAPTER IV.

DISEASES OF THE ŒSOPHAGUS.

MALFORMATIONS.

Congenital anomalies of the œsophagus are much less frequent than those of the lower part of the respiratory tract, with which, however, they are often associated.

There may be, (1) Congenital fistula of the neck, due to a want of closure between the second and third branchial arches. This gives an external opening just above and to the outside of the sterno-clavicular articulation, which communicates with the upper part of the œsophagus or the lower part of the pharynx. (2) The œsophagus may be absent, the pharynx ending in a blind pouch. (3) The œsophagus may be obliterated in certain portions, being represented only by a fibrous cord. (4) There may be stenosis and dilatation or diverticula. (5) There may be a fistulous communication with the trachea, existing either alone or associated with some of the other deformities mentioned.

Congenital narrowing of the œsophagus and fistula of the neck are amenable to surgical treatment. The cases of complete obstruction in the œsophagus are almost of necessity fatal, the patients dying from inanition two or three days after birth.

The symptoms of œsophageal obstruction are regurgitation on attempts at swallowing and the impossibility of passing the stomach tube.

ACUTE ŒSOPHAGITIS.

It is quite remarkable, considering the frequency of pathological processes in the pharynx, that these so rarely extend to the œsophagus. Thrush, when very extensive in the pharynx, may involve the upper part of the œsophagus; but there it gives rise to new symptoms. Diphtheria and pseudo-diphtheria of the pharynx may invade the œsophagus, but this is seen only in very rare instances. In about seventy-five autopsies which I have seen in cases of diphtheria, the œsophagus was involved in but one, and in this case for three or four inches only. Diphtheria of the œsophagus produces no symptoms, and can not be diagnosticated during life.

Catarrhal Œsophagitis is very rarely met with. It may be caused by lacerations due to swallowing a foreign body, which may excite a simple catarrhal inflammation, or, if the foreign body is sharp and angular, lacerations may be produced which result in ulcerations of variable depth. The chief symptoms of catarrhal œsophagitis are soreness and pain on swallowing. These lacerations, when slight, are healed in a few days, and are rarely followed by any after-effects.

Corrosive Œsophagitis.—This is altogether the most frequent form, and the only one which is of clinical importance. The usual causes are the same as of corrosive gastritis, viz., the swallowing of caustic alkalies or strong acids. It is often in the œsophagus that the most extensive injury is done. The effects are superficial or deep, according to the amount of the irritant swallowed and its degree of concentration. There may be simply a destruction of the epithelial layer, which is followed by no serious consequences, or the mucous membrane may be destroyed and the submucous coat invaded; rarely, however, does the injury extend to the muscular layer. If the patient survives the dangers incident to the irritant poisoning and the acute inflammation which follows, healing by granulation and cicatrization takes place, the contraction of the cicatrix gradually narrowing the lumen of the œsophagus until stricture is produced.

The early symptoms of corrosive œsophagitis are mingled with those of inflammation of the mouth, pharynx, and stomach. There is a burning pain in the parts, great thirst, spasm of the œsophagus on attempts at swallowing. There follows a period of acute inflammation of several days' duration, with great dysphagia and pain, and in which the principal danger is œdema of the glottis. After this the patient may be comparatively well until the symptoms of stricture begin, usually in from three to six months after the injury.

The indications for treatment in the early stage, are to neutralize the caustic in order to prevent if possible its deep action, and to give oils, demulcent drinks, and ice for the local effect, and morphine for the pain.

The treatment of œsophageal stricture is purely surgical.

RETRO-ŒSOPHAGEAL ABSCESS.

Acute retro-œsophageal abscess occurs in infancy, though very rarely, the pathology being the same as in acute retro-pharyngeal abscess, the difference being merely one of location. A striking case of this kind occurred in the New York Foundling Hospital in 1904. An infant six months old was admitted with high fever (104° F.), severe dyspnœa, but with no loss of voice, which were the prominent symptoms until death occurred four days later. There was a leucocytosis of 100,000. At autopsy an abscess was found containing about three ounces of pus between the œsophagus and the spine, extending from the larynx to below the bifurcation of the trachea. Shortly afterward I saw a very similar case at the Babies' Hospital, following a retro-pharyngeal abscess which had been opened two weeks before. Similar abscesses have also been observed after acute pharyngitis with the acute infectious diseases.

Retro-œsophageal adenitis, or enlargement of the lymph nodes in this situation without suppuration, is also rare. I once met with a case of this sort in which the gland formed a tumour nearly an inch in diameter at the upper part of the œsophagus, causing pressure symptoms necessitating tracheotomy. The growth was at first thought to be malignant, but completely disappeared after a summer in the country.

Retro-œsophageal abscess may result from the breaking down of tuberculous lymph nodes in the posterior mediastinum, and may give rise to symptoms like those which result from an abscess due to Pott's disease.

Perforation of the œsophagus and a food-fistula connecting the œsophagus and the trachea, may result from ulceration caused by a tracheal canula or by a foreign body. This may be accompanied by abscess.

The most common variety of retro-œsophageal abscess is that due to Pott's disease of the lower cervical or upper dorsal region. The symptoms are obscure, and an exact diagnosis is not often made during life. Death may occur quite suddenly where the previous symptoms have been so slight as to be easily overlooked. The following is a fair example:

A girl two years old was admitted to the Babies' Hospital with caries of the upper dorsal region of two months' duration. The patient was kept in bed and a plaster-of-Paris jacket applied. About a month later dyspnœa was first observed; this was at times quite intense, and again almost absent. It was always on inspiration, expiration being easy. No explanation for this was found in the lungs. There was no difficulty in swallowing, and very little cough. After these symptoms had lasted for about a week, the child while eating was suddenly seized with violent dyspnœa, and in a few moments became completely asphyxiated. Tracheotomy was immediately done, and by means of artificial respiration the patient was restored to comparative comfort. About two hours later a second attack occurred, and the patient died in an hour. At the autopsy there was found an abscess a little larger than a hen's egg, containing about two ounces of curdy pus, overlying the bodies of the first three dorsal vertebræ and communicating with them. These vertebræ were carious. The right pneumogastric nerve, an inch and a half above the bifurcation of the trachea, was compressed between the abscess and a large tuberculous lymph node, with the capsule of which it was blended. In the lungs were a few small tuberculous deposits and the usual conditions found in death by asphyxia. The dyspnœa seems to have been of nervous and not of mechanical origin, and caused by irritation of the pneumogastric. The fatal issue was apparently from an increase of the pressure upon the nerve.

I have seen but one other case, and this closely resembled the one reported. Griffith has collected (Archives of Pædiatrics, January, 1898) twelve cases from the literature, and added one of his own. The symptoms in all were much alike. Dyspnœa, usually of a spasmodic character, was prominent in nearly all, and generally it was the most marked symptom. It was more marked on inspiration, and often accompanied by a spasmodic cough, suggesting laryngeal stenosis. The voice was affected in but two cases, in one complete aphonia being present. It is striking that in no case was there any difficulty in swallowing, in marked contrast to retro-pharyngeal abscess. Swelling in the neck was noted in but three cases. Spinal caries was stated to be present in seven cases and absent in two. The final attack of asphyxia sometimes came without warning, sometimes was preceded for several days or longer by milder attacks.

The diagnosis of this condition is very difficult, and a positive diagnosis almost impossible. It may be suspected in cases of Pott's disease of the lower cervical or upper dorsal regions, when there is spasmodic inspiratory dyspnœa, especially if accompanied by irritative cough. It should, however, be remembered that precisely similar symptoms may depend upon the irritation of a tuberculous node, and that the sudden asphyxia is exactly like that caused by the ulceration of such a node into the trachea or a large bronchus. The latter, however, may occur without the presence of Pott's disease. If the abscess is higher up, there may be a lateral swelling on either side of the neck, just above the clavicle. In most of the cases there are no external signs of disease. Such abscesses are too low to be reached by digital examination of the pharynx. The attack of asphyxia may also be confounded with that due to the presence of a foreign body in the larynx.

The prognosis in cases of retro-œsophageal abscess is exceedingly bad. Death usually results from pressure upon the pneumogastric, as in the cases reported. The abscess may rupture into the œsophagus and recovery follow. This termination is very rare, but such a case has been reported by Knight. A fatal one is reported by Löschner and Lambl. The abscess may burrow along the œsophagus into the abdominal cavity and excite peritonitis; finally, it may open externally.

But little is to be said under the head of *Treatment*. The symptoms are rarely definite enough to justify a radical surgical operation. Tracheotomy gives but temporary relief to the asphyxia. This operation should be performed, however, in every case, because of the impossibility of making a diagnosis of retro-œsophageal abscess from other conditions in which the operation might be curative.

CHAPTER V.

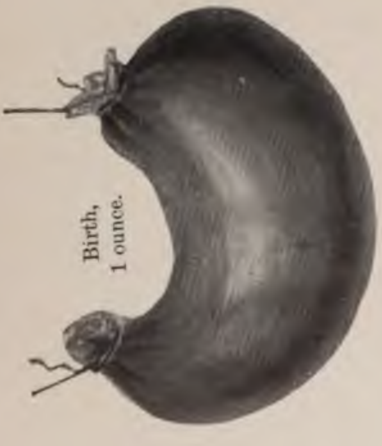
DISEASES OF THE STOMACH.

It is difficult wholly to separate diseases of the stomach from those of the intestines. Although in older children they are often quite distinct, in infancy they are more frequently associated; but at one time the gastric symptoms may be prominent, and at another the intestinal symptoms. Functional disorders particularly are likely to involve the whole tract. Serious organic lesions are more frequently limited in their extent either to the stomach or to the intestine. The former are rare, while the latter are very common. The diseases in which the stomach is alone or chiefly involved will be considered by themselves. Those in which both the stomach and intestine are involved are classed with the intestinal diseases, as the intestinal symptoms usually predominate.

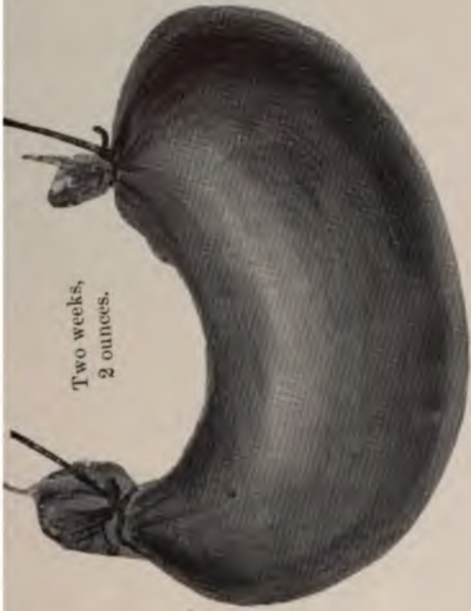
DIGESTION IN INFANCY.

The first step in the process of digestion in the newly-born infant is sucking. During this act the nipple is grasped between the lower lip and tongue below, and the upper lip and jaw above. The back of the mouth is closed by the fall of the palate. A strong downward movement of the lower jaw rarefies the air in the mouth, and produces the suction force which causes the milk to flow. Sucking can be carried on only when the nose is free for respiration and the palate and upper jaw intact. Children with deformities of the mouth, like cleft palate and harelip, suck only

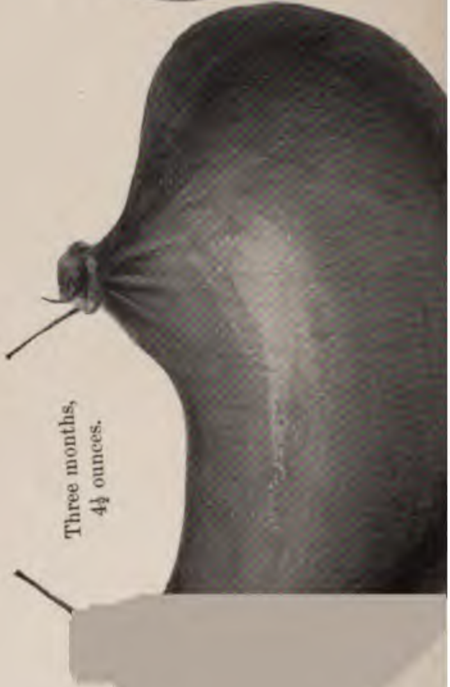
PLATE VII.



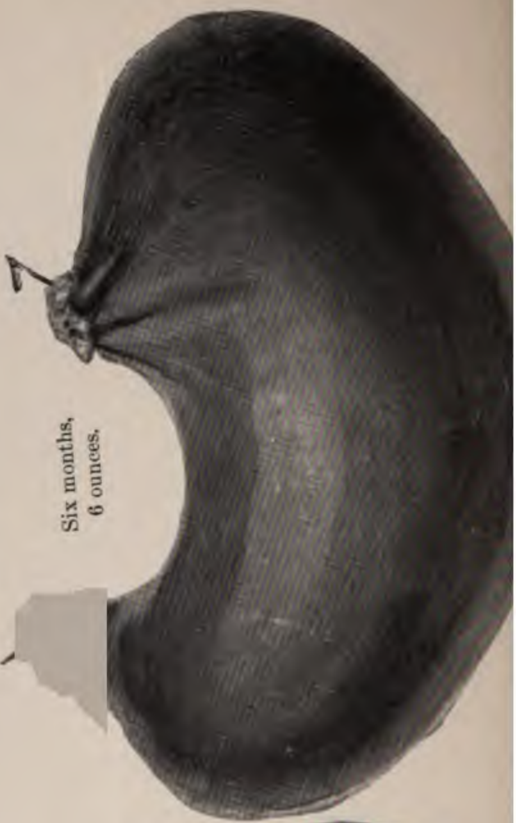
Birth,
1 ounce.



Two weeks,
2 ounces.



Three months,
4½ ounces.



Six months,
6 ounces.

with the greatest difficulty, and complete nasal obstruction prevents nursing.

The Saliva.—This is present at birth only in very small quantity, and the part which it plays in digestion in early infancy is an insignificant one. During the third and fourth months it increases markedly in amount, and at this time it possesses quite actively the power of transforming starch into sugar. This property is present only to a very slight degree during the first eight or ten weeks. With the advent of the teeth there is a further increase in the amount of saliva secreted, indicating a change in the digestion of the infant.

The Stomach.—The position of the stomach in the fœtus is nearly vertical. In the newly-born child it lies obliquely in the abdomen, and at the end of infancy has almost reached the transverse position. The stomach at birth is nearly cylindrical, but the fundus increases in size very rapidly during the first year, although it does not reach its full development until quite late in childhood. In Plate VII are shown the actual size and shape of the stomach at the various periods of infancy. In the following table are given the results of post-mortem measurements of the stomach, which I have personally made in ninety-one infants under fourteen months of age:

The Capacity of the Stomach.

Age.	Number of cases.	Average capacity.	Age.	Number of cases.	Average capacity.
Birth...............	5	1·20 oz.	12 weeks........	6	4·50 oz.
2 weeks............	7	1·50 "	14 to 18 weeks...	12	5·00 "
4 "	4	2·00 "	5 to 6 months...	14	5·75 "
6 "	11	2·27 "	7 to 8 " ...	9	6·88 "
8 "	4	3·37 "	10 to 11 " ...	7	8·14 "
10 "	2	4·25 "	12 to 14 " ...	10	8·90 "

In brief, the average capacity was, at birth, one and one fifth ounce; at three months, four and a half ounces; at six months, six ounces; at twelve months, nine ounces.

Gastric digestion.—The part taken by the stomach in digestion is smaller than was formerly supposed, and not so important in infants as in adults. The food leaves the stomach so rapidly that a large part of the casein must pass into the intestine before it is converted into peptones. The opinion has been steadily gaining ground that the function of the stomach is largely that of a reservoir, into which the milk is received and from which it is allowed to pass gradually into the intestine; and that the gastric process is only a preliminary and partial one, even in the digestion of proteids, this being completed in the intestine.

The only part of the food acted on in the stomach is the proteids, which are transformed successively into acid-albumin, albumoses, and peptones. This is accomplished by the agency of the pepsin and the acid of the gastric juice—generally hydrochloric acid, although lactic acid may take its place. Pepsin is found in the stomach at birth, and even in the embryo as early as the fourth month (Krüger). The reaction of the stomach contents in fasting is acid, and at this time usually free hydrochloric acid can be demonstrated; soon after a meal of human milk it is alkaline or neutral; after one of cow's milk it is acid or neutral. In fifteen minutes after feeding the reaction is always acid (Leo). Free hydrochloric acid can not usually be demonstrated until about an hour after feeding, then only in small quantities, and in very many cases not at all. Some good observers go so far as to say that in health free acid is never found during digestion. The reason for this apparently is, that the acid combines with the casein of the milk, that of cow's milk in particular having a very great power of combining with hydrochloric acid.

Lactic acid is feebler in its digestive power than hydrochloric acid. It is more abundant early in infancy than later; it is derived from the milk sugar. It is rarely found as free acid; never in health, according to many observers.

The coagulation of milk in the stomach is accomplished through the agency of the rennet ferment (the lab-ferment of Hammarsten). This is independent of both the pepsin and the acid of the stomach. It acts in acid, alkaline, and neutral media. Coagulation is the first change in the milk in the stomach. Human milk coagulates in loose flocculi and quite imperfectly, more firmly if the stomach is very acid. Cow's milk, unless diluted, coagulates in firm, compact masses. Under the influence of pepsin and hydrochloric acid, solution of this coagulum now begins; but this is only partially accomplished in the stomach. It goes forward much more rapidly in the case of human milk, because the amount of casein is less and because of the smaller curds. The milk begins to leave the stomach very soon after the meal, and even during the first half hour a considerable part passes into the intestine. At the end of an hour the stomach in a young infant is often empty. In the case of cow's milk, not only are the coagula firmer, but the amount of casein present is much larger, and hence the milk is detained in the stomach a longer time; even then a considerable portion of it must pass but little changed into the intestine.

The duration of gastric digestion varies with the age of the infant and with the food. During the first month the stomach of healthy nursing infants is usually found empty in an hour and a half after feeding; often in one hour. In those taking cow's milk the average is at least half an hour longer. In infants from two to eight months old the average is two hours for those receiving breast-milk, and two and a half to three hours for those fed upon cow's milk. This is influenced by the size of the meal taken. This period is very much longer in all cases of disordered digestion.

The bacteria of the stomach are very few as compared with those of the intestine, and no varieties are constantly present (Booker).

The Intestines.—The length of the small intestine at birth is about nine feet; that of the large intestine about eighteen inches. The great length of the sigmoid flexure is the most striking peculiarity, this being nearly one half the length of the large intestine.

Intestinal digestion.—All the important elements of food—proteids, carbohydrates, and fats—are acted upon by the pancreatic juice. The proteids are converted into peptones by the trypsin, which is active only in an alkaline medium. How much of the proteids of the milk is left for intestinal digestion, depends upon how well the stomach has done its part. In every case something is left; in most cases a large part of the proteids passes but little changed into the intestine. The amylolytic ferment of the pancreas has the power of converting starch into sugar. This action is feeble during the first five or six months, but we can not accept the statements of Koronin and Zweifel, that it is entirely absent in early infancy. Fats are partly emulsified and partly saponified by the pancreatic juice, in connection with bile, which probably furnishes the needed alkali. The pancreatic juice actively emulsifies fat, even at birth.

The very large size of the liver in the newly born indicates how important are its functions in digestion. The biliary secretion is present as early as the third month of fœtal life (Zweifel). Bile assists in the digestion and absorption of fats, as has already been mentioned. In addition it is a stimulus to peristalsis, and in this way aids in the absorption of all kinds of food. Its antiseptic effect is very doubtful. It has a feeble diastatic action upon starch. The greater part of the bile is reabsorbed from the intestine.

Milk sugar is changed into galactose (Biedert), cane sugar into dextrose and levulose, all three being closely allied substances. Through what agency these changes are accomplished is not now positively known, but it is probably the pancreatic juice.

The action of the intestinal juice is not perfectly understood; its chief function is thought to be diastatic. It is alkaline in reaction, and probably facilitates the action of the trypsin, the diastatic ferment, and the absorption of fats.

Absorption.—From the stomach, absorption of water, salts, sugar, and peptones may take place directly into the blood. From the small intestine, in addition to the above elements, fat is absorbed especially by the villi. Absorption is less active than secretion in the small intestine, except in the duodenum. It is accomplished through the agency of the villi and the simple follicles of the mucous membrane. It is perhaps partly by filtration and endosmosis, but chiefly through the activity of the epithelial cells themselves (Hoppe-Seyler, Haidenhain). Absorption from the large intestine is quite imperfect. There are no villi, and hence fat absorption is very slight. Sugar, salts, and peptones, however, may be absorbed with moderate facility. Since there is little or no digestive activity in the large intestine, if this is used as a means of nutrition, the food must be given in a condition in which it is ready for absorption.

Even in healthy nursing infants complete absorption is possible only in the case of milk sugar. From two to five per cent of the proteids and fats taken pass through the intestinal canal. In infants taking cow's milk the fat-residue is from one to three per cent greater than in those who are breast-fed (Uffelmann). Even when the amount of fat given is considerably greater than that usually present in cow's milk, it may be almost entirely absorbed. In infants taking cow's milk the proteid residue is relatively much greater than that of the fat.

In cases of indigestion the increase in the food-residue in most cases is first in the proteids, next in the fat, and least in the sugar. In some of the chronic cases the principal increase may be in the fat.

Intestinal Bacteria.—For the fundamental work upon this subject we are indebted to the researches of Escherich. Bacteria are absent from the entire gastro-enteric tract at birth. They quickly enter by the mouth, and by the end of twenty-four hours they are usually found in all parts of the intestinal tract. The meconium-bacteria are derived from the inspired air, and hence vary somewhat with surroundings. As soon as the ingestion of milk begins these varieties are displaced, and throughout the period in which the infant has this food exclusively, there have been found in healthy conditions but two varieties which are constantly present. These are the *bacterium lactis aerogenes* and the *bacterium coli commune*. The first is found most abundantly in the upper part of the small intestine, diminishing as we descend, in small numbers only in the colon, and usually none are in the fæces. It seems to require for its growth the presence of milk sugar, hence its absence from that part of the intestine where milk sugar is not found. Milk sugar is decomposed by it with the formation of lactic acid (acetic, according to Baginsky), carbon dioxide, hydrogen, and methane. This action is not hindered by the bile. The *b. lactis* has no action of importance on either the fat or casein of the milk.

The *b. coli commune* is found in but small numbers in the upper small intestine, becoming more abundant as we descend. In the colon and in the fæces it is present in immense numbers, and in the fæces is sometimes almost the only variety. The activity of the *b. coli commune* apparently begins where that of the *b. lactis* ends, viz., in the lower part of the small intestine. It does not seem to depend for its growth upon any part of the food, but upon the intestinal secretions. A change from a milk diet to a mixed diet of meat and farinaceous food, produces a constant change in the bacteria of the intestine. The *b. lactis* disappears;

the *b. coli commune,* however, continues to be found as the principal form in the colon.

Regarding the action of these bacteria but little is as yet known. The *b. lactis* is believed not to be pathogenic. There seems to be abundant evidence to show that the *b. coli commune,* though not ordinarily pathogenic, may under a great many conditions become so.

Fæces.—The first discharges after birth are called meconium; this is of a dark brownish-green colour, semi-solid, and usually passed from four to six times daily during the first two or three days. On the third day the stools begin to change in character, and by the fourth or fifth day they have usually assumed the appearance of healthy milk-fæces. Under many abnormal conditions the stools may continue to have the character of meconium for a week or more. The composition of meconium is intestinal mucus, bile, the vernix caseosa, epithelial cells from the epidermis, hairs, fat-globules, and cholesterin crystals. For its formation there are necessary the secretions of the intestine and the liver and the swallowing of a considerable amount of amniotic fluid.

Milk-fæces.—The normal amount of fæces discharged daily by a healthy nursing infant is from two to three ounces. Such stools have the colour of the yolk of egg. They are smooth, homogeneous, of a soft, butter-like consistency, with an acid reaction, and a slightly acid but not unpleasant odour. The reaction is due to the presence of fatty acids or lactic acid. The colour depends upon bilirubin. The stools of an infant fed upon properly modified cow's milk may in conditions of perfect digestion differ in no respect from those described; they are, however, usually firmer, of a paler yellow colour, and may be neutral or even alkaline in reaction, depending upon the decomposition of casein. The principal differences depend chiefly upon the presence of unabsorbed casein.

The only gases present are hydrogen and carbon dioxide (Escherich). Sulphuretted hydrogen and marsh gas, to which the odour of adult stools is largely due, are not present. The following is the chemical composition as given by Wegscheider:

Water			85.13
Solids	Organic	13.71	14.87
	Inorganic	1.16	
			100.00

The proteids of breast-milk are almost entirely absorbed. According to Uffelmann, they form but 1.5 per cent of the dry residue of the fæces. The stools of infants fed upon cow's milk are usually larger, and generally contain casein. If the percentage of casein in the milk as fed is excessive, it may be present in the fæces in large amount, the stools then being of a pale-yellow or white colour, quite dry, often formed, and with an odour sometimes cheesy, at other times foul.

Fat is always present, and forms, according to Wegscheider and Uffelmann, from 9 to 25 per cent of the dry residue of milk fæces. According to Tschernoff and some other recent observers, the proportion is as high as 28 to 35 per cent. It is present as neutral fat, fatty acids, and soaps. Sugar is not found, but its derivative, lactic acid, may be present in a small amount. Inorganic salts form about 8 per cent of the dry residue. They are chiefly the salts of lime. Of the biliary elements there are hydrobilirubin, unchanged bilirubin, and cholesterin in considerable amount. The presence of biliary acids is doubtful. Mucus is always present in considerable quantity; also columnar intestinal epithelium. Leucin, tyrosin, and other products of albuminous decomposition—phenol and skatol—are absent; indol is rarely found (Uffelmann).

Microscopically there are seen epithelial cells, chiefly of the columnar variety, a few round cells, mucous corpuscles, fat-globules and crystals of fatty acids, cholesterin, mucin, protein substance, crystalline inorganic salts, sometimes bilirubin in crystals, yeast fungi, and bacteria in immense numbers.

If the infant is taking a food containing starch, this will appear to a greater or less extent in the stools, a larger amount in the case of very young infants. Starch is recognised by the blue reaction with iodine, or the violet reaction if the starch has been converted into dextrine, as is often the case. Starch granules may be seen under the microscope.

The number of stools during the first two weeks is from three to six daily. After the first month two stools a day are the average; many infants have three, many others but one.

As soon as an infant is put upon a mixed diet, the peculiar characters of the stools cease, and they come to resemble more closely those of the adult, though remaining softer throughout infancy. They become darker in colour and assume the adult odour, while retaining their acid reaction. The bacteria, while still in great numbers, are more varied than are met with in milk-fæces.

MALPOSITIONS AND MALFORMATIONS OF THE STOMACH.

The stomach is sometimes in the thoracic cavity in cases of diaphragmatic hernia. It may be found in a vertical (fœtal) position, variously adherent to the colon and small intestine. Malformations are much less frequent than those of other parts of the alimentary tract. There may be atresia or stenosis at either orifice, and very rarely a constriction is found near the middle of the organ, dividing it into compartments. The symptoms of atresia at either orifice are persistent vomiting, and death in a few days from inanition.

HYPERTROPHIC STENOSIS OF THE PYLORUS.

It is only during the last few years that this condition has been generally recognized. Although many cases have been reported and the clinical picture and the pathological anatomy are now clearly understood, there is still considerable diversity of opinion in regard to many points in the pathogenesis and treatment.

Males are undoubtedly more often affected than females. Of 68 reported cases, 55 were in boys and 13 in girls. In several instances two children in one family have suffered from this condition. The family history bears in no way upon the disease; and that a great majority of reported cases have been in breast-fed infants, is probably not significant.

The view of pathology most widely accepted is that there are two factors present: (1) hypertrophic, an abnormal development of the pylorus, especially its transverse muscular fibres, a congenital condition; (2) spasmodic, consisting of a contraction of these increased fibres. The two elements are associated in varying degrees; in some cases the hypertrophic, in others the spasmodic, predominates.

The reason why vomiting and other symptoms may be delayed for several weeks appears to be that the motor power of the stomach may be for a time sufficient to force the food through the narrowed orifice. The additional spasm at this time may be insignificant. It is, however, after the stomach loses its reserve power that the signs of insufficiency present themselves. Recovery may still take place by the stomach regaining its compensation, and the pylorus losing its spasmodic contraction. That such a thing actually does occur is shown by the occasional finding at autopsies upon older children or adults of non-inflammatory constriction of the pylorus. Recoveries without operation have been reported by Heubner, Ibrahim, and others, even after all the typical symptoms were present.

Another theory advocated by Thomson (Edinburgh) is that the hypertrophy is a secondary condition brought about by a primary spasm of the pylorus. Were this so we would not expect such an increase of the connective-tissue, submucosa and mucosa, as is often found. Furthermore, when symptoms have existed from birth the time seems too short for the development of such an enormous hypertrophy as is present. The same may also be said of cases with symptoms coming on later, but acutely. On this account Thomson was obliged to assume that the spasm began in intra-uterine life.

Lesions.—Uniform pathological changes have been found at autopsy in all cases which gave typical symptoms during life.

The pylorus is elongated, greatly thickened, being often as hard as cartilage, and projects into the duodenum like a cervix uteri. On section the orifice is seen to be much diminished in diameter, but what is especially striking is the great thickness of the wall of the pylorus. It is often one-fifth of an inch (5 mm.) or more in thickness, and of this fully two-thirds is in the muscular layer. Thick folds of mucous membrane may diminish the lumen still further. There may be hypertrophy of the wall of the whole stomach; and while the organ may be much dilated, it is often smaller than usual. Rarely there may be a dilatation of the lower end of the œsophagus.

Microscopically the most marked change is the great increase in thickness of the circular muscular layer, but there may be also an increase in the longitudinal muscle and in the connective-tissue of the submucosa.

Symptoms.—The symptoms may appear in the first days of life. It is, however, more common and more striking for a period of comparative or absolute good health with gain in weight and good digestion to continue for several days or even weeks before the most important symptom begins.

The essential symptom is vomiting. At first there is nothing characteristic about it, but it soon becomes more persistent than is present in any other condition. It resists all measures which under other conditions usually bring relief. Vomiting may come on directly after food is taken or it may be delayed for an hour or more. In some severe cases almost all the food taken is vomited, in others only a portion of it. It may happen, especially after considerable dilatation has taken place, that the vomiting occurs at much longer intervals, possibly only once a day, but the child may then reject the greater part of what has been taken for the previous twenty-four hours.

There need be no exciting cause for the vomiting. It sometimes takes place when the child is absolutely quiet, even asleep. The vomited matters consist of food, the appearance of which is modified by the length of time it has remained in the stomach; there is usually mucus, the amount depending largely upon the duration of the condition; there may be small clots or streaks of blood from hæmorrhagic erosions caused by the excessive contractions of the stomach. Bile is not present. There is a motor insufficiency of a very marked degree, so that after five or six or even ten hours of fasting, food may be removed from the stomach by lavage.

Next to vomiting the most constant symptom is the progressive and often rapid loss of weight. At the end of two or three months the child may weigh a pound or two less than at birth. There are present all the evidences of malnutrition or even marasmus.

The urine is scanty, of high specific gravity, and deposits a heavy sediment of urates upon the napkins. If all the food is rejected there is absolute constipation; when some food passes the pylorus, the stools may be green, but more often are brown and very hard. The condition is not accompanied by fever.

On an examination of the infant's abdomen one is struck by the prominent appearance of the epigastrium as compared with the retracted and sunken portion below the umbilicus. Especially is this the case if emaciation is extreme.

A very striking symptom is the peristaltic waves. These are usually present after food has been taken, but may be seen at almost any time and may sometimes be induced by tapping or rubbing the epigastrium. They pass from left to right across the epigastrium and only for a short distance beyond the median line. They can hardly be mistaken. These waves are not diagnostic, as they may be seen in other conditions and are sometimes wanting in hypertrophic stenosis.

Ibrahim describes a tonic contraction of the stomach that he has observed, the contraction lasting as long as fifteen seconds; the outline of the whole stomach could be seen and the greater curvature distinctly felt. The ingestion of food is sometimes followed by signs of pain. After a few mouthfuls have been taken eagerly, the infant can with difficulty be induced to take more.

Visible peristalsis may occur, however, without any direct evidence of pain. In about one-fourth of the cases a pyloric tumour is present, situated slightly to the right of the median line; but usually this is obscured by the position of the liver. The tumour is movable, quite hard, about the diameter of the little finger, and feels not unlike a large lymph gland. The absence of such a tumour is of no importance in diagnosis.

Concerning the usual course of the disease there is yet considerable difference of opinion. It is difficult to believe that most of these patients go on to recovery; yet many excellent observers, Heubner among them, insist that the vast majority recover completely, even after having exhibited the characteristic symptoms. In such cases it is stated that the vomiting grows less and less and finally ceases, with an improvement in all the other symptoms; the peristaltic waves are usually the last evidences to disappear; these may be seen weeks and even months after all vomiting has ceased.

The more common belief is that unless relieved the cases usually grow slowly or rapidly worse, with progressive loss of weight and strength, with death in a state of extreme marasmus, the vomiting persisting until the end.

Treatment.—Since it is impossible to make a correct diagnosis until the patient has been observed for some time, the early treatment is that of persistent vomiting—stomach washing and the most careful attention to feeding. Saline enemata should be given regularly to furnish the fluid required by the body and occasionally to cleanse the intestine. Nutritive enemata are of no value for prolonged use.

After a positive diagnosis has been made the question of operation must be considered. It is held by many that operation is absolutely contra-indicated on account of its great attendant dangers, and as most cases recover without it. General experience, however, is opposed to this view. If careful and intelligent treatment produces no improvement, and vomiting continues until life is threatened, surgery holds out some hope of relief, though a slender one. Cases apparently hopeless have been rescued by operation. The greatest judgment is necessary not to continue the expectant treatment too long, and thus allow the child to become so wasted and exhausted that operation is inadmissible. About one fourth of the cases operated on thus far have recovered.

Of the various operations employed, pyloroplasty and anterior gastro-enterostomy seem to be the best on account of the small incision necessary, and the rapidity with which they may be done. Stiles (Glasgow) states that after anterior gastro-enterostomy the feeding is much simpler than after any other method of treatment.*

VOMITING.

Vomiting is one of the most frequent symptoms of disease in infants and young children, and occurs from a wide variety of causes. In disorders of digestion it is the one particular symptom which points to the stomach as the seat of disease. At the same time, it is one of the most difficult symptoms to control. From both a diagnostic and therapeutic standpoint, therefore, it is important that the significance of vomiting should be appreciated.

The physician must have in mind both its common and its uncommon causes. Vomiting takes place with great facility in young infants even from slight causes, owing to the position and shape of the stomach.

1. *Vomiting from overfilling of the stomach.*—This is often seen in nursing infants, and there may be no other symptom of disease. It is characterized by the fact that it comes within a few minutes after nursing, that it is easy and without effort, and that the food is but little changed. It may be excited by moving the child or making undue pressure upon the stomach. It often comes with eructations of gas or air which has been swallowed.

Vomiting from overdistention may be regarded as a safety-valve, and requires no treatment except to diminish the quantity of food.

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* For recent literature see Ibrahim's monograph, Karger, Berlin, 1905; Wachenheim, Amer. Jour. of the Med. Sciences, April, 1905; and for references to cases treated surgically—Shaw and Elting, Archives of Pædiatrics, December, 1904.

2. Vomiting is almost invariably present in cases of *acute gastric indigestion*, whether there is inflammation of the stomach or not. It does not usually come immediately after feeding, and it may be delayed for several hours. It is often preceded by fever and by marked prostration, which in young infants may approach collapse. It may cease when the contents of the stomach have been expelled, but often mucus, serum, and, in severe cases, bile, may be vomited for some time afterward. In these cases vomiting is due to the irritation of undigested food, and to the exaggerated reflex irritability of the stomach from congestion of the mucous membrane.

3. In *acute intestinal obstruction* vomiting is rarely absent, and in most cases it is persistent. In the newly born, persistent vomiting is almost invariably dependent upon congenital obstruction of the intestine, which is most frequently in the duodenum. In malformations of the colon and rectum it is less constant and appears later. In intussusception, vomiting is forcible, immediately excited by the taking of food, and is at first bilious, but later may become fæcal. The vomiting in intestinal obstruction is associated with general symptoms of marked prostration, and usually with obstipation.

4. Vomiting is a frequent and almost a constant symptom of general *peritonitis*. It is then associated with abdominal distention, tenderness, and fever.

5. In certain *nervous diseases*, especially tumour of the brain and acute meningitis whether simple or tuberculous, vomiting is very common. In tumour it may be the earliest, and for some time the only marked symptom. In several cases I have observed, exactly the same type of vomiting was present. It occurred only in the morning, sometimes before breakfast, sometimes suddenly during the meal, and was repeated every few days. Cerebral vomiting is usually forcible or projectile. It may have no relation to meals. The vomited matters are not characteristic, and the tongue may be clean. Headache, dulness, slight fever, constipation, and irregular pulse and respiration are usually present sooner or later.

6. In infants, and less frequently in older children, vomiting is one of the usual symptoms to mark the *onset of acute infectious diseases*, especially the beginning of scarlet fever, pneumonia, and malaria. In these cases vomiting may be due simply to the arrest of digestion, or to the effect of the poison upon the nerve centres.

7. An accumulation in the blood of various *toxic* materials may provoke vomiting; the most frequent example is uræmia. In cyclic vomiting it is quite probable that the cause is the accumulation of some toxic agent in the blood. The absorption of ptomaines and other poisons taken in with milk or other food, or developed in the gastro-enteric tract, may excite vomiting. In some of these conditions it is possible that

the vomiting may be eliminative—an effort on the part of Nature to get rid of the toxic materials. The cases dependent upon renal disease are discovered by frequent and careful examination of the urine. The other forms are often exceedingly obscure, and recognised only by the exclusion of all other frequent and infrequent causes of vomiting.

8. Vomiting may be *reflex* from irritation in the pharynx. This is frequent in young infants, who may induce vomiting by stuffing the fingers into the mouth. In certain cases the irritation from worms in the intestinal tract may cause vomiting, and it is possible that even dentition may produce it.

9. *Habit* is a frequent cause in cases of chronic vomiting. I have seen a child who had the power of vomiting at will anything in the nature of food which he did not like, yet whose stomach at the same time would bear large doses of quinine, to which he had no aversion, without the slightest disturbance. In young infants a habit of regurgitating the food may be acquired, so that this takes place more or less during the process of digestion after every meal. This is sometimes preceded by a movement of the mouth and fauces resembling swallowing, until finally the milk appears in the mouth. Habit is a potent cause in continuing vomiting where it has occurred frequently. In children who have this habit the most trivial cause will provoke it. It may be present without any other sign of gastric disease, and appears simply to depend upon exaggerated reflex irritability of the organ. I have seen a number of children who up to the third or fourth year objected so strenuously to taking solid food that they would immediately vomit it, no matter of what variety or in how small a quantity, although fluids were taken and digested without the slightest difficulty.

10. *Chronic vomiting* may depend upon habit, as just described, or upon chronic indigestion; or it may be associated with chronic pulmonary disease—vomiting here being excited by the attacks of cough, at first only when the paroxysms are severe, and later even when they are slight. In chronic indigestion the vomited matters are always characteristic, they have a distinct relation to meals, and they are accompanied by other symptoms of deranged nutrition.

The diagnosis of a case in which vomiting is the chief symptom may be difficult. The first important distinction to be made is between cases in which the vomiting is of gastric origin, and those in which it depends upon other causes, like intestinal obstruction, cerebral disease, toxic conditions, etc. It is only by a careful consideration of the other symptoms associated that an accurate diagnosis can be reached.

The treatment of vomiting is the treatment of the cause upon which it depends.

CYCLIC VOMITING.

This is not an infrequent disease; it has, however, as yet attracted but little attention except in this country. Although the clinical picture is a very clear and definite one, its exact pathology is undetermined. It has also been described under the names of periodical vomiting, recurrent vomiting, and a gastric neurosis. It is characterized by periodical attacks of vomiting, which recur at regular or irregular intervals of weeks or months, apparently without any adequate exciting cause. The usual duration of the attacks is two or three days, during which all attempts to control the vomiting are usually without avail, but at the end of this time it generally ceases spontaneously.

Etiology.—The first attacks are usually seen between the ages of two and four years, but they may date back to infancy. The two sexes seem to be almost equally liable. A few of the patients are strong children, but the great majority are rather delicate and of a highly nervous temperament. The cases are seen chiefly in private practice, often occurring among those who have the best surroundings. In most cases the antecedents of patients are of the neurotic type, and in the family of some there is a marked tendency to gouty manifestations. The attacks are not traceable to distinct or flagrant errors in diet, and yet the habitual diet seems to bear some relation to the disease. In my own cases I have most frequently found the diet to be excessive in carbohydrates, particularly in the amount of oatmeal and potato. The exciting cause is often a nervous one—great fatigue or unusual excitement, sometimes a railroad journey or a child's party; in many instances it seems to be induced by some minor illness having no relation to the digestive tract, such as an attack of tonsillitis or bronchitis.

Symptoms.—The clinical picture presented by these cases is very characteristic, and is well illustrated by the history of the following case:

The patient was a well-nourished boy of six years when he first came under treatment. He belonged to a neurotic family, and the attacks dated back to infancy. From this time they had recurred usually at intervals of a few months; occasionally five or six months would pass without one. The symptoms in all the attacks were similar in kind, differing only in degree. They were preceded by a prodromal period lasting from twelve to twenty-four hours, marked by languor, dulness, dark rings under the eyes, loss of appetite, and a general sense of discomfort in the epigastrium. At this time the temperature was generally slightly elevated. The vomiting then began suddenly. It was attended with great retching and distress; it was often repeated every half-hour or hour for two days. On one occasion it occurred seventeen times in a single night. Vomiting was immediately excited by the taking of any food or drink, but it occurred when nothing was taken. The vomited matters consisted of frothy mucus and serum, frequently streaked with blood, apparently from the violence of the emesis. The reaction was very strongly acid; sometimes there was bilious vomiting. The temperature usually fell to about 100° F. when the vomiting began, and continued at or below this point throughout the attack. By the end of the second day the exhaustion was very marked—so severe, in fact, as apparently to threaten life.

The child lay in a semi-stupor, with eyes half open, lips and tongue dry, rousing at times to beg for water. The pulse was rapid and weak, and sometimes slightly irregular. There was no distention of the abdomen; it was usually flattened. By the third day the vomiting became less frequent and then ceased entirely. Convalescence was rapid, and by the end of the week the boy was almost as well as usual. The attacks continued to recur at gradually lengthening intervals until they finally ceased altogether at about the twelfth year.

Over forty of these cases have come under my observation, and in many of them I have had an opportunity to witness several attacks. The usual duration is one to three days. In one patient they lasted regularly for five days. Occasionally a severe attack will last a week. The average number of attacks is three or four a year.

Prodromal symptoms are present in most of them—headache, general languor, coated tongue, and anorexia are the most frequent; in some there is marked constipation, with a history of very white stools for some time. The tongue is usually coated at the beginning of an attack, and at its height it is often dry and brown. The abdomen seems empty and its walls sunken; pain and tenderness are both rare. The bowels are constipated and move only by artificial means, and even then not freely.

There is, as a rule, no desire for food, but the continual cry is for water to quench the constant, burning thirst. The pulse after the second day becomes rapid, soft, and often somewhat irregular. The respiration is shallow, and at times this also may be irregular. The temperature is seldom over 100.5° F., a point of much diagnostic value. The patients are dull, apathetic, and usually wish to be left alone. Headache is very common.

The disposition to vomit is sometimes so great that patients are afraid to move or even to talk lest it may be provoked. The vomited matter is large in amount, considering that the patient is fasting. It is essentially gastric juice, containing free HCl, mucus, serum, many epithelial cells, and often traces of blood. The urine is concentrated, and frequently contains at the height of the attack a trace of albumin, a few hyaline casts, and some blood cells—evidences of a moderate renal hyperæmia. There is usually an excess of indican. A condition practically constant, and first pointed out by Edsall (Philadelphia), is the presence in the urine of acetone, diacetic and oxybutyric acids. This is thought to give some reason for the belief that cyclic vomiting is a form of acid intoxication. The above findings are so constant as to be of some diagnostic value. On the other hand, it should be stated that some hold that these urinary conditions are simply the result of the starvation.

In two cases of my own, where careful determinations of urea and uric acid were made during and following attacks, it was observed that the excretion of urea was but little altered, while that of uric acid fell during the early days of an attack to one-half or one-third the normal for the same individual in health.

The Nature of the Attacks.—These cases have little in common with the ordinary attacks of indigestion. With our present knowledge they are to be regarded as nervous explosions due to faulty metabolism, having many points of resemblance to migraine in the adult. The effect upon uric-acid elimination in the case cited is very similar to that which occurs in migraine; and Rachford has observed a patient, and I have myself seen one, in whom the vomiting attacks were later in life replaced by migraine. Whether it is to be looked upon as a manifestation of the lithæmic state in children must be determined by future study. It is probable that not all the cases depend upon the same condition.

Prognosis.—Although these patients very often seem to be most alarmingly ill, the danger to life is slight. I have seen but one fatal case, and in this the diagnosis is open to question, as no autopsy could be obtained. The patient died in the eighth week of her fifth attack.

Griffith reports two fatal cases, the autopsy in one showing nothing characteristic; the symptoms in the other case were fairly typical. The probabilities are always in favour of a recurrence of the attacks. In most of the patients who have been observed they have extended over a series of several years, although by a careful régime much may be done to reduce their frequency.

Toward puberty there appears to be a strong tendency to spontaneous recovery.

Diagnosis.—Organic disease of the brain and kidneys must first be excluded, the latter only by careful and repeated examination of the urine. The first attacks witnessed may strongly suggest the onset of tuberculous meningitis; and only the course of the symptoms may show that this is not present. Usually a history of many previous attacks may be obtained. From acute indigestion, cyclic vomiting is differentiated by the fact that the attacks are not brought on by indigestible food, and also by the persistence of the vomiting. It is distinguished from gastritis by its severity, the shorter duration of its symptoms, and its self-limited course.

Appendicitis is excluded by the absence of pain, tenderness, and temperature; intussusception by the fact that the symptoms are less severe, by the absence of blood and mucus from the stools, and by the fact that most of the attacks occur after infancy.

Treatment.—When the premonitory symptoms appear, free purgation by calomel offers the best prospect of aborting an attack. If the vomiting has once begun, nothing seems to have the slightest influence in controlling it. It is usually increased by the taking of food or drink or by any medication by the mouth, and all should be withheld. The patient should be kept absolutely quiet and water given, per rectum, at regular intervals, usually six to eight ounces, four or five times a day. This keeps up the urinary secretion, allays thirst and often restlessness, and adds much to the patient's comfort. In the more protracted cases rectal feeding should be employed. When the vomiting has ceased for several hours it is not likely to recur if food is very judiciously administered, at first in small quantities. Broth, barley water, kumyss, or small quantities of iced milk and lime-water in equal proportions may then be given.

The alkaline treatment has been strongly advocated; it consists in giving between the attacks bicarbonate of soda in doses of fifteen to thirty grains three times daily, and when the prodromal signs of an attack appear, to administer very large doses, as much as thirty grains every hour. I have used this plan of treatment with some apparent success and think it deserves further trial, although sufficient facts are not yet available to enable one to speak with confidence regarding it. Acting upon the theory that the symptoms are analogous to those of migraine, the treatment I have adopted in the interval has been dietetic; it consists in excluding all sugar and sweets, and carefully limiting the amount of starchy foods. The diet prescribed has been composed principally of meat, green vegetables, milk, and stale bread. In addition to careful regulation of the diet the general nutrition should be considered, and the patient's life so regulated that extreme fatigue and exhaustion are prevented. In most cases close attention to these matters has resulted in a very great diminution in the frequency of the attacks.

GASTRALGIA.

This term is applied to sudden, severe attacks of abdominal pain. Gastralgia occurs as a symptom in most of the severe attacks of acute gastric indigestion; in such cases it is more marked in older children than in infancy. The pain of diaphragmatic pleurisy is often referred to the epigastrium, and may be so severe as to lead one to think that the stomach is the seat of disease. Another cause may be appendicitis. In vertebral caries of the dorsal region epigastric pain is a very frequent, early symptom. It is also common in children who suffer from malaria, at the onset of acute attacks, and it may be severe when the febrile symptoms are not well marked. In other cases pain in the stomach is of the nature of a true neuralgia, which may be excited by exposure to cold, by wetting the feet, by drinking ice-water, and by many other causes.

In mild cases there is an intermittent pain, and usually no other symptoms. In severe cases the pain may be so great as to cause pallor, faintness, cold perspiration, and very marked prostration. The epigastrium may be hard and sometimes retracted, the stomach appearing to be in a state of spasm.

The principal interest attaches to diagnosis. If the pain is acute, one should carefully exclude appendicitis, renal and hepatic colic, and ulcer with perforation; if more chronic, Pott's disease should not be forgotten.

Treatment.—During the attacks the patient should be put to bed, and counter-irritation used over the stomach, best by means of a turpentine stupe or a mustard paste. Internally there should be given hot water containing brandy or gin and five drops of spirits of chloroform; all food should be withheld. Hot bottles should be applied to the feet if they are cold. In the interval between the attacks the treatment should be directed to the patient's general condition; especially should the cause be discovered, and if possible removed. In cases of recurring pain of a neuralgic character arsenic in the form of Fowler's solution, two or three drops three times a day, may prove of benefit. In all cases attention should be directed to the diet.

ACUTE GASTRIC INDIGESTION.

This occurs whenever the stomach is unequal to the task imposed upon it. It may be either because the task is too great or because the capacity of the stomach for work is diminished. Under these two heads we may group the principal causes of acute indigestion.

Under the first head the most important thing is the giving of improper food. In infants this is sometimes improper breast-milk; but more often cow's milk containing too high proteids—i. e., milk without sufficient dilution. Other common causes are sudden weaning or any other abrupt change in diet, the too early use of solid food, and overloading the stomach. In older children the usual causes are indigestible articles of food, such as unripe fruits, pastry, etc., overloading the stomach, and swallowing food without sufficiently masticating it. Conditions which may diminish for the time the capacity of the stomach for work are fatigue, depression induced by atmospheric heat, chilling of the surface, especially the extremities, dentition, and the nervous impression caused by the onset of any acute disease. The effect is seen both on the glandular and muscular apparatus of the stomach. The secretions are diminished or altered in character, and the motor activity of the organ is arrested.

Symptoms.—One of the first consequences of arrested gastric digestion is that the food remains long in the stomach. Instead of being empty in two or two and a half hours after feeding, as is normal in infancy, the food may remain in the stomach five or six hours, or even longer. The irritation from this undigested mass excites vomiting, which usually ceases after the stomach has been emptied. The vomiting may be preceded by nausea, pain, and constitutional depression which varies with the age and susceptibility of the child; in infants it may be very alarming.

It seems probable that, as a consequence of arrested gastric digestion, the proteids are not converted into peptones, but remain in the form of albumoses. These products have been shown by experiments on animals to be toxic, producing stupor and circulatory disturbances. They are diffusible and are undoubtedly absorbed with great rapidity, and may be the cause of nervous symptoms of a striking character. There may be dulness, stupor, and sometimes contracted pupils, so as to suggest opium narcosis, or there may be restlessness, excitement, and even convulsions. There is also marked prostration, weak pulse, and fever. The temperature in most cases of acute indigestion is from 101° to 103° F.; not infrequently it rises to 104° or 105° F. The tongue is coated and the appetite entirely lost. In infants these symptoms are usually associated with or followed by more or less intestinal disturbance—generally diarrhœa, with undigested food in the stools. Epigastric distention may be present. Usually the vomiting ceases in from six to twelve hours, and after the stomach has been thoroughly emptied the temperature falls. Provided rest to the organ can be secured, and the exciting cause is one that can be removed, the patient may be quite well in two or three days. Relapses are, however, easily excited; and in a susceptible patient it is surprising to see how trivial a cause may excite one.

The diagnosis between a simple attack of acute indigestion and one of gastritis can not be made at the outset. The former is much more frequent, and may be quite as severe, but is of shorter duration. The continuance of the severe symptoms, especially pain, thirst, fever, and vomiting of mucus tinged with blood, justify the inference that inflammatory changes exist. The prognosis in these cases is good, except in very young or very delicate infants. In such patients an attack of acute indigestion is not infrequently fatal.

Treatment.—The indications are, to empty the stomach as completely as possible and then to secure to it absolute rest. If proper treatment is employed at the outset, the majority of such attacks can be cut short. Nothing is so efficient in infants as stomach-washing. A single washing usually suffices. If for any reason this can not be employed, the child may take from its bottle a large amount of lukewarm water. The free vomiting which this usually produces may be sufficient to cleanse the stomach fairly well, but by no means so thoroughly as stomach-washing. Persistent vomiting is sometimes arrested by giving small quantities of hot water.

The subsequent treatment is chiefly dietetic. Everything should be withheld for three or four hours, when barley water, albumin water,* or whey may be given frequently, and in small quantities—e. g., half an ounce to one ounce every hour. After twenty-four hours raw beef-juice or broth may be tried, but no milk should be given for at least three days. When begun, it should be peptonized and diluted with five or six parts of water. In a nursing child, the breast should be withheld altogether for twenty-four hours, and then nursing allowed for two minutes every three hours, the time of nursing being gradually increased to three, five, and ten minutes as improvement occurs. The great mistake made in these cases is to begin food too soon and to give too much, especially of cow's milk.

Drugs are relatively of little value. If the measures mentioned have been used promptly they will not often be required. In many cases injudicious medication aggravates the symptoms and prolongs the attack. Unless the bowels have acted freely, calomel (gr. $\frac{1}{8}$ every hour) may be given until this effect is obtained. Where there is continuous vomiting of very acid mucus and serum, alkalies are indicated—lime-water, chalk mixture, or the subcarbonate of bismuth. It is important to keep the child as quiet as possible. Local applications to the epigastrium are very often useful. Either dry heat may be applied by means of a hot-water bag or hot flannels, or more active counter-irritation by mustard. In older children the stomach is to be emptied by an emetic accompanied by large draughts of warm water. After this it should be kept entirely at rest for half a day, only carbonated waters or barley water being allowed in small quantities to allay thirst. Later, broth or beef-juice may be given, afterward milk diluted with two parts of lime-water. The patient should be kept upon a very low diet for four or five days.

ACUTE GASTRITIS.

In comparison with the frequency of inflammatory diseases of the intestine, those of the stomach are rare, particularly so in infancy. Owing largely to the character of its secretion and its contents, the stomach is much more resistant to infection than are the intestines. Gastritis seldom exists alone, but is usually associated with enteritis or colitis.

Etiology.—The causes of gastritis are, in the main, those of acute gastric indigestion—improper food or feeding—plus infection. This may be of many kinds, probably the most frequent being due to the streptococcus. Other organisms concerned are the bacillus of tuberculosis, of diphtheria, the bacillus pyocyaneus, etc. Gastritis may also be caused by the introduction of irritants, which may either be swallowed accidentally or given as drugs.

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* *Albumin water:* The white of one fresh egg, one-half pint cold water, previously boiled, a little salt, one teaspoonful of brandy; shake thoroughly, and feed cold.

Lesions.—The mucous membrane of the stomach may be the seat of acute catarrhal, ulcerative, or membranous inflammation, all forms except the catarrhal being rare. There is also seen a mixed form, which from its cause is usually termed "corrosive" gastritis.

Catarrhal gastritis.—This is characterized by hyperæmia of the mucous membrane, exudation of cells into the mucosa, a great increase in the secretion of the mucous glands, and changes in the epithelium. About the only change which can be recognised by the naked eye is congestion and swelling of the mucous membrane. These are usually more marked toward the pyloric end and along the greater curvature. There may be small extravasations of blood into the mucosa. The stomach contains undigested food and mucus, which may be thick and tenacious, adhering very closely to the mucous membrane. The mucus may be stained brown from the capillary hæmorrhages. The stomach may be either distended or contracted. Under the microscope the changes are seen to be almost entirely in the mucosa. In some places there is loss of the superficial epithelium, in others only degenerative changes in it are seen. The mucosa is infiltrated with round cells, this process being rarely diffuse, but generally occurring in patches. The blood-vessels are distended and many small extravasations are seen. Sometimes there is a moderate infiltration of the submucosa. Acute catarrhal gastritis alone is rarely severe enough to cause death. It is usually seen in cases which prove fatal from other causes, particularly diseases of the intestine.

Gastric softening (*gastromalacia*) is a condition dependent upon post-mortem changes—probably self-digestion of the stomach. It is found both where gastric symptoms were present and where they were absent. It is situated nearly always in the posterior wall, and usually covers a considerable area, about one-third or one-fourth of this wall. It is recognised by the gelatinous, translucent appearance of the walls of the stomach, which are so softened that the finger may be pushed through them without force, or that sometimes the stomach ruptures while it is being removed. This condition is rarely seen when the stomach is empty. It can scarcely be mistaken for a pathological condition, if its occurrence is borne in mind.

Ulcerative gastritis.—This was met with six times, not including tuberculous cases, in 390 consecutive autopsies upon infants in the Babies' Hospital. Three of the patients were less than four months old, and all were females. The ulcers varied from one twenty-fifth to one quarter of an inch in diameter, and usually from ten to fifty were present. They seldom extended to the muscular, and never to the peritoneal coat. The lesion was most marked in the posterior wall, toward the pyloric end and along the greater curvature. Evidences of catarrhal inflammation were present in most of the cases, and in four, of membranous inflammation. Under the microscope these ulcers resemble those of the colon. Lesions in some other part of the digestive tract were present in all but one case, in two there was thrush in the œsophagus; in three there was ulceration somewhere in the intestines. Cultures showed that two cases were due to pyocyaneus infection,* which was found to be general throughout the body.

Membranous gastritis.—This is even more rare than the varieties previously mentioned. I have met with it but four times in infants. One case was associated with a membranous colitis; a second case with pseudo-diphtheria of the fauces and larynx in an infant but six weeks old. The œsophagus was not involved in this case; and indeed it often escapes. No Klebs-Loeffler bacilli could be found either in cover-slip preparations or by culture. Both these cases have been very fully reported by Dr. Wollstein.† To the naked eye the membrane appears as of a grayish-green colour; it is adherent, but can be detached in quite large patches. Only a portion of the stomach was covered in any of the cases; in two the principal disease was about the pylorus; in another along the greater curvature. In Fenwick's case the entire surface of the stomach was lined with membrane. The microscopical appearances resemble those of membranous colitis. There is a pseudo-membrane composed of fibrin, granular matter, epithelial cells, and bacteria. The mucosa shows a moderately dense infiltration with round cells, and in places superficial ulceration. There is also infiltration of the submucosa, and in some places even the muscular coat is involved.

Membranous gastritis occurring in patients dying of diphtheria is not common. Councilman, Mallory, and Pearce noted its presence in only five of one hundred and twenty-seven autopsies.

Corrosive gastritis (*toxic gastritis*).—This form of inflammation is excited by various irritating and caustic substances, which are usually taken by accident, sometimes for the purpose of producing emesis. The most frequent substances are carbolic acid, caustic alkalies, mineral acids, arsenic, salts of copper, zinc, or antimony, croton oil, and corrosive sublimate.

The lesions in the stomach depend upon the amount of the substance swallowed, the degree of concentration, and whether the stomach was full or empty at the time. Strong caustics, whether acids or alkalies, usually act more deeply and extensively in the pharynx and œsophagus, for, owing to the spasmodic contraction of the muscles of these parts, often but a small amount of the substance reaches the stomach. Concentrated irritant poisons produce in the stomach, especially along the greater curvature, irregular ulcers, which may be so deep as to cause perforation, or they may affect the mucous membrane only. In severe cases death takes place early, often in a few hours. Dark, ragged ulcers are found in the stomach, the surrounding mucous membrane is the seat of intense congestion, and in places there are extravasations of blood. If death is delayed there are evidences of intense inflammation, sometimes with the production of a pseudo-membrane. If the amount of poison is not sufficient to cause death, and if the patient recovers from the resulting gastritis, a cicatricial condition of the stomach results, which later may lead to stenosis of the pylorus or other deformity of the organ.

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* See Martha Wollstein, M. D., Archives of Pædiatrics, 1897, p. 760, for full report.

† Archives of Pædiatrics, July, 1892.

Symptoms.—*Catarrhal gastritis* can not be distinguished at its beginning from an attack of acute indigestion. There are fever, pain, vomiting, thirst, loss of appetite, coated tongue, and prostration. The presence of inflammatory changes is indicated by the continuance of these symptoms, particularly the pain, vomiting, fever, and thirst. With the pain there may be epigastric tenderness. All food or liquids are immediately rejected, and even when nothing is taken the retching and vomiting may continue, nothing but frothy mucus or serum being brought up, sometimes streaked with blood. The vomited matters are usually very sour; they may be bilious. The temperature is rarely high except at the outset. After the first or second day it usually ranges between 100° and 101·5° F. Thirst is intense, and all liquids are taken with avidity, especially if cold, even though they are immediately vomited. The tongue is thickly coated with a white fur, and the breath may be foul. The constitutional symptoms are generally most severe at the outset. The usual duration of such attacks is from four to seven days, but with improper management, especially injudicious feeding, the disease may be much prolonged. One attack may follow another until a chronic condition is established. In most of the cases there is some disturbance of the intestines, usually a sharp attack of diarrhœa. Sometimes the gastric symptoms subside after a few days and those of the intestines become the predominant ones. The symptoms above given are those in infancy. In older children there is less of fever, prostration, and diarrhœa, but pain and vomiting are prominent. The attacks are usually shorter and altogether less severe.

The rare cases of *ulcerative gastritis* have nothing by which they can be distinguished from the form described, except a more prolonged course and a greater liability to hæmorrhage.

Membranous gastritis also presents no peculiar symptoms. In fact, in the cases I have personally seen, the gastric symptoms were insignificant, and the condition not suspected during life.

In *corrosive gastritis* the effects of the caustic may be seen in the mouth and pharynx, the mucous membrane being of a gray or whitish colour. Pain and a sense of constriction are felt in the œsophagus and stomach, and thirst is great. Vomiting follows almost immediately, and the matters vomited are usually bloody. The subsequent course in most of the cases is the rapid development of collapse, and death in a few hours from shock. The younger the child the sooner does the case terminate. In irritant poisoning not severe enough to produce death, the symptoms of acute gastritis follow, usually accompanied by more or less enteritis owing to the passage of the irritant into the intestine. There is seen a continuance of the vomiting, pain and epigastric distention, and diarrhœa, and from these symptoms death may result in two or three days. It is extremely rare in infancy for the patient to survive both the stage of shock and that of acute inflammation, so that the deformities of the stomach and the chronic conditions mentioned, are practically never met with excepting in older children.

Treatment.—Cases of acute catarrhal gastritis are to be managed very much like those of acute gastric indigestion. Thirst may be relieved by swallowing bits of ice. Where there is continuous vomiting of acid mucus, relief is sometimes afforded by repeating the stomach-washing once in twelve hours with a 1-per-cent solution of bicarbonate of soda, at 110° F. In older children, beneficial results sometimes follow the use of bismuth subcarbonate (gr. x every two hours); but in infants I must confess to have seen but little effect from any form of medication, the reliance being upon rest, careful feeding, and stomach-washing.

Cases of corrosive gastritis require special treatment. The first indication is to administer the proper chemical antidote to the substance swallowed, and the next to use bland mucilaginous or oily fluids, such as milk, albumin-water, oils in large quantities, etc. Especially should stomach-washing be avoided. Opium is always required, on account of pain, and should be given hypodermically. The general symptoms are to be treated according to the indications of the individual case.

GASTRO-DUODENITIS.

This is a catarrhal inflammation of the stomach and duodenum. Sometimes only the duodenum is involved. The inflammation commonly extends from the intestine into the common bile duct, the swelling of which causes jaundice. The term gastro-duodenitis is sometimes used synonymously with catarrhal jaundice. The condition is a rare one in young children, and especially so in infancy. I have never seen it in a child under two years old.

The causes are for the most part obscure. It occasionally complicates malarial fever. I have seen it several times with influenza, and it may occur with any of the infectious diseases. Rehn has described a form which occurred epidemically.

The symptoms of the disease are quite uniform. When primary, the onset is like an ordinary attack of indigestion, with vomiting, pain, slight fever, and a moderate amount of prostration. The vomiting in some of the cases is repeated for several days. The pain may be quite severe, and localized in the region of the duodenum. It may be associated with tenderness in this region. The bowels are usually constipated. After three or four days, icterus, which is the only diagnostic symptom, appears. It is first seen in the conjunctiva, afterward in the skin, varying in degree according to the severity of the attack, but in most cases not being very intense. It is accompanied by the regular symptoms of obstructive jaundice. The stools are gray, sometimes white; there is a marked amount of intestinal flatulence. The urine is very dark, of a yellowish-green or bronze hue, and stains the clothing. There is complete anorexia; the tongue is thickly coated with a white fur. Headache, dulness, and languor are present, and the patient feels generally wretched. The slow pulse and the itching skin are uncommon symptoms in children. The liver is usually found, upon examination, slightly enlarged, and sometimes tender on pressure. The duration of the disease is about two weeks, the general symptoms disappearing before the icterus.

The diagnosis rarely presents any difficulty, and the prognosis is invariably good.

Treatment.—In the diet, fats and starches should be reduced to a low point or be entirely prohibited. Patients usually do much better upon a diet of rare meat, fruit, and a moderate amount of milk. If there is very much vomiting, the milk should be largely diluted with lime-water or partially peptonized. The amount of food given should be small, but water should be allowed freely, particularly the mineral waters. The bowels should be opened every other day by calomel, followed by a saline purgative. In most of the cases no other treatment is necessary. When the pain is severe it may be relieved by counter-irritation by mustard, turpentine, or even cantharides. The gastric symptoms should be managed as are those of ordinary acute gastritis. The restricted diet should in all cases be continued for at least a week after the jaundice has disappeared.

CHRONIC GASTRIC INDIGESTION—CHRONIC GASTRITIS—GASTRIC CATARRH.

Although from a pathological point of view these conditions are not identical, from a clinical standpoint there is no advantage in attempting to separate them. Nothing distinguishes chronic indigestion from chronic gastritis except that in the latter, in addition to continued derangement of function, there is a great increase in the production of gastric mucus. Chronic indigestion seldom exists long without the production of a slight amount of catarrhal inflammation. This condition in the stomach seldom, if ever, exists without more or less involvement of the intestine, and in the majority of cases the intestinal condition is the more important. In some, however, the gastric symptoms predominate, and it is only those which are here considered.

Etiology.—Chronic gastric indigestion may follow acute attacks, or it may be chronic from the outset. If the latter, it depends in infancy upon the continued use of improper food or bad methods of feeding. The improper food is very often a modified cow's milk of improper proportions. Sometimes the proteids are too high, but the most frequent mistake is the use of too high a percentage of fat. As a consequence of imperfect digestion, fermentation in the residuum takes place, and the irritating products of this fermentation soon cause a catarrhal inflammation with a production of mucus, decomposition of which adds still further to the irritation. Chronic gastric indigestion also complicates most of the constitutional diseases of infancy, especially rickets, syphilis, tuberculosis, malnutrition, and marasmus. It may follow any of the acute infectious diseases. In older children it is due chiefly to the use of improper food, sometimes to the habit of rapid eating and insufficient mastication. It is associated with constitutional diseases as in infancy, and may complicate valvular disease of the heart.

Lesions.—The changes found in chronic gastritis are usually confined to the mucosa. In the mild form there are degenerative changes of the epithelium of the tubules, with increased production of mucus; there may be a slight infiltration of the mucosa with round cells. The more severe form, with marked cell infiltration and the production of new connective tissue, is extremely rare. The submucous coat may be thickened and the muscular coat attenuated. The lesion can not be recognised by the naked eye. The stomach is apt to appear more or less dilated, and its surface is coated with thick and very adherent mucus. This lesion rarely exists alone, practically never in infancy, but is associated with similar lesions in the intestines, the latter being more severe.

Symptoms.—*In infants.*—For our knowledge of the conditions existing in the stomach in chronic indigestion we are indebted to the work chiefly of Cassel, Leo, Troitzky, and Wohlmann. The results obtained in the examination of stomach contents have not been uniform, and in practice one should not lay much stress upon the absence of the normal secretions. The constant presence of mucus in the vomited matters or in the washings from the stomach distinguishes chronic gastritis from simple chronic gastric indigestion. This greatly interferes with digestion, even though secretions are normal. The reaction of the stomach is almost invariably acid. The rennet ferment is present. Pepsin is absent in about half the cases. Hydrochloric acid is generally deficient, but is increased by irrigating the stomach. The following changes are present in nearly all cases: Fermentation takes place in the fats, the carbohydrates, and in the gastric mucus. The results of fermentation are the production of lactic, acetic, butyric, and other volatile fatty acids, which are especially irritating to a mucous membrane. New products are also formed from the decomposition of the proteids, and gases are always present. Food remains long in the stomach because of motor inactivity, which is partly the cause and partly the result of the disease. It often continues after all other symptoms have disappeared.

The most important local symptoms are vomiting or regurgitation of food, vomiting of mucus, regurgitation of a sour watery fluid, belching of gas, and pain from gastric distention. Vomiting is almost invariably present, and may occur soon or long after feeding. It is often accompanied by regurgitation of food, which may begin soon after one feeding and continue in small amounts quite to the time for the next. In nearly all protracted cases the vomited matters contain mucus, and sometimes this is a conspicuous feature. The regurgitation of a sour irritating fluid occurs even when but little food is rejected, and usually accompanies the belching of gas. In infants some of the most striking symptoms are due to the gas. The stomach may be distended and hard most of the time, and often so much gas is present that infants find the greatest difficulty in taking food. Though evidently very hungry, they can take so little at a time that an hour or more may be required to take four or five ounces. That the food remains long in the stomach is best demonstrated by stomach-washing. Instead of the stomach being empty in two or three hours, as it should be, food is almost invariably found four or five hours, and in some cases six or eight hours, after feeding.

The appetite may be abnormally great, or it may be very poor. As a rule, children take less food than in health. The tongue is usually coated. The general symptoms are those of malnutrition; there is constant fretfulness and sleep is irregular or disturbed; the weight is stationary, or there is steady loss; there is also anæmia, and the child's development is arrested. There is nearly always some derangement of the bowels—constipation or diarrhœa. There may be dilatation of the stomach, especially in rachitic children, when overfeeding has been practised.

There is little tendency to spontaneous improvement or recovery, the prognosis depending almost entirely upon the treatment employed. Unless relieved the condition is apt to continue, until some serious acute disease develops which may be fatal. In young infants, chronic gastric indigestion should not be confounded with hypertrophic stenosis of the pylorus.

In older children.—The disease is not so common as in infants. In all cases the most constant symptom is vomiting, which may occur regularly after meals, or only in the morning before breakfast. If the latter, the vomited matters consist chiefly of mucus. In addition to these regular attacks there may be the frequent regurgitation of small quantities of food. There are gastric flatulence and pain, due to hyperacidity or to acid fermentation. The appetite is variable—sometimes inordinate, sometimes entirely lost; it may be capricious, there being usually a craving for highly seasoned food. The tongue is constantly furred, and the breath usually disagreeable. These symptoms are seen in all degrees of severity. Intestinal disturbances are not so frequent as in infancy. Constipation is more common than diarrhœa. The general symptoms are those of malnutrition. There are anæmia, wasting, constant fretfulness, disturbed sleep, and various other nervous disorders.

Prognosis.—The prognosis depends upon the age of the patient, the duration of the disease, the surroundings, and upon how well treatment can be carried out. In infants under three months the prognosis as to life is bad. If children live to the age of seven or eight months, they may recover with proper treatment. These patients do much better in private practice than in institutions. Much depends upon the co-operation of an intelligent mother or nurse. Chronic gastric indigestion is not dangerous to life except in young infants. Its principal danger consists in the predisposition it gives to acute diarrhœal diseases in summer, which in such patients are very likely to be fatal. It may also lead to the development of marasmus.

In older children, as in the case of infants, these symptoms may continue indefinitely; there is little tendency to spontaneous recovery, but under favourable circumstances, with constant care, much may be done for all these patients and many of them may be completely cured.

Treatment.—*Infants.*—The general treatment is too apt to be ignored, but it is just as important as measures directed more specifically to the stomach. A large, roomy nursery, and plenty of fresh air by night and by day, are very important; sometimes under the influence of these alone improvement begins. General friction of the body with cocoa-butter is useful in delicate children with poor circulation. Infants must be properly covered, and it is of the utmost importance that the feet be kept warm. Of the measures directed to the stomach, two are chiefly to be depended upon—stomach-washing and diet.

Stomach-washing (page 62) is useful, first, in removing the mucus which is so abundant in most of these cases; secondly, in cleansing the organ thoroughly at least once a day, this of itself being most important; thirdly, as a stimulant to the gastric secretions, especially hydrochloric acid. Plain boiled water, or a weak alkaline solution—sodium bicarbonate, one drachm to the pint—may be employed. In the early part of the treatment the washing should be done daily; later, every second or third day. The time selected is not very important, but it is better to make this about three hours after feeding. The mother or nurse may easily be taught to wash the stomach, so that it may be done as frequently and for as long a period as circumstances require.

The question of diet has been quite fully discussed in the chapter on Infant-Feeding, particularly in the pages in which the feeding in difficult cases is considered. If milk is being given, one should first endeavour to determine which of the elements is the chief cause of the trouble. This is most frequently the fat, next the proteids, and only rarely the sugar. The fat should be reduced, and if trouble also exists with the proteids, these should be managed in the manner indicated on pages 208–211. Where very serious and long-continued trouble exists with both the fat and proteids, a change of diet to a farinaceous food may be the most efficient means of checking the gastric fermentation. Malted foods seldom succeed.

The quantity of food and the frequency of feeding are both matters of importance. As a rule with a serious amount of chronic gastric disturbance in infants over three months old the feedings should not be less than three and seldom more than five hours apart; four hours is a good average. Small meals of a somewhat concentrated food are usually better than large feedings of a very dilute food. Careful study of the individual child is indispensable to success.

Drugs have a very limited application in the treatment of this condition in infants. Generally they are too much used, too little attention is given to the details of feeding, by which means alone permanent improvement is reached. The continued use of pepsin and hydrochloric acid has given me but little satisfaction. But for the relief of one symptom drugs may be of considerable advantage; wherever the production of gas and constant eructations are prominent symptoms, the salicylate of soda is useful. It may be given with the feeding in doses of one or two grains.

The management of these cases in older children must be conducted along the lines laid down for infants. With them, stomach-washing can not be so easily employed, and other means must be used to clear the stomach of mucus. The best is undoubtedly the use of large draughts of water, as hot as can be borne, an hour before eating. From six to eight ounces should be taken, preferably slowly by sipping. To this may be advantageously added, in many cases, fifteen or twenty grains of bicarbonate of soda.

The diet should consist of milk diluted at least three times, kumyss or matzoon, beef juice, raw meat, beef peptones, and a moderate amount of starchy food, preferably dried bread or zwieback. Sweet fruits, and in many cases all fruits, must be avoided. The amount of water taken at meal-time should be carefully restricted. Beneficial results are obtained in most of these cases by the use of nux vomica or simple bitters before meals, and the regular administration of hydrochloric acid (gtt. v to viij of the dilute acid) shortly after meals. All pastry, sweets, nuts, and candies must be absolutely prohibited. With improvement in the symptoms green vegetables may be added to the diet, and the amount of starchy food increased. The general treatment must not be neglected. The patient should lead an out-of-door life as much as possible, and regular but very moderate exercise allowed. Great caution is necessary against over-fatigue. Iron may be given in most cases during convalescence; but cod-liver oil should be carefully avoided until the gastric symptoms have quite disappeared. Relapses are easily excited, and the most constant care regarding the food must be maintained for months, or even years.

DILATATION OF THE STOMACH.

Moderate dilatation of the stomach is quite a frequent condition, although it is not so large a factor in the disorders of digestion in infancy and childhood as many who have written upon the subject would lead us to believe. A very marked degree of dilatation is rare, but in these cases its recognition is important and its treatment difficult.

Dilatation is almost invariably regular or cylindrical; it is usually most marked at the cardiac extremity (Fig. 61). Cases of irregular or saccular dilatation, except when associated with cicatricial conditions, are of somewhat doubtful occurrence. The irregular shapes of the stomach found at autopsy dependent upon the contraction of the muscular coats, may be easily mistaken for hour-glass contraction or saccular dilatation. The degree of dilatation may be very great; thus, the stomach of a child three months old measured at autopsy nine ounces; another, four and a half months old, ten ounces. The greatest dilatation I have measured during life was in a child four months old, where the stomach held twelve ounces.

In rare instances dilatation may result from congenital stenosis of the pylorus. The most important predisposing cause, however, is the muscular atony which accompanies rickets. It is found to a slight degree in almost all severe cases of rickets. The principal exciting causes are continued distention from overfeeding and chronic indigestion.

In most cases the only symptoms are those of the chronic indigestion which almost invariably accompanies dilatation. If there is pyloric stenosis, vomiting is present. In young infants the pressure symptoms may be very serious. This is particularly true in infants with acute bronchitis or broncho-pneumonia, or in those with atelectasis. In these patients I have seen very grave symptoms accompany the rapid distention of a dilated stomach, and in one very delicate infant of three months this was apparently the cause of death. A positive diagnosis of dilatation is only made by the physical signs. There are epigastric fulness and distention, and in some very thin patients the outline of the stomach can be distinctly seen. Dilatation of the transverse colon, however, may be mistaken for dilatation of the stomach. In the latter, the lower outline is convex, while in the former it is usually slightly concave. The most satisfactory means of diagnosis is by percussion. The examination should be made three or four hours after feeding, at which time the whole abdomen is apt to be tympanitic. The stomach should then be filled with water; the lower limit of the area of flatness will be the lower border of the stomach. This is much more satisfactory than determining the outline after the generation of gas in the stomach. If the lower border comes nearly to the umbilicus the stomach is dilated; if it is below the umbilicus, it is much dilated. In many cases the capacity of the stomach can be measured by simply seeing how much water can be easily introduced into it by means of the funnel and stomach tube.

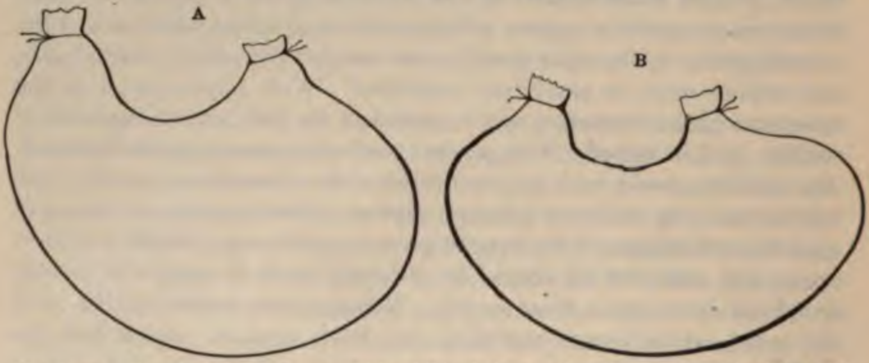

FIG. 59.—A, dilated stomach from rachitic child of six months; B, stomach of healthy child of same age. (Outlines reduced from photographs.)

In moderate dilatation of the stomach the prognosis is good except when it is due to pyloric stenosis. If the infant has any acute or chronic pulmonary disease, dilatation of the stomach may add to the discomfort and even to the danger from that condition.

In the management of these cases the first point is to restrict the use of fluids, reduce the size of the meals, and regulate the diet in accordance with the general plan outlined in the chapter on Chronic Indigestion. If the dilatation is marked, the stomach should be washed once a day. The general condition of the patient usually requires tonics, the best of which is strychnine; and rickets, if present, should receive its appropriate constitutional treatment.

ULCER OF THE STOMACH.

Ulceration of the stomach may be found in connection with several pathological processes which are quite distinct from one another:

1. Ulcers in the newly born. These have already been referred to in the chapter on Hæmorrhages of the Newly Born. The only characteristic symptom is hæmorrhage.

2. Ulcers resulting from acute gastritis. These also are not frequent (page 338). As a rule they give no symptoms except those of gastritis, although in several cases I have known severe hæmorrhage to result from them. This symptom will be considered later.

3. Tuberculous ulcers. These are quite rare. I met with gastric ulcers five times in one hundred and nineteen autopsies on tuberculous cases; however, the evidence was not conclusive in all of them that the ulcers were tuberculous; but in three the tubercle bacilli were found. Usually there were several small ulcers; in one case but two were present, the larger one being nearly three-fourths of an inch in diameter, and situated on the posterior wall near the middle of the greater curvature. All but one of these cases were in infants, one child being only ten months old. The ulcers gave no symptoms during life, and death took place from general tuberculosis. This is the history of nearly all the few cases on record. In one, however, reported by Casin, a tuberculous ulcer perforated the stomach and caused death from peritonitis. Active symptoms—bloody vomiting and bloody stools—were excited by the use of an emetic.

4. Simple perforating ulcers. These are of great rarity and uncertain pathology. I have found but five recorded cases in young children in non-tuberculous patients, two of these being young infants. Rotch's patient was but seven weeks old, and Cade's but two months. Two other cases were under four years old.

The symptoms of ulcer before perforation are gastric pain and tenderness, vomiting of blood, and often bloody stools. In most of these cases in children there were no symptoms until perforation, then followed collapse, sometimes high temperature, the rapid development of tympanites, and death from shock or from peritonitis.

The prognosis is bad in all forms of ulcer of the stomach, except the small follicular variety. In this, however, the diagnosis can not positively be made except by gastric hæmorrhage, and it is only this which makes these cases serious.

Treatment.—The treatment is absolute rest, ice, small doses of opium, rectal feeding, stimulants; later, bismuth, arsenic, or nitrate of silver. If symptoms of perforation occur the abdomen should be opened without delay, as offering the only chance of recovery.

TUMOURS OF THE STOMACH.

Although exceedingly rare, tumours of the stomach occur in childhood, and are seen even in infancy. A case of *sarcoma* of the stomach in a child of three and a half years has been reported by Finlayson (British Medical Journal, December 2, 1899). It was apparently primary. The microscopical examination showed it to be of the spindle-celled variety. This writer could find no other recorded case under the age of fifteen.

Lymphadenoma of the stomach in a rachitic infant of eighteen months has been recorded by Rolleston and Latham (Lancet, May 14, 1898). There were multiple tumours arising from the mucous membrane in the pyloric region. The case in many features resembled leukæmia.

Six cases of *cancer* of the stomach in children under ten years are collected in an article by Osler and McCrae (New York Medical Journal, April 21, 1900). Four of these were in young infants and probably congenital. One case, in a child of eight, presented the usual symptoms and lesions of the adult disease.

HÆMORRHAGE FROM THE STOMACH (HÆMATEMESIS).

The most frequent variety of hæmorrhage from the stomach, that met with in the newly born, has already been considered. (See page 105.)

I have met with three fatal cases in young infants, the eldest being fifteen months old. In the first case there were symptoms of ordinary gastro-enteritis. On the seventh day the vomiting of blood began, and was repeated about ten or twelve times during the next twenty-four hours, when death took place. The blood was quite abundant, as much as a drachm of red blood being discharged at once. At autopsy there were found in the stomach about two ounces of dark-brown fluid, but no gross lesion was discovered, and no explanation of the bleeding. This hæmorrhage was apparently capillary. In the second case there were symptoms of acute gastro-enteritis of thirty-six hours' duration. After this time there was marked abdominal distention with symptoms of collapse; then a profuse hæmorrhage from the stomach, the child dying while vomiting blood. At least half a pint was discharged. The stomach contained at autopsy two ounces of dark fluid blood, and the mucous membrane was filled with minute ulcers extending quite through the mucosa. In the third case there was no vomiting of blood, but the patient died with symptoms of internal hæmorrhage. There was blood in the upper part of the intestine, and the stomach was filled with blood; it contained many small follicular ulcers resembling those found in the previous case.

Hæmorrhage from the stomach may occur in purpura, hæmophilia, scurvy, and rarely in malaria. In young girls about puberty it may be a form of vicarious menstruation. Occasionally blood may be vomited in cases of hæmorrhagic measles. Two cases are reported in which fatal hæmorrhage followed the swallowing of a foreign body. In both, vomiting of blood occurred long after the original accident. In one case two and a half years had elapsed. The autopsy in this case showed impaction of the foreign body and ulceration into the arch of the aorta. Spurious hæmorrhages may occur where blood has been swallowed and then vomited. The source of this is most frequently the nose or pharynx. It may happen in infants at the breast, where the blood is drawn from a fissure or ulcer in the nipple. The amount of blood vomited under these circumstances may be large enough to be quite alarming. It may be recognised by the child's general condition being normal, and by the presence of fissures or ulcers upon the nipple. It may sometimes be noticed that the vomiting of blood follows nursing from one breast and not from the other.

Symptoms.—There may be no symptoms except those of internal hæmorrhage, but this is rare. Usually there is vomiting of blood, and blood appears in the stools. If the hæmorrhage is rapid and vomiting speedily occurs, the blood may be of a bright-red colour. If it has been long in the stomach it is of a dark-brown or black colour resembling coffee-grounds. The stools containing blood from the stomach are black and tarry in appearance. The general symptoms will depend upon the amount of blood lost.

In a case where blood is vomited, the first point is to distinguish spurious from true gastric hæmorrhage. The nose and pharynx, especially its posterior wall, should be carefully examined. If the child is at the breast, the nipples should be examined. In older children it is important to distinguish vomiting of blood from hæmoptysis. This distinction is to be made in accordance with the rules laid down in text-books on general medicine. The prognosis is bad if the hæmorrhage is due to ulcer, if it is very profuse, or if it occurs in young infants. When it occurs in connection with constitutional diseases the prognosis depends upon the original disease.

Treatment.—Altogether the most efficient remedy is the suprarenal extract. It may be given very freely, at least two grains every half hour to a child of one year. The patient should be kept quiet, preferably upon the back; if there are signs of collapse, stimulants may be given hypodermically or by the rectum. No food should be given by the stomach for at least twenty-four hours after the hæmorrhage has ceased.

CHAPTER VI.

DISEASES OF THE INTESTINES.

MALFORMATIONS AND MALPOSITIONS.

MALFORMATIONS are not very frequent, but are of great variety. With the exception of those situated at the lower end of the intestine they are not of much practical importance, for the condition is such ordinarily as to be incompatible with life. Malformations may be met with at any point in the canal, but most frequently in the rectum and anus. Aside from these, malformations of the large intestine are much less common than those of the small intestine.

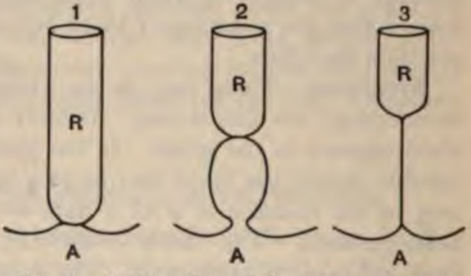

FIG. 60.—Malformations of the rectum. A, anus; R, rectum.

Malformations of the Rectum.—In Fig. 60 are shown the usual varieties of malformation of the rectum. The most frequent is atresia of the anus (1). In this the cutaneous septum has not been absorbed, but the intestine is normal to its lower extremity. This form is readily curable by a surgical operation. In the next variety (2) the cutaneous orifice and the lower part of the rectum are normal, but a membrane separates this portion from the upper part of the gut; this is usually situated within two or three inches of the anus. The bulging of the lower part of the distended intestine can usually be felt by the finger in the rectum, and a simple division of the membrane by a guarded bistoury may relieve the condition. The third form (3) is more serious. Here the rectum terminates in a blind pouch at a variable distance from the anus, and is represented below by an impervious fibrous cord. The diagnosis of this condition can not positively be made without opening the abdominal cavity. The bulging of the intestine appreciable by the finger in the rectum, is the only point which differentiates the preceding variety from this one. Instead of atresia of the rectum there may be stenosis of varying degrees, which may give rise to the usual symptoms of stricture. This is often curable by dilatation.

Malformations of the Small Intestine.—There may be stenosis or atresia at any point, often at many points. Obstruction is much more frequent in the upper than in the lower part of the small intestine, the most common seat being the duodenum.* Atresia is more often seen than stenosis. There may be a single point of obstruction, or the lumen of the intestine may be obliterated for a considerable distance, the intestine being represented only by a fibrous cord which connects the two open portions, or there may be no connection between them. In all cases the intestine above is found very greatly distended, while that below is empty and usually atrophied. The causes of these multiple deformities are mainly two—fœtal peritonitis and volvulus.† In fœtal peritonitis there are usually found bands of adhesions between the intestinal coils, and between the intestine and the solid viscera. Syphilis has been assigned as a cause in many cases. Volvulus, or a twisting of the intestine during its development, is a more satisfactory explanation for the majority of the cases, especially where there are multiple points of atresia. All these conditions are beyond the reach of surgical treatment. The symptoms appear soon after birth and are those of intestinal obstruction. (See page 117.) The higher the point of obstruction the shorter the duration of life; it is rarely more than a week in any case of atresia; in stenosis it may be two or three months.

Meckel's diverticulum.—This is the remains of the omphalo-mesenteric duct, which in fœtal life forms a communication between the intestine and the umbilical vesicle. It is given off from the ileum, usually about a foot above the ileo-cæcal valve. Most frequently it exists as a blind pouch from one-half to two or three inches long, communicating with the intestine. At the extremity of this there may be a fibrous cord, which is free in the abdominal cavity or attached to the umbilicus. In other cases the duct may remain pervious quite to the umbilicus, so that there is a fæcal fistula. Prolapse of the mucous membrane of the duct may lead to an umbilical tumour. (See page 114.) Meckel's diverticulum, especially when present as a cord connecting the ileum with the umbilicus, may compress a coil of intestine, leading to obstruction or even strangulation. This may occur in infancy or later in life.

Malpositions.—The ascending colon may be found upon the left side. There may be a complete transposition of the abdominal viscera. In cases of congenital umbilical hernia a large part of the intestines may be found in the tumour, and in diaphragmatic hernia they may be in the thoracic cavity.

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* See Cordes, Archives of Pædiatrics, June, 1901, for a report of fifty-seven cases.

† Silbermann (Jahrb. für Kinderh., Bd. xviii, p. 420); Gaertner (Jahrb. für Kinderh., Bd. xx, p. 403).

DIARRHŒA.

The term *diarrhœa* is used to cover all conditions attended by frequent loose evacuations of the bowels. These depend upon an increase in peristalsis and in the intestinal secretions.

The importance of diarrhœal diseases in children can best be appreciated by reference to the following table showing the mortality of diarrhœal disease in children under two years as compared with that from certain infectious diseases for all ages.

Deaths in New York City for Five Years.

	1900.	1901.	1902.	1903.	1904.	Totals.
Measles, all ages	816	449	710	508	895	3,378
Scarlet fever, all ages	465	1,162	940	734	851	4,152
Pertussis, " "	584	289	606	324	197	2,000
Typhoid, " "	718	727	764	653	661	3,523
Diphtheria, " "	1,920	2,068	2,015	2,190	2,084	10,277
Total deaths from five diseases						23,330
Diarrhœal disease under two years	5,744	5,796	4,938	4,439	5,646	26,563

There are several important underlying factors upon which diarrhœal diseases depend. Their great frequency belongs to the first two years of life; after this time a notable diminution both in frequency and severity is seen, and a fatal outcome is relatively rare. The extreme susceptibility of infancy is due to several causes. The digestive organs are severely taxed to provide for the needs of the growing body. The mucous membrane of the gastro-enteric tract is very delicate in structure, and has not much resistance; it is constantly exposed to injury by irritation, and to infection.

The next most striking fact about diarrhœal diseases is their prevalence during the summer season. This is graphically shown in Figs. 61 and 62, where are given by months the cases treated in a large New York dispensary for ten years, and the mortality records for the entire city during the same period. The enormous increase in the number of cases occurring in the summer months does not have reference to any single form of diarrhœa, but to all forms.

While diarrhœal diseases are especially frequent in cities and among the poor, still they are not essentially diseases of the city or of poverty. Severe and even fatal cases are constantly met with among all classes and in all places. Diarrhœal diseases are not essentially filth-diseases;

yet their frequency and severity are both increased by want of cleanliness in apartments, and in the persons and clothing of infants, especially the napkins, chiefly because these lead to a contamination of the food. Poverty and bad surroundings predispose to diarrhœa in summer, just as they do to other forms of acute disease in the cold season.

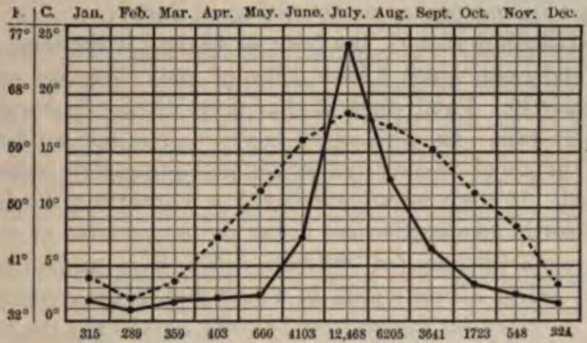

FIG. 61.—Mortality from diarrhœal diseases in New York for ten years in children under five; compared with the mean temperature for the same period. ———, mortality; - - - - -, mean temperature. (Seibert.)

But more important still is the sort of care that the infant receives. Intelligent care, even in very poor surroundings, may enable children to escape serious diarrhœa in summer. This result is due not only to the care of the person, but includes intelligent management of feeding, without which all methods are alike unsuccessful.

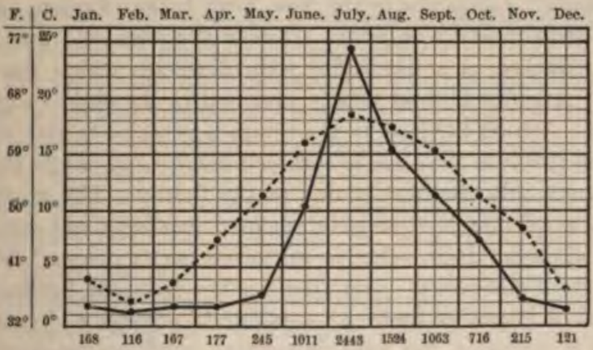

FIG. 62.—Cases of diarrhœal disease treated in the German Dispensary (New York) in ten years in children under five; compared with the mean temperature for the same period. ———, cases of diarrhœa; - - - - -, mean temperature. (Seibert.)

Anything which lowers the general vitality increases the liability to diarrhœal diseases. Marasmus, malnutrition, and rickets are especially important factors.

There are cases in which diarrhœa and dentition are closely associated, for the bowels quickly become normal when the teeth have pierced the gum. These cases, although rare, do occasionally occur. The infrequency of diarrhœa during dentition in the cold season, is the best argument against its importance as an etiological factor.

Of all etiological factors, the form of feeding is the most important. Of 1,943 fatal cases which I have collected, only three per cent had the breast exclusively. Fatal cases of diarrhœal disease in nursing infants are extremely rare. In most cases, however, it is not artificial feeding *per se,* but artificial feeding ignorantly and improperly done, which is to be blamed. If cow's milk is employed as a substitute for breast-milk, the differences in composition are either not appreciated or else ignored, so that many artificially-fed children suffer from malnutrition. The comparative safety of cow's milk in winter and in the country, however, shows that the chemical composition of cow's milk is not the most important factor. Another common and very serious mistake is that of over-feeding. Artificially-fed children are almost always over-fed. The common practice of feeding an infant every time it cries, or of keeping the bottle at its mouth the greater part of the time, is productive of untold harm.

The feeding of impure milk is an important cause of diarrhœa, especially among the poor in cities during the summer. The different ways in which milk may be contaminated have already been considered in a previous chapter. It is surprising to see how quickly diarrhœa is excited by impure milk. I once saw in the New York Infant Asylum every one of the twenty-three healthy children, all over two years old and occupying one ward, attacked in a single day with diarrhœa which was traced to this cause. Articles of food totally unsuited to the child's digestion are often given. Among the poor it is a common practice to give all kinds of solid food to children from six to eighteen months old, while those of two years often get only the regular diet of the family. The great majority of the attacks of diarrhœa in children over two years old can be traced directly to improper food, often to unripe or partly decayed fruit.

The factors mentioned—over-feeding, too frequent feeding, and the habitual use of improper food—all combine to produce a chronic indigestion which is probably the most important predisposing cause of diarrhœal diseases.

The opinion has long been held that some close connection exists between bacteria in milk and the prevalence of diarrhœal disease in summer. In the years 1901 to 1903 an investigation * was undertaken by the Rockefeller Institute in co-operation with the Health Department of

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* The full report of this investigation was published by Prof. William H. Park and the author in the Medical News, December 5, 1903.

New York to secure more definite data regarding the following points: (1) The results in infant-feeding obtained with milk of different degrees of purity both in winter and in summer, as shown by the gain or loss in weight, the amount of gastro-intestinal disturbance, and the death rate; (2) the relation, if any, existing between the number of bacteria present in the milk and the frequency of diarrhœal disease; (3) whether any organisms with pathogenic properties could be found in milk to which diarrhœal disease could be ascribed as a cause; (4) whether the practice of heating milk—pasteurization or sterilization—affected the results obtained with any given milk; (5) to what degree older children as well as infants were affected by bacterial contamination of milk.

Altogether observations were made upon 592 bottle-fed infants living in tenements of New York; 202 were observed in winter and 390 in summer. The infants were well when the observations were begun, and were watched for a period of about three months, being visited regularly by physicians, who gave advice when needed. For some of the children no change was made in the milk which they were already taking; for others special milk was provided. Samples of milk as fed to the children were frequently examined as to the number and character of the bacteria present. Observations were possible upon infants taking (1) condensed milk, (2) the cheapest grade of store milk, such as is usually purchased by the poor, (3) a better grade of milk delivered in bottles, (4) the best bottled milk sold in the city, all of the above being prepared at home, (5) milk modified at central distributing stations and furnished to patients in separate feeding-bottles.

During the winter period of observation, the mortality was but 2.5 per cent, and in but one instance was death due to disease of the digestive tract. The health of the infants observed was not appreciably affected by the kind of milk nor by the number of bacteria which it contained. The different grades of milk varied much less in the amount of bacterial contamination in winter than in summer, the cheap store milk averaging only about 750,000 per c.c.

During the summer period, the mortality was 10.5 per cent, four-fifths of the deaths being due to diarrhœal disease. At this season the kind of milk influenced greatly both the amount of illness and the mortality. The worst results were seen in those who took the cheap grade of store milk and those who took condensed milk; the best results in those who took the best grade of bottled milk, or modified milk from central distributing stations.

The number of bacteria which may accumulate in milk before it becomes noticeably harmful to the average infant in summer, differs with their nature, the age of the milk and the temperature at which it has been kept. Of the usual varieties present, no strikingly deleterious results were seen until the number approached the one million mark. If much above this point, however, the injurious effects were usually manifest. But below it other factors rather than the number of bacteria seemed of greater importance. Thus in the use of condensed milk, prepared as it usually was with hot water, the bacterial contamination was relatively small, yet the results were almost as bad as with the most highly contaminated milk.

An effort was made to discover whether a relationship existed between any special forms of bacteria present in city milk and the health of children. The observations were continued for two years and altogether the pathogenic properties of 139 varieties of bacteria isolated from milk were tested upon animals in various ways, chiefly by feeding pure cultures to young kittens. The results were entirely negative. Nor could a relationship be established in any other way between any special form of bacteria in milk and the summer diarrhœas of infancy.

To test the effect of heating milk, observations were made in the summers of 1901 and 1902 upon 92 infants who were taking the modified milk prepared at a central depot. The milk used was from a good farm, and had been kept properly cooled. The infants were divided into two groups as nearly alike as possible in their surroundings and in the care they received. To one group the milk was given pasteurized (165° F. for thirty minutes), to the other group it was given raw. All the infants were well at the beginning of the period of observation. The results are shown in the following table:

FOOD.	Total number of infants.	Remained well entire summer.	Had severe diarrhœa.	Average days diarrhœa.	Deaths.
Pasteurized milk containing 1,000 to 50,000 bacteria per c.c. at the time of use....................	41	31	10	4	1
Raw milk containing 1,200,000 to 20,000,000 bacteria per c.c. at the time of use................	51	17	34	11½	2

Thirteen of the fifty-one infants on raw milk were changed before the end of the season to pasteurized milk because of serious diarrhœa; but for this the results with raw milk would have been even more unfavourable. A similar experiment was made a third season with almost identical results. Although the number of cases is not large, the results, which were practically uniform for three successive seasons, show unmistakably that in hot weather fairly pure milk given raw, causes illness in a much larger number of cases than when it has been previously heated. However, a considerable percentage of infants apparently do quite well upon raw milk.

Sterilized milk cannot be kept indefinitely, owing to the development of spore-bearing bacteria. Although heating may destroy all the lactic acid groups, which cause souring of milk, such milk, if kept at summer temperature for any considerable length of time (over twenty-four hours), may contain immense numbers of other bacteria, and be very poisonous although not sour. This indicates the particular danger which may come from the general sale of pasteurized or sterilized milk, which is popularly supposed to be safe for two or three days, even without ice.

After the first two years, children are less and less affected by bacteria in milk. The observations seemed to show that milk from healthy cows, produced under cleanly conditions and kept at a temperature below 60° F., although containing large numbers of bacteria, sometimes amounting to many millions per c.c., might be taken in considerable quantities and for long periods by children over three years old, without any appreciably harmful effects resulting either from the living bacteria or their toxins. A single example is typical of a number of observations made. An orphan asylum, containing 650 children from three to fourteen years old, used during an entire summer, milk in which the bacteria ranged from 2,000,000 to 20,000,000 per c.c.; yet during this period there occurred no case of diarrhœa of sufficient severity to call a physician. The milk was kept cold (below 60° F.) until used; but was given without sterilization.

Mere numbers of bacteria certainly appear to count for much less than was once supposed. But the fact should not be overlooked that milk abounding in bacteria because of careless handling is also always liable to contain pathogenic organisms derived from human or animal sources. An important factor is the temperature at which the milk has been kept. If this is above 60° F., poisons are much more likely to develop, as the history of many epidemics of ptomaine poisoning from milk shows.

The Different Varieties of Acute Diarrhœa.—*Mechanical diarrhœa.*—This includes cases in which diarrhœa is produced by foreign bodies, or substances taken as food which virtually act as foreign bodies: such are partially cooked rice or other cereals; green corn, radishes, celery, cabbage, or other vegetables; nuts and unripe fruits. The irritation caused by such substances may produce only increased secretion and peristalsis by which the offending articles are removed, or, if sufficiently severe and continued, it may lead to actual inflammation of the mucous membrane of the intestine.

The indications for treatment are first to give an active cathartic, and, after thorough evacuation of the bowel has taken place, to quiet the excessive irritation by opium. For two or three days after such an attack the diet should be very light, and of such a character as to leave but little residue. The patient should be kept quiet, preferably in bed, until the stools are quite normal.

Diarrhœa from drugs.—In susceptible infants any of the ordinary cathartics may cause an attack of diarrhœa, because the physiological effects have been either exaggerated or prolonged. It is doubtful whether such attacks are often produced in nursing infants by cathartics taken by the mother.

Diarrhœa from nervous influences.—Certain nervous impressions seem to be able to produce diarrhœa when no other factors are present. The most important are chilling of the surface, depression caused by atmospheric heat, fatigue, exhaustion, fright, and dentition. It is a characteristic of many of these cases, that the taking of food into the stomach immediately excites a movement of the bowels. The chief abnormal condition in such cases is exaggerated peristalsis. This is best controlled by rest and opium.

Eliminative diarrhœa.—This term has been applied to cases in which diarrhœa is evidently an effort on the part of Nature to rid the body of some irritant or toxic product. The best-known example is the diarrhœa of uræmia. It is, however, very probable that the diarrhœa of many acute infectious diseases belongs in this category.

Acute intestinal indigestion.—Diarrhœa is a constant symptom of this condition, which is of such importance that it will be subsequently considered at length.

Diarrhœas of infectious origin.—In the forms of diarrhœa above enumerated there are no lesions, and the bacteria found in the stools are the ordinary bacteria of the intestines. There is merely altered functional activity, both motor and secretory; so that the normal chemistry of digestion is disturbed. All other forms of acute diarrhœa are to be regarded as infectious.

All infectious diarrhœas are associated with some anatomical lesions, the extent and severity of which depend upon the nature and degree of the infection and the duration of the process. In the mildest cases and in those of short duration, even though severe, the lesions involve chiefly or solely the epithelial lining of the intestine. These changes may be compared to acute degenerations of toxic origin in other organs, the kidney, for example. Nearly the whole intestinal tract is usually affected, and often the stomach in addition. The symptoms in this group of cases are due not so much to the anatomical changes as to functional disturbance and to the toxins produced in the intestine. These act as local irritants, and are absorbed into the circulation, producing the constitutional symptoms of the disease.

These cases have been classed as *acute gastro-enteric intoxication.*

In the more severe forms and in cases of longer duration more extensive lesions are present. The epithelium is destroyed; the bacteria penetrate into the deeper layers of the intestines, producing lesions which vary greatly in character and degree. They are important as modifying the symptoms, course, and termination of the disease. These cases are sometimes classed as inflammatory diarrhœa; here, from the location of the lesions, they are grouped under the term *ileo-colitis*.

The pathological relation existing between the different forms of diarrhœal disease is a very close one. The same case may pass successively through the stages of acute indigestion, gastro-enteric intoxication, and ileo-colitis. This transition may be very slow, or it may be so rapid that the different stages can not be distinguished. Instead of passing through the entire series, the process may stop at any stage and the case recover, or it may at any stage prove fatal.

ACUTE INTESTINAL INDIGESTION.

In infants, acute indigestion is seldom limited either to the stomach or to the intestine, although in one case the disturbance of the stomach is slight and that of the intestine serious, and in another the reverse may be observed. In these little patients the intestinal symptoms are much more frequent, and as a rule they are more severe than those referable to the stomach. There will be considered in this connection only the intestinal symptoms of acute indigestion; the gastric symptoms have already been described. It should be remembered that these may be seen in all possible combinations. In older children it is not uncommon to see the intestinal symptoms alone.

Etiology.—The causes are essentially the same as those mentioned under Acute Gastric Indigestion—the use of improper food, over-feeding, sudden change of food as in weaning, or the change from some other food to a rich breast-milk; also various conditions affecting the nervous system, such as heat, cold, fatigue, or the onset of any acute disease. A predisposition to such attacks is furnished by summer weather, a delicate constitution, a feeble digestion, and by previous attacks of any intestinal disorder. In susceptible children, both infants and those who are older, the slightest error in feeding may induce an attack.

Symptoms.—In infants, if the attack develops suddenly, gastric symptoms are usually present; if more gradually, they are usually absent. The local symptoms are colicky pain, tympanites, and later diarrhœa. The important constitutional symptoms are fever, prostration, and various nervous disturbances. In older children the pain generally precedes the diarrhœa by some hours, and is referred to the region of the umbilicus. Pain is indicated by the sharp, piercing cry, great restlessness, and drawing up of the legs. Tympanites is rarely very marked.

The stools are always increased in number and are from four to twelve a day. If more frequent they are very small. The first stools are more or less fæcal, but this character is soon lost. In infancy the colour is first yellow, then yellowish-green, and finally often grass-green. Wegscheider has shown that this colour is due to biliverdin. The exact nature of the process in the intestine, in consequence of which biliverdin takes the place of bilirubin as the colouring matter of the stools, is still a disputed point, but in infancy this change in colour is nearly constant. The reaction of the stools is almost invariably acid. The odour may be sour, or it may be very foul. The stools are much thinner than normal, and frothy from the presence of gases. Blood is not present, nor is mucus seen, unless the symptoms have lasted several days. Undigested food is always present; in infants upon a milk diet, this occurs as fat or lumps of casein. Fat may appear as small, yellowish-white masses resembling casein, but distinguished by their solubility in equal parts of alcohol and ether. Casein masses are more numerous, larger, and whiter. Unchanged starch may be recognized by the iodine reaction. The microscope shows, in addition to food-remains, mucus, epithelial cells, and bacteria. Epithelial cells, usually of the cylindrical variety, are numerous in proportion to the severity and duration of the attack. The bacteria are the ordinary forms found in the fæces.

In the cases with sudden onset the temperature is invariably elevated. In infants it ranges from 102° to 105° F.; in older children from 100° to 103° F. The high temperature does not continue. Usually after twelve or twenty-four hours it falls nearly or quite to normal. In the cases with a more gradual onset, or in those of a less severe character, the temperature does not often go above 101° F. The general prostration, like the temperature, is greatest in infants and in the cases beginning abruptly. It is sometimes so severe as to threaten life. There are seen rapid pulse, pallor, drawn features, and general muscular weakness. There may be restlessness, due to pain and the general discomfort, or there may be dulness, apathy, or convulsions.

The course and termination of the disease depend upon the previous condition of the patient, the nature of the exciting cause, and the treatment employed. In a previously healthy child, if the cause is at once removed and proper treatment instituted, the severe symptoms rarely last more than a day or two, and in four or five days the patient may be quite well. In delicate infants, a severe attack of acute intestinal indigestion in the hot season is likely to prove the first stage of a pathological process which may continue until serious organic changes in the intestine have taken place. This result may not follow the first attack, but one is often succeeded by others until it occurs. If circumstances are such that proper dietetic treatment and general hygienic measures can not be carried out, this termination is very common.

Diagnosis.—It is impossible to recognize an attack of acute intestinal indigestion until the diarrhœa begins; the previous symptoms of fever, prostration, etc., are seen in many infantile diseases. From the other forms of diarrhœa, this is distinguished by its brief duration, although its symptoms may be very alarming. The nervous symptoms are usually less marked than in gastro-enteric intoxication, and vomiting is less frequent.

Prognosis.—Such attacks do not endanger life except in very young or very delicate infants, in whom they may be fatal. The worst feature of most cases is that such attacks predispose to more serious intestinal diseases, many of which have their origin in acute indigestion which has been either neglected or badly managed.

Treatment.—The same general plan is to be followed as in cases of gastric indigestion—viz., first to empty the bowels as completely as possible of all decomposing or irritating masses of food; secondly, to secure to the patient, and especially to the digestive organs, as complete rest as possible. For the first indication nothing is better than calomel, which may be given in one-eighth-grain doses, and repeated every hour until the full effect is seen. Any other active purge, such as castor oil or syrup of rhubarb, may be substituted. Thirst is always great on account of the fever and the loss of fluid by the stools, but digestion even in the stomach is feeble, and often arrested altogether. For the first twenty-four hours no plan succeeds better than that of withholding everything in the shape of food, giving to allay thirst such articles as whey, albumin-water, mineral waters, or cold boiled water. Small quantities must be given—i. e., one to four teaspoonfuls—but the interval may be as short as ten or fifteen minutes. If the prostration is very great, stimulants may be needed. Brandy is the best form for their administration. After the offending materials have all been swept from the intestine, but never before, opium may be given in doses large enough to control the excessive catharsis. For a child a year old, one-quarter grain of Dover's powder after each stool is usually sufficient, and often a smaller dose may answer the purpose.

The difficult problem is to feed these cases during the latter part of the attack. In nursing infants, the breast may be given after twenty-four hours, the nursing interval being six hours, and the time of one nursing not longer than five minutes. Between the nursings other food may be given. In the case of infants past the nursing age, or those who are being artificially fed, cow's milk should be withheld in all forms for three or four days, and the child kept upon a diet of broths, farinaceous or malted foods. As improvement continues milk may be cautiously and very gradually added, at first to one or two feedings each day, and later to every feeding. It should be boiled. Since the fat is especially likely to cause disturbance, plain milk diluted is better than a milk-and-cream mixture. In some cases there is an advantage in using partially or completely peptonized milk.

In the acute stage the diet of older children should be much like that of infants. Later it should consist of meat, broths, eggs, boiled milk, and a small quantity of dried bread. All cereals, vegetables, and especially all fruits, should be withheld for some time, and then given only in small quantities, and the effect on the stools closely watched. Kumyss, buttermilk, and matzoon are frequently better borne than plain milk.

The use of drugs in these attacks, except those already referred to as indicated during the early stage, seems to me to influence the disease very little. Sometimes good results follow the giving of the extractum pancreatis half an hour after meals, or some of the preparations of malt when farinaceous food is first allowed. If the diarrhœa following the acute symptoms is prolonged or excessive, it usually indicates that either intestinal infection or inflammation is present, and the case should be treated accordingly. General measures, especially rest, frequent bathing, fresh air, and change of air, are very important in the management of all these cases, especially when they occur during the summer.

CHAPTER VII.

DISEASES OF THE INTESTINES.—(Continued.)

ACUTE GASTRO-ENTERIC INTOXICATION.

Synonyms: Summer diarrhœa, gastro-enteritis, cholera infantum, mycotic diarrhœa.

This is the form of diarrhœa which is so prevalent in summer. It occurs regularly each season, being epidemic in most large cities of the temperate zone. The lesions in the intestines are slight, amounting in most cases only to a superficial catarrhal inflammation, often bearing no relation to the severity of the symptoms which are due mainly to the absorption of toxic materials, the result of the putrefactive changes in the stomach and intestine. This form of diarrhœa may follow closely upon an attack of acute indigestion, in which it very often has its beginning. When the infection is of sufficient intensity and duration, it leads to the development of marked structural changes in the intestine, especially in the lower ileum and the colon. Acute gastro-enteric intoxication thus stands midway between acute indigestion and ileo-colitis.

Etiology.—Among the causes of acute gastro-enteric intoxication are to be mentioned, first, those which give rise to acute indigestion, and, secondly, the general factors mentioned as predisposing to all forms of diarrhœal disease—age, surroundings, constitution, food, and methods of feeding. The most striking thing about these cases is their prevalence during hot weather. While all varieties of diarrhœa are more frequent in summer, it is the form under consideration which is especially prevalent. Year after year are repeated in New York the conditions which are graphically represented in Figs. 61 and 62—viz., an epidemic which, beginning in June, rapidly increases in severity, reaching its height in July, from which time it diminishes steadily during August and September, regularly coming to an end in October. What is true of New York is true also of Philadelphia, Baltimore, and other large American cities, as well as of Berlin and other cities of central Europe. A study of these charts shows that while the mean temperature rises gradually during April and May, it is not until June is reached with its mean temperature of 61° F., that any notable increase in diarrhœal diseases begins. It appears then that an average mean temperature, or, according to Seibert, an average minimum temperature, of about 60° F. is needed to start the epidemic. Not many cases are seen until such a temperature has lasted for some days, usually about a week. The epidemic then begins in force and increases in severity through July. The explanation of the high mortality of this month appears to be, not the 4° or 5° F. by which the temperature of July exceeds that of June and August, but that the majority of the susceptible infants are unable to withstand the first very hot month. Humidity and rainfall, according to the careful investigations of both Seibert in New York and Baginsky in Berlin, do not influence either the prevalence of summer diarrhœa or its mortality.

The action of heat in producing diarrhœa was formerly regarded as a direct one. Severe cases were looked upon as examples of heat stroke or thermic fever. If such a thing exists it must be regarded as extremely rare. There is, however, no doubt that the constitutional depression produced by high atmospheric temperature does seriously interfere with digestion, and that acute indigestion so produced is very often the first stage in the pathological process, and prepares the way for infection. The view almost universally held at the present time regarding summer diarrhœa is that it is of infectious origin.

Despite the fact that since 1886 many series of bacteriological studies of the intestinal discharges have been made by Booker and Park in this country, by Baginsky, Escherich, and others in Germany, our knowledge of this subject is still very incomplete. The conditions are exceedingly complicated, and the problem is a very difficult one. So far as is now known, no one form of bacteria can be assigned as the cause of this group of diarrhœas. The evidence seems to be conclusive that the Shiga bacillus may, in a certain percentage of cases, produce diarrhœal disease of this type. It is, however, wanting in so large a proportion of cases, that it cannot be regarded as the specific cause. With existing knowledge it seems probable that there are a number of organisms present in the intestines in slight disorders of digestion which, under favourable conditions, may multiply to such a degree as to produce very serious disease.

There are certain cases in which toxic symptoms of a severe type develop abruptly in children previously quite well. These only are to be regarded as examples of acute milk poisoning. Although the bacteria in the milk may have been previously destroyed by sterilization, the toxins produced by them may still be present. This is doubtless the explanation of the simultaneous development of several cases in families or institutions.

With our present knowledge we can not believe that direct contagion is the usual way in which this disease is spread. When occurring in institutions or in families, it usually happens that a number of children are attacked simultaneously rather than successively, this indicating a common cause, usually to be found in the food. However, disinfection of stools and napkins is indicated in all cases.

Relation of the different etiological factors.—The predisposition to attacks of summer diarrhœa is partly general and partly local. The general influences are age (under two years), feeble constitution, unhygienic surroundings, and a condition of general malnutrition dependent upon improper food or feeding. The most important of the local causes is a previous derangement of digestion. In addition there may be present a low grade of catarrhal inflammation. The attack may begin as acute indigestion, not infrequently the direct result of high atmospheric temperature. In consequence of the presence of undigested food in the stomach or intestines there are furnished conditions in which bacteria, previously present in small numbers, may multiply very rapidly; bacteria may be introduced in such numbers and of such virulence as to overpower the digestive organs; or, finally, bacterial products may be ingested with the food, requiring only absorption to produce their effects.

Lesions.—The statements which follow are based upon a study of forty autopsies, in twenty-two of which microscopical examinations were made. The lesions may be briefly described as a superficial catarrhal inflammation affecting the entire gastro-enteric tract, although it varies much in severity in the different regions and in the different cases. The colon, the lower ileum, and the stomach, are apt to suffer most, the duodenum and the jejunum least.

The gross appearances.—These are usually disappointing, and may often show but little that is abnormal. The stomach is distended with gas, and contains undigested food. Its walls may be coated with mucus. The upper part of the small intestine is empty. The lower portion contains particles of food, and yellow, gray, or green material, often offensive, resembling the stools passed during life. The transverse colon, the cæcum, and sigmoid flexure are apt to be distended with gas, and contain materials similar to those mentioned, while the rest of the large intestine is usually empty and its walls contracted. It may be coated with mucus. The mucous membrane of the stomach may show intense congestion, generally in patches, or it may be pale. The mucous membrane of the small intestine may be pale throughout; there are often irregular areas of congestion, or a very intense congestion of a large part of its surface, particularly in the ileum. With this there may be redness and swelling of Peyer's patches and the lymph nodules (solitary follicles). In the colon the mucous membrane is congested, especially upon the rugæ. This congestion may be general or in patches. The lymph nodules are usually swollen; but this may be due to an antecedent process, and not to the final attack. There is no thickening of the intestinal walls. The changes described are not at all uniform, and do not differ very greatly from the appearances often seen in the intestines when patients have died of other diseases.

In the cases classed clinically as cholera infantum, the pathological changes are more characteristic. The greater part of the small intestine, and sometimes the entire colon, are distended with gas, and contain material of a grayish-white colour about the consistency of a thin gruel. It has a mawkish odour, but usually not a very offensive one. The mucous membrane of the entire intestinal tract has in most cases a pale, "washed-out" appearance. Sometimes this is seen only in the small intestine, while there are areas of congestion in the colon. If cholera infantum has been ingrafted upon some other pathological process in the intestines, as is not infrequent, there is found post-mortem evidence of this in the form of severe catarrhal inflammation, sometimes old ulcerations. In some cases, where the symptoms have been those of choleriform diarrhœa, there are found evidences of an intense diffuse gastro-enteritis, as shown by congestion of the stomach and almost the entire intestinal tract, with swelling of the mucous membrane, and especially of Peyer's patches.

The microscopical appearances.—Unless autopsies are made very soon after death—at least within four hours—it is not safe, in most of the cases, to draw conclusions from the conditions found, as post-mortem changes take place so readily in the intestines, and these changes are so like those of the disease under consideration. This applies particularly to the condition of the epithelium. One should also be cautious in interpreting the appearances of portions of the intestine which have been greatly distended with gas.

The essential lesion consists in degenerative changes in the epithelium of the stomach and intestines. The cells may still be present, but with the cell protoplasm and nuclei so changed that they do not stain normally. Bacteria are found in the epithelial layer and in the upper portion of the crypts of Lieberkühn. In more severe and prolonged cases the superficial epithelium in places is entirely destroyed, and through such breaks the bacteria can be seen penetrating into the deeper structures of the intestine; these changes mark the beginning of ileo-colitis. In simple intestinal intoxication the bacteria are not, as a rule, found in the deeper structures of the intestines nor in the lymph nodes of the mesentery. Unless autopsies are made immediately after death, little significance can be attached to the presence of bacteria, particularly the colon bacillus in the deeper layers of the intestine, in the other organs, or in the blood.

The changes in and about the blood-vessels are variable. The small vessels may be distended, and there may be hæmorrhages or an exudation of leucocytes in their neighbourhood. These conditions are seen either in the mucous or submucous layer. The exudation from the blood-vessels is usually slight, and in many cases is wanting. Peyer's patches and the lymph nodules may be enlarged from cell-proliferation. Pathologically no sharp line can be drawn between these lesions and those of the early stage of ileo-colitis; the latter affect the lower ileum and colon chiefly, often exclusively, are more advanced, and involve the deeper parts of the intestinal wall.

Lesions in other organs.—These are much less frequent and less severe than in the more protracted cases of ileo-colitis. Acute bronchitis and broncho-pneumonia are frequent. Acute degeneration of the kidney is found to some degree in every case which is severe enough to cause death, and in a few there is acute diffuse nephritis. In rare cases a general septicæmia, due most frequently to the streptococcus, is present with its usual manifestations. Degenerative changes are sometimes found in the liver cells, and even in the nervous centres. Some of these lesions are accidental, while others are the direct result of the circulation in the blood of toxins derived from the intestines.

Clinically, there are two quite distinct forms of gastro-enteric intoxication, which will be separately considered—(1) the simple form and (2) true cholera infantum.

SIMPLE GASTRO-ENTERIC INTOXICATION.—There are seen in infants mild attacks, which do not differ clinically from cases of intestinal indigestion.

Under favourable conditions and with proper treatment most such cases recover after active symptoms lasting from one to three weeks, although it may be one or two months before a steady gain in weight begins (Fig. 63). Severe symptoms may, however, supervene at any time, and the attack become one of a very grave type. This often takes place with great suddenness, and is frequently coincident with a few days of very hot weather, or follows some gross dietetic error. In other cases the symptoms may continue with the gradual formation of follicular ulcers, the case becoming one of ileo-colitis. The entire illness may continue, with exacerbations and remissions, until the cool weather of autumn.

In the cases developing suddenly, the clinical picture is quite a different one. The attack may begin abruptly in a child previously healthy, or there may have been for some days a slight intestinal derangement. If an infant, it is restless, cries much, sleeps but a few minutes at a time, and seems in distress. The skin is hot and dry, the temperature rises rapidly to 102° or 103° F., sometimes to 106°, and all the symptoms indicate the onset of some serious illness. The infant may lie in a dull stupor, with eyes sunken, weak pulse, and general relaxation, or there may be restlessness, excitement, and even convulsions. There may be great thirst, so that everything offered is eagerly taken, or everything may be refused. Vomiting may be an early and important symptom. It is first of food, often that which was taken many hours before; retching continues even after the stomach has been emptied, so that mucus, serum, and sometimes bile may be ejected. It does not usually persist throughout the attack, and in many cases it is absent altogether. Diarrhœa is sometimes delayed for twenty-four hours or even longer after the beginning of the grave constitutional symptoms. At first there are fæcal stools, then great bursts of flatus, with the expulsion of a thin yellow material with an offensive odour. Four or five such discharges may occur in as many hours. At other times the stools are gray, green, or greenish-yellow, and sometimes brown. They often do not differ at first from those of an ordinary attack of acute intestinal indigestion. The

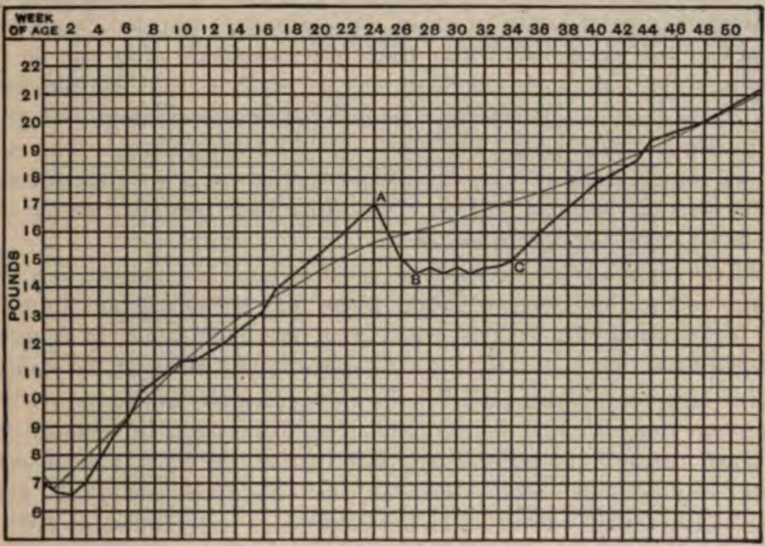

FIG. 63.—Weight curve of artificially fed infant for the first year, showing the effect of acute gastro-enteric intoxication. Normal progress until *A*, acute attack with fever; *B*, acute symptoms relieved, but continued intestinal indigestion; *C*, digestion practically normal, and child put back upon its modified-milk food.

characteristic features are the amount of the gas expelled, the colicky pains preceding the discharges, and the foul odour. After the first day the stools may be almost entirely fluid, varying in number from six to twenty a day, and often large even then. Their offensive character usually continues. After two or three days mucus may appear. The microscopical examination of the stools shows, besides the things mentioned in the stools of acute indigestion, great numbers of separate epithelial cells, and sometimes groups of cells attached to a basement membrane. In addition there may be round cells and some red blood-corpuscles.

In many cases the free evacuation of the bowels is followed by a drop in the temperature and subsidence of the nervous symptoms, and the child may fall asleep, to awaken after a few hours for a stool. The prostration, though often great in the beginning, may not be of long duration. Under the most favourable circumstances, after one or two days of severe symptoms, the case may go on to a rapid convalescence. The stools continue abnormally frequent for five or six days, but gradually assume their normal character, and recovery follows. The chief factors contributing to such favourable results are a good constitution on the part of the child, energetic and intelligent treatment at the outset, and proper feeding afterward.

If the circumstances are not so favourable, if the patient is a very young, delicate, or cachectic infant, there may be no reaction from the first severe symptoms, and the attack may terminate fatally in from one to three days. In such cases the temperature remains high; the stomach may or may not be disturbed; but the diarrhœa, prostration, and nervous symptoms continue, and death occurs from exhaustion, in coma or convulsions. Instead of a rapidly fatal termination, the severity of the early acute symptoms may abate somewhat, and the attack assume the character of ileo-colitis, with a lower but continuous temperature of 100° to 102° F., frequent mucous stools, wasting, etc. The urine is scanty and concentrated, and in most of the severe cases with very high temperature contains a small amount of albumin, and occasionally a few hyaline and granular casts. These are the result of degenerative changes in the renal epithelium from the irritating toxins. In rare cases there are evidences of acute nephritis. (See Cholera Infantum.) Broncho-pneumonia is also sometimes seen.

Relapses.—Re-infection.—It not infrequently happens, after the storm of the acute attack with its high temperature, intense prostration, and grave nervous symptoms is passed, and the stools are so much improved that the patient is regarded as out of danger, that all the former symptoms may develop with such rapidity and severity as sometimes to carry off the patient in from twelve to twenty-four hours. Such relapses are usually the result of re-infection of the intestinal tract, generally excited by some mistake in the diet, usually that of allowing milk too soon. The amount of milk given may be small, and yet the symptoms follow its administration so soon that there can be no doubt regarding the connection between them. This only indicates that virulent bacteria may remain in the intestine for a considerable time after the disappearance of severe symptoms, waiting only for favourable conditions to develop again with all their former intensity (Fig. 64). Besides such severe cases, many of a milder grade of re-infection are seen, and the cause is usually some error in diet; occasionally, however, it is due to checking the discharges by the too free use of opium.

Cases without diarrhœa.—Attacks of acute intestinal intoxication in which there is no diarrhœa, but constipation instead, are most puzzling and frequently most serious. Fortunately, they are not of common occurrence. I have, however, seen several striking examples with very high temperature, grave nervous symptoms, and sometimes marked abdominal distention in which it seemed almost impossible to move the bowels by drugs. Castor oil, calomel, and salines have in some cases been tried in succession in four or five times the ordinary doses without the slightest effect, even when supplemented by frequent intestinal irrigation. It has sometimes been nearly two days before free movements were finally produced. These are often exceedingly foul. It is somewhat difficult to explain such cases. There seems to exist for the time almost complete intestinal paralysis. The toxic materials are locked up in the small intestine, for the colon is frequently quite empty. When

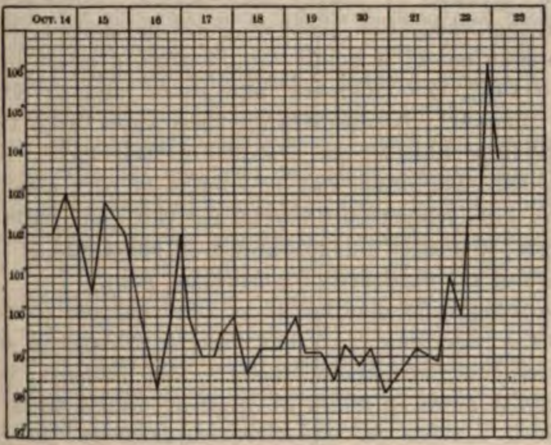

Fig. 64.—Acute intestinal intoxication with fatal re-infection.

Infant five months old; early symptoms, both intestinal and nervous, severe; rapid improvement followed stopping milk, free catharsis and irrigation. After stools had been nearly normal for three days relapse occurred, apparently from adding milk to the diet, although less than two ounces a day were given. Autopsy: Intestines showed the usual changes of intoxication; other organs essentially normal.

one meets such a case he can appreciate the fact that in acute intestinal intoxication diarrhœa is a conservative process of the greatest possible value.

In children over two years old there are seen some features which differ from those of the cases above described as occurring in infants. The attacks are more often due to other causes than to milk. Vomiting does not occur so readily as in infants, pain is a more prominent symptom, and the temperature, as a rule, is lower. The nervous symptoms are much less prominent. Skin eruptions, however, are more frequently seen, particularly urticaria, which is a feature of most severe attacks, and in obscure cases has some diagnostic value. Although often beginning with severe symptoms, these cases usually make good recoveries; there is much less danger of their going on to the development of ileocolitis than in the case of infants.

Diagnosis.—Attacks of acute gastro-enteric intoxication can not always be distinguished from those of acute indigestion, but as a rule they are characterized by a higher temperature, greater disturbance of the nervous system, very offensive fluid stools, and by occurring epidemically in summer. To differentiate these cases from those of ileocolitis, may be impossible for the first two or three days. Nor is it important to do so. The onset may be similar in both conditions. The continuance of high temperature beyond the third day points to inflammatory changes; so also do the appearance of blood and of much mucus in the stools, and the existence of continuous pain.

The acute indigestion manifested by vomiting and diarrhœal stools which marks the beginning of so many febrile diseases in infancy, particularly scarlet fever, pneumonia, malaria, and influenza, is often difficult to distinguish from an attack of intestinal intoxication. The question to decide is whether the digestive symptoms are the cause or the result of the fever. It is sometimes not until the case has been watched for at least forty-eight hours that one can be certain as to the diagnosis. Usually where digestive symptoms are secondary they diminish after the first day or two, although the severity of the general symptoms may steadily increase. Where the nervous symptoms are prominent at the outset, it is sometimes difficult to distinguish acute intestinal intoxication from meningitis. I have seen many cases where great doubt existed for several days. One should always hesitate to make a diagnosis of meningitis when marked diarrhœa is present.

Prognosis.—Simple cases of gastro-enteric intoxication do not often prove fatal, except in young infants or those already suffering from malnutrition. Such patients are often overcome in the first stage of intoxication. Even an apparently mild attack may prove fatal.

In other cases the prognosis resolves itself into this question: What are the probabilities of arresting the attack before the production of serious intestinal lesions? If the child is delicate, living in poor surroundings, has previously suffered from digestive derangements or acute diarrhœa, and does not receive proper early treatment, the attack will probably result in structural changes in the intestines. In hot weather this is especially liable to be the case. The existence of rickets, pertussis, or any other disease, greatly increases the gravity of the attack.

Prophylaxis.—A better understanding of the etiology brings with it great possibilities in the prevention of this disease.

Prophylaxis must have regard, first, to the hygienic surroundings of children, and to all sanitary conditions in the cities. City children should be sent to the country, whenever it is possible, for the months of July and August. Where a long stay is impossible, day excursions do much good. The fresh-air funds and seaside homes have done more in New York to diminish the mortality from diarrhœal diseases in summer than all medicinal treatment.

The second part of prophylaxis relates to food and feeding. Maternal nursing should be encouraged by every possible means. Nothing is better established than the close relation existing between artificial feeding and diarrhœal diseases. Yet, as stated elsewhere, it is not artificial feeding *per se,* but ignorant and improper feeding. Among infants in private practice who are properly fed these attacks are not common. The general rules laid down elsewhere on the subject of artificial feeding should be carried out, as to the quantity of food, frequency of feeding, modification of cow's milk, and all matters relating to the care, transportation, and handling of milk. The important dangers to be emphasized in this connection are overfeeding, too frequent feeding, the use of improper foods or impure foods, especially impure milk.

Overfeeding is particularly to be avoided during days of excessive heat. It is at such times an excellent rule with infants to diminish each meal by at least one-half, making up the deficiency with water, and to give water very freely between the feedings. All water given to infants or young children should first be boiled. Children, like adults, require less food in very hot weather, but more water. Infants cry more from thirst and heat than from hunger, and even those at the breast are likely to be given too much food. Infants should never be fed more frequently, but always less frequently during hot weather.

No more important work in practical philanthropy can be done among the poor of our large cities in summer than to provide means for supplying pure milk to infants. This has been done on a large scale in many American cities, and it has effected a very decided reduction in the death-rate from diarrhœal diseases. (See page 43.) In some places this has been accomplished through private generosity, in others by the Department of Health. It is not enough to furnish to the poor a pure, clean milk in bulk, or even in sealed quart bottles. The advantages of such milk may be entirely lost by the way in which it is cared for in the home or by the method of feeding. The most successful plan is that in which milk is modified and sterilized at central stations, from which it is distributed in small feeding-bottles, each containing enough only for a single feeding. A twenty-four hours' supply is furnished at each daily visit. Sometimes the milk is given free, sometimes a nominal charge, generally one cent a bottle, is made. Since the milk must usually be kept at home without ice, sterilization at 212° F. is advisable. A physician is in charge of the milk distribution who gives advice when needed, keeping a general supervision over the children and deciding the quantity of food, number of feedings, and the formula to be used. It is not necessary to have a large number of formulas. In summer three or four simple ones will be found to answer all requirements. Those derived from dilutions of whole milk (see page 194) in which the fats are low will generally be found to be best for hot weather; e. g., fat, 1; sugar, 6; proteids, 0.90 (one-fourth milk); or, fat, 2; sugar, 6; proteids, 1.80 (one-half milk); or, fat, 3; sugar, 7; proteids, 2.70 (three-fourths milk). The dilution is made with plain water or with barley-water and milk-sugar added to bring up the percentage of sugar to the desired amount. Further dilution of these formulas to secure lower percentages may be made at home by simply adding boiled water before feeding. In observations made upon infant-feeding in the tenements of New York already referred to (see page 357) the plan of feeding described above gave by far the best results, and it is the one to be recommended.

Second only in importance to proper food is the education of the poor in all matters relating to infant hygiene. Early and prompt attention should be given to all the milder derangements of the stomach and intestines. The larger proportion of serious attacks are preceded for some time by mild symptoms, which are often easily managed by prompt attention at the outset.

In brief, prophylaxis demands (1) sending as many infants out of the city in summer as possible; (2) the education of the laity as to the importance of proper rules of feeding, the dangers of overfeeding, and as to what constitutes a suitable diet for infants just weaned; (3) proper legal regulations regarding the transportation and sale of milk; (4) sterilization of milk used by the poor during the summer; (5) scrupulous cleanliness in bottles and nipples; (6) prompt attention to all mild derangements; (7) reducing the amount of food and increasing the amount of water during the days of excessive summer heat.

Hygienic Treatment.—If the attack occurs in the city in midsummer, and does not yield in three or four days to the treatment employed, the child should, if possible, be sent to the country. Convalescent cases should also be sent away on account of the dangers of relapses. Usually the seashore is to be preferred to the mountains, but this is not so important as that the child shall go where it is likely to have the best food and the best surroundings. Children must not only be sent away; they must be kept away until quite recovered. In cases which have become somewhat chronic, more can sometimes be accomplished by a change of air than by all other means.

Fresh air is of the utmost importance for all diarrhœal cases in summer. No matter how much fever or prostration there may be, these cases always do better if kept out of doors the greater part of the day. Nothing is so depressing as close, stifling apartments. Children should be kept quiet, and especially should not be allowed to walk, even if they are old enough and strong enough to do so. They can be kept out in carriages, in perambulators, or in hammocks.

The clothing should be very light flannel; a single loose garment is preferable. Linen or cotton may be put next the skin if this is very sensitive and there is much perspiration. At the seashore and in the mountains, care should be taken that sufficient clothing at night is supplied.

Bathing is useful to allay restlessness, as well as for cleanliness and the reduction of temperature. For the reduction of temperature, only the tub bath is to be relied on. The temperature of the bath should be about 100° F. when the child is put into it, and should then be gradually reduced to 80° or 85° F. by adding ice. The bath should be continued, with gentle friction of the body, for from five to twenty minutes.

Scrupulous cleanliness should be secured in the child's person and clothing. Napkins, as soon as soiled, should be removed from the child and from the room and placed in a disinfectant solution. Excoriations of the buttocks and genitals are to be prevented by absolute cleanliness and the free use of some absorbent powder, such as starch and boric acid.

Dietetic Treatment.—It is of the first importance to remember that during the early stage of the acute cases, digestion is practically arrested. To give food at this time, manifestly can do only harm.

In nursing infants the severe forms of the disease are extremely rare; but the breast should be withheld so long as a disposition to vomit continues, and no food whatever given for at least twenty-four hours. Thirst may be allayed by giving frequently, but in small quantities, cold whey, thin barley water, or albumin water. Stimulants may be added if required. If they are refused or vomited, absolute rest to the stomach will do more than anything else to hasten recovery. After the stomach has been allowed to rest for twenty-four hours, it is generally safe to permit a nursing child to take the breast tentatively. The intervals of nursing should not be shorter than four hours, and the amount allowed at one feeding should not be more than one-fourth the usual quantity. This may be regulated by allowing an infant to nurse at first only two or three minutes. Between the nursings may be given whey, barley water, or albumin water, so that something is given every two hours. Nursing may be gradually increased, so that in three or four days the breast may be taken exclusively. If there is any reason to suspect the quality of the breast-milk, such as menstruation or pregnancy, it may be necessary to stop the nursing for a longer time.

In infants under four months who are being artificially fed, all food, and especially milk, should be stopped at once. Milk should be withheld during the period of acute symptoms, and for several days thereafter. Besides the articles mentioned above as suitable for the period of most acute symptoms, the following substitutes for milk will be found useful: rice or barley water, either plain or dextrinized; the farinaceous foods; the malted foods; broth or bouillon made of veal, chicken, or beef, and such beef preparations as Mosquera's fluid beef jelly, panopepton, liquid peptonoids, or bovinine. Water may be allowed freely at all times unless there is much vomiting.

Sterilized cow's milk should be used at first in very small quantities, and the effect upon the stools and temperature watched. The indications for modifying milk are the same as in acute intestinal indigestion. Buttermilk with barley water (page 160) sometimes agrees better than any other milk derivative. Wet-nurses are not to be employed during the acute symptoms, but during the period of prolonged malnutrition which follows an acute attack, they may be of the greatest service.

The same general principles of feeding must be applied in older children. All food is to be withheld until the vomiting ceases, when broths and beef juice may be given; later, kumyss or matzoon, afterward sterilized milk, or thin gruels made with milk. Solid food should not be allowed for several days after the stools have become normal.

Summary.—All food, but especially cow's milk, should be stopped at once. No food whatever should be given upon a very irritable stomach; but thirst should always be relieved by bland fluids given frequently in small quantities, and cold. Articles requiring the least digestion and leaving the smallest residue should next be tried. Food prescriptions must be made with the same care and exactness as those for drugs, for in most cases they are more important. Quantity and frequency must be definitely stated, as well as the articles ordered. Directions should be given in writing, or they will be forgotten before the physician is out of the house. A practical acquaintance with the proper appearance and taste of every food ordered, is absolutely indispensable. It is a common mistake to give too much at a time, to feed too frequently, to try too many articles at once, and to change before a thing has been fairly tested. For a single feeding the quantity allowed will vary according to the tolerance of the stomach, but it should generally be much less than is given in health, usually from one-fourth to one-half that amount. It is very rarely, if ever, necessary to nurse or feed a sick child oftener than every two hours, and four-hour intervals are in many cases to be preferred. In all cases water should be allowed frequently and freely; and if there is great prostration, stimulants should be given in addition.

It is a difficult problem to feed these children under three years of age, capricious as they are by nature and still more by education, and the judgment and tact of the physician are taxed to their utmost. We must have many resources, for a food which one child takes well the next utterly disdains. The best plan is to select from a list of articles of accepted value, such as circumstances will permit, and such as are most likely to be properly prepared, and try them patiently, one after another, until one is found which the child under treatment will take, and which agrees best with him.

Medicinal and Mechanical Treatment.—It must be borne in mind that we are not treating an inflammation of the stomach or intestines, although such may be the ultimate result of the process. The essential condition, it should be remembered, is one of acute intoxication arising from the intestinal contents—food-remains from arrested digestion, altered secretions, acids, and other toxic substances produced by bacteria —to which not only the constitutional symptoms, but the local lesions are chiefly due. We can hardly do better than to imitate and assist Nature in her treatment of this condition. Let us consider what this is. Lest too much food be swallowed, appetite is taken away; by vomiting, the stomach is emptied; to neutralize the acid poisons in the intestine, an alkaline serum is poured out from the intestinal walls; to remove irritant poisons, increased peristalsis is excited.

The first indication is, therefore, to evacuate the stomach and the entire intestinal tract at the earliest moment, and to do this as thoroughly as possible. Under no circumstances should the treatment be begun with the use of measures to stop the discharges. To empty the stomach is not necessary in every case, since the initial vomiting may have done this effectively. Whenever vomiting persists one should immediately resort to stomach-washing. A single washing is generally sufficient, and if employed at the outset may do much to shorten the attack. With high fever and great thirst, it is often advisable to leave an ounce or two of water in the stomach. If the vomited matters have been very sour, ten grains of bicarbonate of soda may be introduced with the portion which is to be left behind. As a substitute for stomach-washing in children over two years old, or where it can not be employed, copious draughts of boiled water may be given. This is taken readily, and as it is usually vomited almost at once it may cleanse the stomach thoroughly; but it is inferior to stomach-washing.

To clear out the small intestine, only cathartics are available. For the colon, we may in addition employ irrigation. Calomel, castor oil, or the salines may be used as cathartics, and enough of any one of them must be given not simply to move the bowels, but to clear out the intestinal tract thoroughly. There is little danger from too free purgation at the outset. Calomel has the advantage of ease of administration: one-fourth of a grain should be given every hour up to six or eight doses, or until the characteristic green stools are seen. When the stomach is not disturbed, I prefer castor oil in most cases, as it sweeps the whole canal, causes little griping, is very certain, and its after-effects are soothing. Two drachms should be given to a child a year old, and half an ounce to one of four years. Of the salines, Rochelle salts and magnesia are the best; either the sulphate, citrate, or the milk of magnesia may be used. Of the sulphate as much as one drachm should be given in divided doses in the course of two or three hours, and an equivalent amount of the other preparations.

The occasional use of cathartics is an important part of the later treatment. Whenever there are signs of an accumulation, or fresh symptoms of intoxication develop, such as increase in temperature, nervous symptoms, etc., another thorough cleaning out of the intestinal tract is indicated. The accumulation may not be the result of food, but simply of intestinal secretions. So long as the processes of fermentation and decomposition continue active, the indications are to facilitate elimination, not to check the discharges.

Irrigation of the colon is advisable in all cases, as it hastens the effect of the cathartic and removes at once much irritating and offensive material. It should be done two or three times the first day, but afterward once daily is sufficient. A saline solution (one tablespoonful of salt to two quarts of water), at a temperature of about 100° F., is to be preferred; and a long rectal tube should always be used. Thorough initial evacuation, almost no food, but plenty of water for twenty-four hours, and careful feeding after that time, are all the treatment that is necessary in a large number of cases.

Other drugs are of secondary importance. Their value is certainly very much overestimated. This statement is made after a thorough and honest trial, in hospital and private practice, of most of those that have been recommended. Since the recognition of the fact that putrefactive processes play so important a rôle in these cases, the drift of opinion and practice has been toward the use of drugs believed to act in the alimentary tract as antiseptics. In comparison with the gastric and intestinal contents the amount of any drug which can be given is small, it is true, and we have still much to learn regarding the nature of the putrefactive processes we are seeking to control. It may therefore be questioned whether as yet any scientific antiseptic treatment of the gastro-enteric tract is possible. However, clinical experience points to the fact that the internal use of antiseptics is of value, even though such remedies do no more than inhibit bacterial growth. Those which are soluble can be expected to influence only the stomach and upper small intestine. The insoluble ones may affect the lower small intestine and colon. Those which in my experience have been found most useful are bismuth, salol, salicylate of soda, and resorcin; although the list might be very greatly extended.

Bismuth has the advantage that it rarely causes vomiting, and that most of its preparations can be given in large doses. Of the newer preparations, the subgallate is easily superior to the others. This may be given in doses of from two to four grains every two hours, to a child of one year. Like the subnitrate it is insoluble and is best given suspended in mucilage. For most cases, however, I think the subnitrate is still to be preferred. To be efficient, from one to two drachms should be given daily to a child two years old. It usually blackens the stools. It may be kept up throughout the attack. Of the salicylate of soda, to a child of one year, two grains may be given, dissolved in water, every two hours, after feeding. This is not to be used if the stomach is very irritable, as it may excite vomiting. Its best effect is seen after the vomiting has stopped, and when the stools are fluid. It should be given alone. Salol is decomposed in the intestine into salicylic and carbolic acids. To a child of two years one or two grains may be given every two hours; sometimes more will be borne. Resorcin may be used in doses half as large. Either of these, however, may cause vomiting. The best results are seen from acids in the later stages and in the subacute cases; of the dilute hydrochloric acid, from one to three drops may be given, best alone. Alkalies are of value only in the acute stage, especially where there is acid fermentation in the stomach, with vomiting and eructations of gas. Lime-water, bicarbonate of soda, magnesia, or chalk-mixture may be employed. My own experience accords with that of most recent writers in according a very limited place to astringents. They do little good, and often much harm. They are indicated only in the catarrhal diarrhœa which often follows the symptoms of acute intoxication, but may be advantageously used in this condition in combination with opium. A useful astringent is tannalbin, which may be given in two-grain doses every two hours to an infant of one year.

While opium in some form is required in many cases, as often used it undoubtedly does great harm. The chief indications for opium are great frequency of movements and severe pain. It is contraindicated until the intestinal tract has been thoroughly emptied by cathartics and irrigation; also when the number of discharges is small, particularly if they are very offensive; it is especially to be avoided in the early stage of very acute cases, and never to be given when cerebral symptoms and high temperature coexist with scanty discharges. Opium is admissible in the early part of the disease after the tract has been thoroughly emptied. It is particularly indicated when there is a persistence of large,

fluid movements attended by symptoms of collapse, and in all cases approaching the cholera-infantum type. In such circumstances morphine should be given hypodermically, one one-hundredth of a grain to an infant of six months, to be repeated in an hour if no effect is seen. Opium is useful during convalescence, when the administration of food is immediately followed by a movement of the bowels; and when without an elevation of temperature, often with good appetite, the stools are frequent and contain undigested food, because peristalsis is so active that the intestinal contents are hurried along with such rapidity that there is not time for complete intestinal digestion and absorption. Nothing requires nicer discrimination than the use of opium in diarrhœa. It is wise to administer it always in a separate prescription, and never in composite diarrhœal mixtures. The dose should be regulated according to its effect upon the number of stools. Enough is to be given to produce a distinct effect—the diminution of pain and the control of excessive peristalsis—but never enough to check the discharges entirely, or to cause stupor. The uncertainty of absorption must also be remembered; a second full dose should not be given until a sufficient time has elapsed for the effect of the first to pass away. For an average child of one year, five minims of paregoric, one-fourth minim of the deodorized tincture, or one-fourth grain of Dover's powder, may be used as an initial dose, to be repeated every one, two, or four hours, according to the effect produced.

Stimulants are required in the majority of the severe cases. The prostration is great and develops rapidly; frequently almost no food can be assimilated for twenty-four or thirty-six hours, while the drain from the discharges continues. The general condition of the patient is the best guide as to the time for stimulation and the amount required. Often stimulants are not begun early enough. Old brandy is the best preparation for general use, champagne being possibly preferred for older children when the stomach is very irritable. An infant a year old will, under most circumstances, take half an ounce of brandy in twenty-four hours. Stimulants should always be diluted with at least eight parts of water, and be given in small quantities, at short intervals.

In cases of extreme prostration, the hot bath, mustard to the extremities, and sometimes the mustard pack, are beneficial. When the drain is rapid and very great, and in all cases approaching the cholera-infantum type, subcutaneous saline injections should be used, in the manner described under Cholera Infantum.

General considerations in treatment.—(1) All severe cases must be watched very closely, especially those in infants under six months. If the temperature is rising and the passages are very fluid, one should always be apprehensive. (2) The character of the discharges is a better indication than is their number, of the patient's condition and of the

effect of any plan of treatment. (3) Nothing is more simple than to give opium enough to reduce the number of passages; but unless there is some other sign of improvement, very little good, and probably much harm, will be done. (4) We must treat the patient, and not direct all our thought to acid or alkaline stools, ptomaines, or bacteria. The value of every therapeutic measure is to be estimated by its effect upon the patient's general condition. (5) No matter how strongly we may believe in the value of any drug or combination of drugs, if they continue to disturb the stomach they are worse than useless. (6) Both the mother and nurse should be impressed with the fact that the diet is an important part of the treatment, and that foods need to be given just as carefully as drugs. (7) In the management of any single case the important thing is prompt and thorough evacuation of the stomach and bowels, then rest for these organs for from twelve to twenty-four hours, or, as some one has tersely put it, "bold starvation"; but it is necessary in all cases that water be given freely. No cases do worse than those in which the mother or nurse in charge can not be made to appreciate the value of starvation, but insists upon giving food, especially milk, in violation of the rules laid down. (8) Great care is required during convalescence, and in fact during the remainder of the summer, to prevent relapses; these usually occur from errors in diet, particularly during days of excessive heat.

Cholera Infantum.—This may be regarded as only one clinical type of acute intestinal intoxication, yet it differs from the others sufficiently to deserve separate consideration. It is not, however, the most frequent form met with, and it is not a good generic name for the disease. As yet this type has not been connected with a specific form of intoxication. The peculiar symptoms may depend upon the rapidity of absorption and the other conditions present in the intestine, or possibly upon some form of infection not yet determined. Cholera infantum is more closely connected with impure milk than is any of the other forms of diarrhœa, and may be due to some poison developing in the milk before its ingestion, or in the stomach or intestines after the milk is taken. The symptoms are due primarily to the effects of the poison upon the heart, the nerve-centres, and the vaso-motor nerves of the intestines; secondarily to the abstraction of fluid from the various organs and tissues of the body, especially the nerve-centres.

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Usually there are general symptoms, such as prostration and a steadily rising temperature, for a few hours before the vomiting and purging, or

fluid movements attended by symptoms of collapse, and in all cases approaching the cholera-infantum type. In such circumstances morphine should be given hypodermically, one one-hundredth of a grain to an infant of six months, to be repeated in an hour if no effect is seen. Opium is useful during convalescence, when the administration of food is immediately followed by a movement of the bowels; and when without an elevation of temperature, often with good appetite, the stools are frequent and contain undigested food, because peristalsis is so active that the intestinal contents are hurried along with such rapidity that there is not time for complete intestinal digestion and absorption. Nothing requires nicer discrimination than the use of opium in diarrhœa. It is wise to administer it always in a separate prescription, and never in composite diarrhœal mixtures. The dose should be regulated according to its effect upon the number of stools. Enough is to be given to produce a distinct effect—the diminution of pain and the control of excessive peristalsis—but never enough to check the discharges entirely, or to cause stupor. The uncertainty of absorption must also be remembered; a second full dose should not be given until a sufficient time has elapsed for the effect of the first to pass away. For an average child of one year, five minims of paregoric, one-fourth minim of the deodorized tincture, or one-fourth grain of Dover's powder, may be used as an initial dose, to be repeated every one, two, or four hours, according to the effect produced.

Stimulants are required in the majority of the severe cases. The prostration is great and develops rapidly; frequently almost no food can be assimilated for twenty-four or thirty-six hours, while the drain from the discharges continues. The general condition of the patient is the best guide as to the time for stimulation and the amount required. Often stimulants are not begun early enough. Old brandy is the best preparation for general use, champagne being possibly preferred for older children when the stomach is very irritable. An infant a year old will, under most circumstances, take half an ounce of brandy in twenty-four hours. Stimulants should always be diluted with at least eight parts of water, and be given in small quantities, at short intervals.

In cases of extreme prostration, the hot bath, mustard to the extremities, and sometimes the mustard pack, are beneficial. When the drain is rapid and very great, and in all cases approaching the cholera-infantum type, subcutaneous saline injections should be used, in the manner described under Cholera Infantum.

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these symptoms may be the first to excite alarm. Vomiting may precede diarrhœa, or both may begin simultaneously. The vomiting is very frequent. First, whatever food is in the stomach is vomited, then serum and mucus, and finally bilious matter. If vomiting subsides for a time, it is almost sure to begin anew with the taking of food or drink. The stools are frequent, large, and fluid, and in the course of half a day twelve or fifteen may occur. If less frequent they are proportionately larger. They are of a pale green, yellow, or brownish colour in the beginning, but as they become more frequent they often lose all colour and are almost entirely serous. The sphincter is sometimes so relaxed that small evacuations occur every few minutes. The first stools are usually acid, later they are neutral, and when serous they may be alkaline. In most cases they are odourless; in rare instances they are exceedingly offensive. Microscopically the stools show large numbers of epithelial cells, some round cells, and immense numbers of bacteria.

Loss of weight is more rapid than in any other pathological condition in childhood. Baginsky records a case in which it reached three pounds in two days. The fontanel is depressed, and in rare instances there may be overlapping of the cranial bones. The general prostration is great almost from the outset. The face, better, perhaps, than any single symptom, indicates what a profound impression has been made upon the system. The eyes are sunken, the features sharpened, the angles of the mouth drawn down, and a peculiar pallor with an expression of anxiety overspreads the whole countenance. In the early stages the nervous symptoms are those of irritation. Later, these symptoms give place to dulness, stupor, relaxation, and coma or convulsions.

The temperature, in my experience, has been invariably elevated, and usually in proportion to the severity of the attack. In cases recovering, it has generally been from 102° to 103° F., while in fatal cases it has risen almost at once to 104° or 105° F., and often shortly before death it has reached 106° or even 108° F. Such rectal temperatures may occur with a clammy skin and cold extremities, and are discovered only by the thermometer. The pulse is always rapid, and very soon it becomes weak, often irregular, and finally almost imperceptible. The respiration is irregular and frequent, and may be stertorous. The tongue is generally coated, but soon becomes dry and red, and is often protruded. The abdomen is generally soft and sunken. There is almost insatiable thirst. Everything in the shape of fluids, especially ice-water, is drunk with avidity, even though vomited as soon as it is swallowed. Very little urine is passed, sometimes none at all for twenty-four hours; this depends upon the great loss of fluid by the bowels.

In the fatal cases there is hyperpyrexia, a cold, clammy skin, absence of radial pulse, stupor, coma or convulsions, and death. The diarrhœa and vomiting may continue until the end, or both may entirely cease for some hours before it occurs. The patients may pass into a condition resembling the algid stage of epidemic cholera, and die in collapse. In other cases, after the first day of very severe symptoms, the discharges diminish, but the nervous symptoms become specially prominent. There is restlessness and irritability or apathy and stupor. The fontanel is sunken; the eyes are half open and covered with a mucous film; respiration is irregular and superficial, sometimes even Cheyne-Stokes; the pulse is feeble, irregular, or intermittent; the muscles of the neck drawn back; the abdomen retracted. The temperature is not elevated, but normal or subnormal. From this condition recovery may take place or the symptoms may merge into those of ileo-colitis; but much more frequent than either of the foregoing is the fatal termination.

These nervous symptoms are ascribed to cerebral anæmia, cerebral hyperæmia (venous), œdema of the meninges, thrombosis of the cerebral sinuses, and uræmia.

Although I have examined the brain in almost all my autopsies upon patients dying from diarrhœal diseases, I have never in such cases seen sinus thrombosis, and but rarely œdema. Cerebral hyperæmia was often met with in cases dying in convulsions, but not with any regularity otherwise. Nor have my observations upon the kidneys confirmed the observations of Kjellberg, whom most of the writers since his day have quoted, as to the great frequency of nephritis. A scanty, concentrated, and hence irritating urine is the rule, and a small amount of albumin and an occasional hyaline cast not uncommon; but either clinical or pathological evidence of a serious amount of nephritis has been, in my own experience, extremely rare.

We can hardly regard either the renal or the cerebral changes as an explanation of the nervous symptoms of most of these cases; they seem rather to depend upon impeded circulation due to a thickening of the blood, to acute inanition, and intestinal toxæmia.

Of the cases of true cholera infantum which have come under my notice, fully two-thirds have died. The result depends more upon the severity of the attack than upon anything else.

An infrequent complication of cholera infantum is sclerema. This condition is found associated with muscular contractions, subnormal temperature, and other signs of the most extreme depression. These cases are invariably fatal.

Treatment.—Restricting the term to the class of cases described above, all who have seen much of the disease must admit that the results of treatment are extremely unsatisfactory, and that the most severe cases pursue their course but little, if at all, influenced by the treatment employed.

The best view of the treatment will be gained if we keep in mind that we are treating cases of poisoning; that the toxic materials cause great depression of the heart and the system generally by acting on the nerve-centres, and by paralyzing the vaso-motor nerves of the intestines.

The main indications are: (1) to empty the stomach and intestine; (2) to neutralize the effect of the poison upon the heart and nervous system; (3) to supply fluid to the blood to make up for the very great drain of the discharges; (4) to reduce the temperature; (5) to treat special symptoms as they arise.

For the first indication we must rely upon mechanical means—stomach-washing and intestinal irrigation—there is no time to wait for cathartics. For the second, nothing in my hands has proved so useful as the hypodermic use of morphine and atropine. I believe this to be more efficient than any other means of treatment we possess. Morphine is contra-indicated where the purging has ceased or is slight, and where there is drowsiness, stupor, or relaxation. The effects of the dose should always be carefully watched; a small dose repeated is better than a single large dose. For a child a year old, not more than gr. $\frac{1}{80}$ of morphine and gr. $\frac{1}{800}$ of atropine should be the initial dose. It may be repeated in an hour unless the desired effects are produced; these are, arrest of the vomiting and purging (or at least their diminution), improvement in the heart's action, and in the nervous symptoms.

For the third indication the only thing that can be depended upon is the injection of normal salt solution into the cellular tissue of the abdomen, buttocks, thighs, or back. At least half a pint should be given in the course of every twelve hours. A very much larger quantity can often be used with advantage. This causes no irritation, and is absorbed with surprising rapidity. The injection is made slowly, and the exact amount introduced at each time measured.

For the reduction of temperature baths should be used. They may be continued from ten to thirty minutes, and to be efficient, must be used frequently—as often as every hour if symptoms are threatening. Iced cloths or an ice cap should be applied to the head. Cold-water injections are a valuable accessory to the treatment by baths. Nothing should be allowed by the mouth except ice and brandy. The stimulants must be given in small quantities and frequently. When stimulants taken by the mouth are vomited, they should be given hypodermically. Brandy, ether, or camphor may be used freely. During the stage of most acute symptoms, to attempt to give food or drugs of any kind by the mouth is worse than useless. After the stage of violent symptoms has subsided and reaction is established, the subsequent management in respect to feeding and medication should be the same as in the cases considered in the previous chapter. If cerebral symptoms are present, opium is to be avoided, stimulants by the mouth used freely, and, if these are not retained, they should be given hypodermically. For cold extremities and subnormal temperature, hot mustard baths should be used to establish reaction, mustard paste applied all over the body, and hot-water bags and bottles placed about the patient.

CHAPTER VIII.

DISEASES OF THE INTESTINES.—(Continued.)

ACUTE ILEO-COLITIS—DYSENTERY.

Synonytms: Entero-colitis, enteritis, enteritis follicularis, inflammatory diarrhœa.

THE term *ileo-colitis* is a general one, embracing those forms of intestinal disease in which the more serious lesions are present. In gastro-enteric intoxication recovery or death takes place before anything more than superficial changes have occurred, while in ileo-colitis the pathological process continues until there have been produced marked lesions, often involving all the walls of the intestine. Sometimes the transition is so gradual that it is impossible, by symptoms, to draw a line between them. This is especially true of the cases terminating in follicular ulceration of the colon. In some of the other forms—acute catarrhal and acute membranous colitis—the evidences of a severe intestinal inflammation are often manifest from the very outset. This difference is probably due to a difference in the character of the infection. The extent of the lesions depends much upon the duration of the process.

Etiology.—The predisposing causes of ileo-colitis are those common to diarrhœal diseases in general, and have already been considered. Although seen with especial frequency in summer, and in children under two years old, it may affect those of any age, and occurs at all seasons. Epidemics are not uncommon in the early fall months. While usually primary, it often follows infectious diseases, especially measles, diphtheria, and broncho-pneumonia. It frequently occurs, in institutions chiefly, as a terminal infection in infants suffering from extreme malnutrition or marasmus. Any other intestinal disease may precede ileo-colitis. The question of contagion is unsettled; if at all communicable, it is feebly so. When it occurs epidemically a common origin seems more probable than that the disease spreads from one patient to another.

The only bacterium that up to the present time has been shown to be capable of producing this form of intestinal disease is the *B. dysenteriæ* of Shiga. This organism, or, more properly speaking, this group of closely allied organisms, has now been found in all parts of the world in a sufficient number of cases to establish its etiological connection with ileo-colitis. The *B. dysenteriæ* was shown by Shiga, in 1898 and 1899, to be the cause of epidemic dysentery in Japan. In 1900, Flexner established its association with tropical dysentery in the Philippines, and in

1902, Duval and Bassett, pupils of Flexner, demonstrated its presence in a series of cases of diarrhœa in children at Baltimore.

In the summer of 1903 the Rockefeller Institute undertook a collective clinical and bacteriological investigation in New York, Baltimore, Boston, and Philadelphia, to discover what part the *B. dysenteriæ* played in the diarrhœal diseases of children. In all 412 cases were studied, in 270 of which the bacillus was present. It was almost invariably found in cases showing blood and mucus, or much mucus in the stools. The number of the specific bacteria present, as shown by culture, corresponds in a general way with the severity of the symptoms and the lesions of the disease. Although usually the *B. dysenteriæ* is greatly outnumbered by other organisms, it is not uncommon to find it in pure culture. A number of minor differences have been found in the bacilli from different cases; there are, however, two main groups, the division being made by reason of the difference in reaction with litmus mannite; one group is known as the "true Shiga," or "alkaline" type; the other, as the "Flexner," or "acid" type. The latter has been most frequently found in the diarrhœal diseases of children in this country, although the true Shiga is occasionally present, and in rare cases they may be associated.

The *B. dysenteriæ* has been in a few instances discovered in normal stools of apparently healthy children, although extended observation by Wollstein at the Babies' Hospital upon 56 infants failed to show its presence in any normal case. The *B. dysenteriæ* has never been found outside the body; we are therefore entirely ignorant both of its habitat and its mode of entry. There are grounds for believing that it appears at times among the saprophytic bacteria of the intestinal contents.

The rôle played by other bacteria, especially the streptococcus, in the production of the deeper lesions of the intestine may be an important one. This appears, however, to be rather in the nature of a secondary invasion.

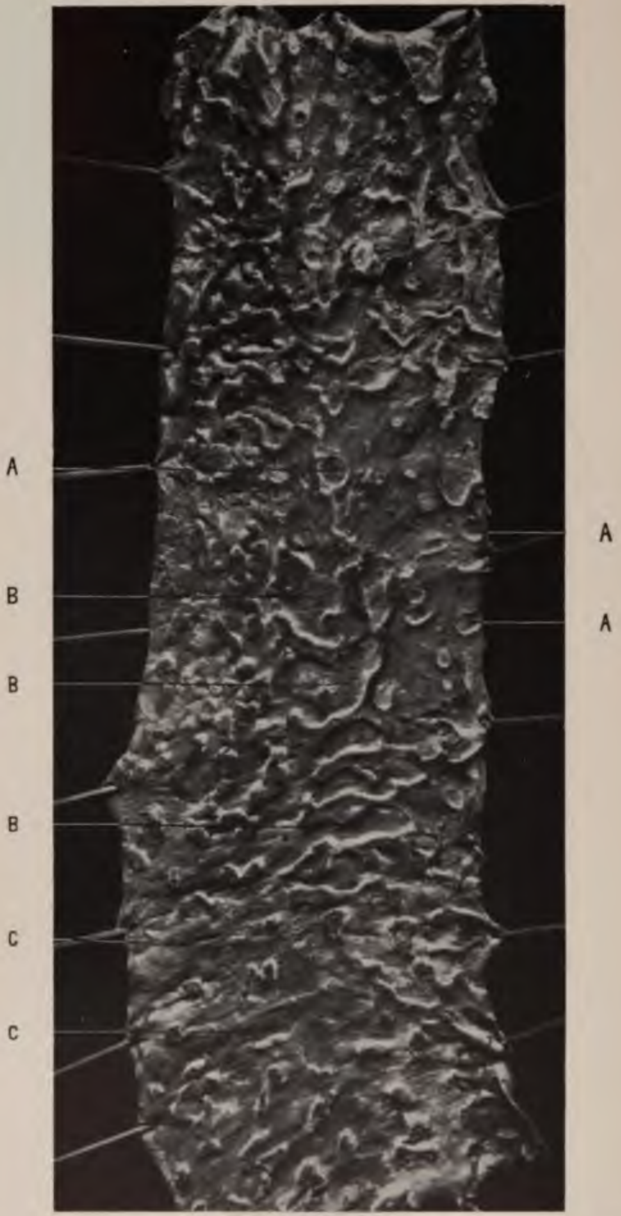
Lesions.—It is surprising that, so far as is known, a single specific cause can excite such a variety of lesions. The nature of the anatomical changes apparently depends upon other factors, such as the intensity of the infection, the local resistance, and still more upon the duration of the disease.

The nature of the lesions in ileo-colitis differs greatly, but their position is quite constant: they affect the lower ileum and the colon. In about half the cases only the colon is affected. The lesions of the ileum are usually limited to the lower two or three feet.

The frequency with which the different varieties of ileo-colitis were found in eighty-two of my own autopsies was as follows:

Follicular ulceration	36
Catarrhal inflammation	26
Catarrhal inflammation with superficial ulceration	6
Membranous inflammation	14
	82

PLATE VIII.



EXTENSIVE SUPERFICIAL ULCERATION OF THE COLON.

Female child nine months old; symptoms of acute ileo-colitis of fifteen days' duration; temperature, 101° to 104·5° F., and from six to eight stools daily—thin, green, and yellow, but no blood.

Extensive ulceration throughout the colon, most marked in descending portion, from which specimen is taken.

A A are small circular ulcers; B B, larger ones from coalescence of several of these; C C, large areas of ulceration, the mucous membrane being almost entirely destroyed.

Acute catarrhal ileo-colitis.—In the milder cases there are changes in the epithelium and infiltration of the mucosa. In the severer cases the submucosa is involved, and the infiltration of the mucosa may be so great as to lead to necrosis and the formation of ulcers.

Gross appearances.—While the lower ileum and the colon are most seriously affected, it is not uncommon to find quite marked changes in a considerable portion of the small intestine, and even in the stomach. In the cases of short duration, the lesions are sometimes more marked in the small intestine than in the colon. The stomach contains undigested food, and mucus which is commonly stained a dark-brown colour. It may be dilated or contracted. The mucous membrane is pale or congested; if the latter, it is usually in patches, and more about the pyloric orifice.

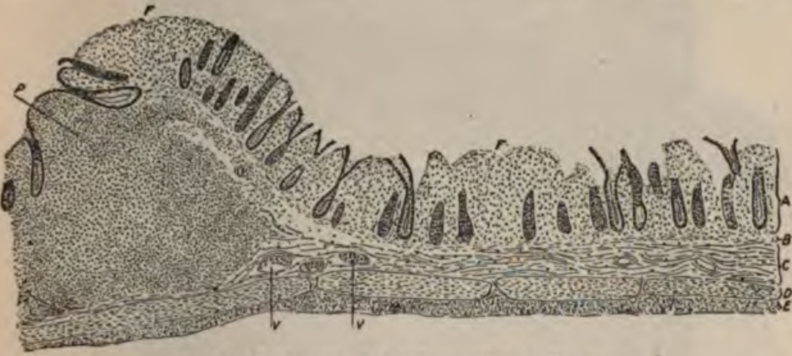


FIG. 65.—Acute catarrhal inflammation of the ileum.

At the left is seen the edge of a Peyer's patch (*P*) greatly swollen. The most striking feature of the lesion is the loss of the superficial epithelium, which is shown in all parts of the specimen. The significance of this depends upon the fact that the autopsy was made but two hours after death. At several points, *F*, *F*, the tubular follicles have loosened and fallen out. The mucosa, *A*, is slightly infiltrated with cells, especially near the Peyer's patch. The submucosa, *C*, and muscular coats, *D*, *E*, are normal. *V*, *V*, are small veins. *History.*—Infant, nine months old, previously healthy; sick three days with severe intestinal symptoms; temperature. 103° to 105° F. *Autopsy.*—Acute catarrhal inflammation of ileum and colon; Peyer's patches red and swollen. The specimen is taken from the lower ileum. The superficial character of the lesion is chiefly due to the short duration of the process.

The intestinal contents are generally green in colour, and thin. The mucous membrane is often coated with tenacious mucus. The small intestine is distended with gas, the large intestine nearly empty, except the transverse colon. The mucous membrane may appear somewhat swollen. In the small intestine there are occasionally seen swelling and œdema of the villi, so that they project abnormally and give a plush-like appearance. Congestion is a constant feature, and it may be simply upon the folds of the mucous membrane, or about the solitary lymph nodules; or it may be intense and involve the whole intestine for some distance. Small hæmorrhagic areas are often seen here and there, widely scattered. In the most severe cases there are marked thickening and uniform congestion, and the appearance is sometimes much like that seen in membranous inflammation. The lymph nodules (solitary follicles) throughout the colon are usually swollen, projecting above the mucous membrane about the size of a pin's head. Peyer's patches may be normal, or they may be swollen and congested, with other evidences of catarrhal inflammation in the surrounding mucous membrane, or more rarely they may be involved when the rest of the mucosa appears healthy. The same is true of the lymph nodules of the small intestine. The lymph nodes of the mesentery are usually swollen and acutely congested, but they may appear normal.

Microscopical appearances.—In interpreting the changes found in the mucosa, the same precautions must be observed as previously stated.

There is usually loss of the superficial epithelium and of that lining the tubular glands at their orifices. Upon the surface of the mucosa and within the tubular glands, fine granular matter is seen derived from the broken-down epithelium. The goblet cells are distended with mucus, and do not stain clearly. The lumen of the tubular glands is narrowed from pressure due to the swelling of the lymphoid tissue which separates them, which is partly from œdema, and partly from cell infiltration (Fig. 65). A thick layer of mucus and round cells, adhering closely to the surface, may resemble a pseudo-membrane (Fig. 66). In fatal cases of moderate severity the superficial portion of the mucosa is infiltrated with round cells and crowded with bacteria of many kinds, the depth to which this infiltration extends depending upon the severity and dura-

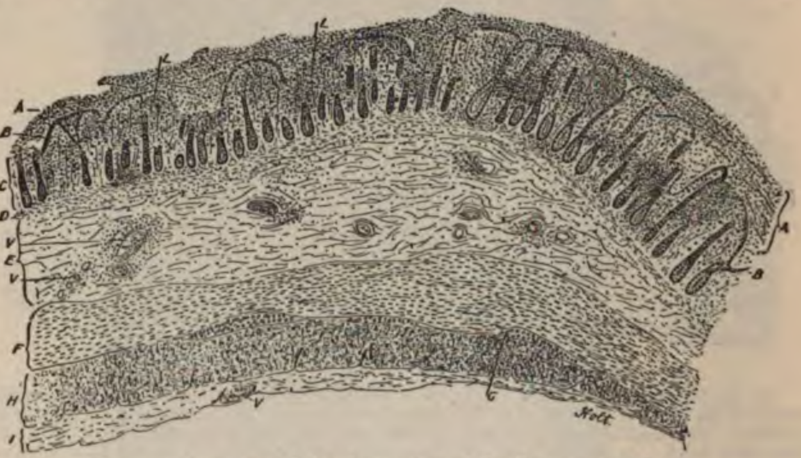
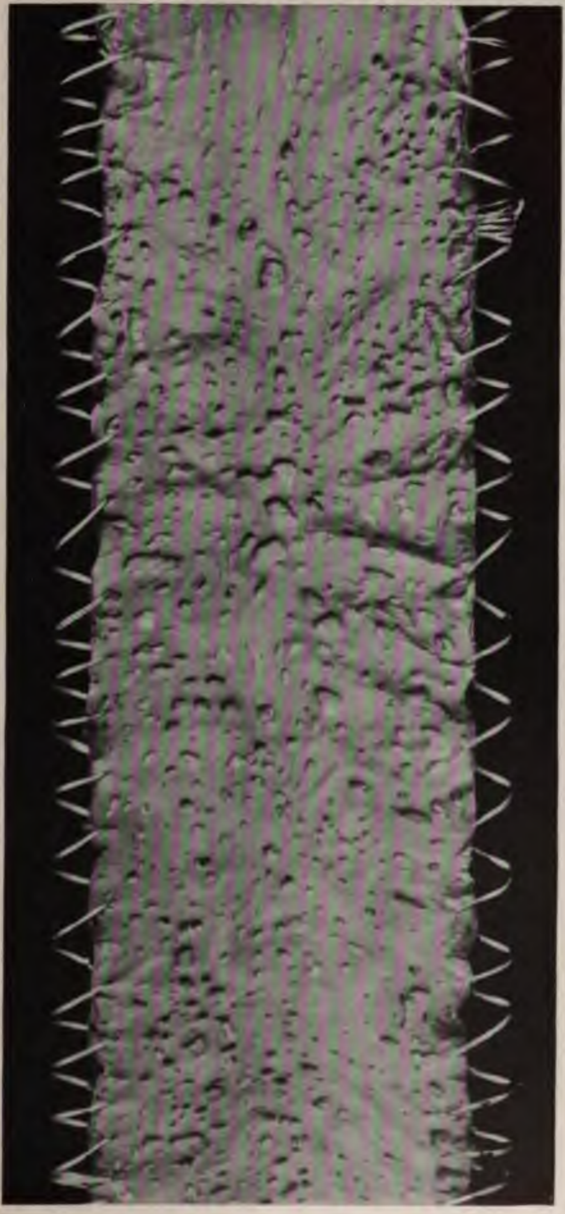


Fig. 66.—Acute catarrhal inflammation of the ileum; severe form.

The mucosa, *C*, is everywhere densely infiltrated with round cells, compressing the tubular follicles, and in places, *L*, *L*, almost effacing them. Upon the surface of the mucosa is a thick layer of cells and mucus. Beneath this the epithelial arches, *B*, *B*, covering the villi can be seen. The lesions are almost entirely of the mucosa. The only changes in the submucosa, *E*, are groups of cells about the small blood-vessels, *V*, *V*. *History.*—Infant six months old; moderate diarrhœa twelve days; severe symptoms with high temperature for six days. There was intense inflammation of the entire colon and lower three feet of the ileum. Intestine greatly congested and thickened. Specimen is from the ileum.

PLATE IX.



Deep Follicular Ulcers of the Colon.

A delicate child, fourteen months old, sick twelve days; stools green, yellow, brown, and watery; no blood; temperature, 100° to 101° F.

The small intestine was normal; ulcers throughout colon. The specimen is from descending colon; the ulcers are deep, and most of them extend to the muscular coat. (For microscopical appearance, see Fig. 68.)

tion of the process. In very severe cases there is found a dense infiltration of the mucosa and of the submucosa also, which in places extends quite to the muscular coat. These cases closely resemble those of the membranous variety, lacking only the exudation of fibrin. The lymph nodules of the colon are swollen to a greater or less degree, chiefly from an increase in the number of lymphoid cells. This swelling may be the most prominent feature of the lesion. If the process is sufficiently prolonged, the lymph nodules may break down and ulcerate. The changes in the lymph nodules of the small intestine and in Peyer's patches are similar to those seen in the colon, but are less marked, and frequently absent altogether. Ulceration in Peyer's patches is extremely rare.

The small veins and capillaries of the mucosa and submucosa are usually distended with blood; small extravasations are very common, and occasionally larger ones are seen.

Catarrhal inflammation, except in its very severe form, which is not frequent, causes no lesions that can not readily be repaired. The most persistent change is usually the swelling of the lymph nodules, which may last a long time, and appears to be an important factor in the tendency to relapses and recurring attacks. If there is a continuance of the exciting cause, or the patient's constitution is a bad one, the process may become chronic.

Catarrhal inflammation with superficial ulceration.—In the most severe form of catarrhal inflammation which does not prove fatal in the earlier stages, extensive ulceration occasionally takes place; usually these ulcers are seen throughout the entire colon, and, in rare cases, a few are found in the lower ileum. They generally begin in the mucosa overlying the lymph nodules, and while they have a wide superficial area, they do not extend deeper than the mucosa. The small ulcers are circular and usually show at the centre a small granular body—the lymph nodule. The larger ulcers result from the coalescence of several small ones, and are irregular in shape. They may be two or three inches in diameter. Sometimes for a considerable distance a large part of the mucosa may be destroyed. Often the entire surface presents a worm-eaten appearance (Plate VIII). On microscopical examination there is seen, in the greater part of the ulcer, complete destruction of the mucosa, the submucosa being densely packed with round cells quite to the muscular coat.

Inflammation of the lymph nodules with ulceration (follicular ulceration).—Follicular ulcers are found at autopsy in about one-third of the cases dying from diarrhœal diseases. They are rarely seen in those which have lasted less than a week, and not often before the middle of the second week. The average duration of the disease in these cases is about three weeks.

In thirty-six cases in which follicular ulcers were found at autopsy, they were present in the small intestine alone in but three cases; in the small intestine and in the colon in six cases; in the remaining twenty-seven they were present only in the colon. When in the small intestine they were seen only in the lower ileum. Ulceration was seen a few times in one or two of the nodules of a Peyer's patch. Ulceration of the large intestine involved the whole colon in about half the cases; while in the remainder the process was limited to its lower portion. The deepest and also the largest ulcers were usually in the descending colon and sigmoid flexure.

In the early stage these ulcers appear as tiny excavations at the summit of the prominent lymph nodules. Later, the whole nodule may be destroyed, and a small round ulcer is formed from one twelfth to one fourth of an inch in diameter (Plate IX). These are quite deep and have overhanging edges; when closely set they give the intestine a sieve-like appearance. By the coalescence of several of them, larger ulcers may form which are an inch or more in diameter. At the bottom of these larger ones the transverse striæ of the circular muscular coat are often plainly seen. I have never known them to cause perforation.

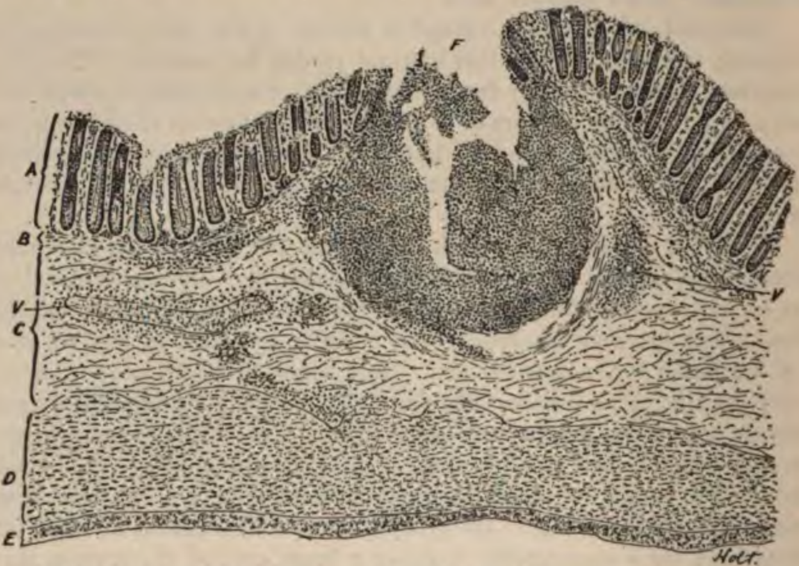


FIG. 67.—Lymph nodule of the colon in the early stage of ulceration—Follicular ulcer.

The nodule, *F*, is much enlarged, and is breaking down and discharging into the intestine. The other changes are not marked. The superficial epithelium is gone: the mucosa, *A*, shows a slight increase of cells, and in the submucosa, *C*, are nests of cells about the small vessels, *V*, *V*. *History.*—Delicate child, thirteen months old; slight diarrhœa four weeks; severe symptoms five days. The colon was filled with ulcers one twelfth of an inch in diameter, one of which is shown in the illustration.

Microscopical appearances.—The lymph nodules are swollen, principally from the accumulation within them of round cells. This is followed by softening, which usually begins at the summit of the nodule and extends downward; the reticulum breaks down, and the cellular contents escape into the intestine (Fig. 67). Softening may begin at the centre of the nodule, which ruptures like an abscess. The destruction of the whole nodule leaves a cavity, which is the follicular ulcer. At first the ulcers correspond in size to the nodule, but infiltration of the adjacent tissue soon takes place, and this may become necrotic. In this way the ulcer extends chiefly in the submucous coat. The lesion is never limited to the lymph nodules; but the extent of the other changes found depends upon the severity and the duration of the process. In cases dying after an illness of a week or ten days, we usually find only moderate changes in the mucosa, and in the submucosa a slight infiltration of round cells, especially about the small blood-vessels (Fig. 67, *V*, *V*). In those which have lasted three or four weeks the ulcers are deeper, and all the structures of the intestine in their neighbourhood are usually involved (Fig. 68). The mucosa is densely packed with round cells, as are also all the tissues in the vicinity of the ulcers; even the muscular coat may be infiltrated. The ulcers, however, rarely extend deeper than the circular layer.

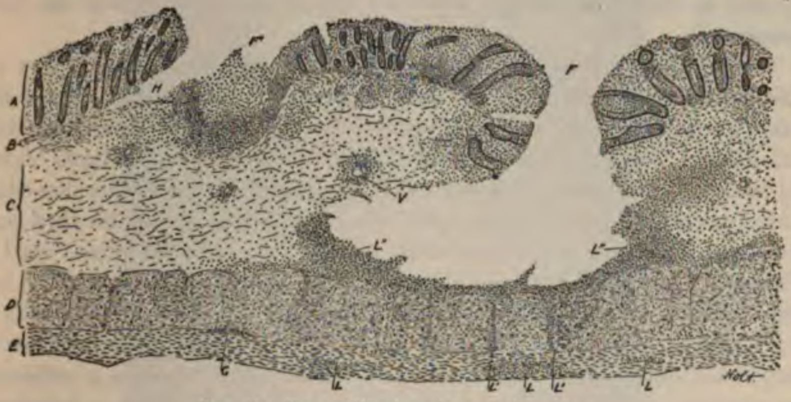


Fig. 68.—Deep follicular ulcer of the colon.

A deep ulcer is shown at *F*, a smaller one at *F'*. The separation of the mucosa at *H* is accidental. There is no trace of the lymph nodule from which the large ulcer had its origin. The destructive process has extended laterally in the submucosa, *C*, and the mucosa, *A*, is falling in to fill up the space. In the vicinity of the ulcers, the submucosa is densely infiltrated with round cells, *L''*, *L''*, which also are seen in the lymph spaces between the bundles of circular muscular fibres, *L'*, *L'*, and some are seen in the longitudinal muscular coat, *L*, *L*. *History.*—Thirteen months old, delicate; continuous diarrhœal symptoms for three weeks. Ulcers found throughout the colon, the largest, one half an inch in diameter. The illustration shows one of the small ones like those in Plate IX.

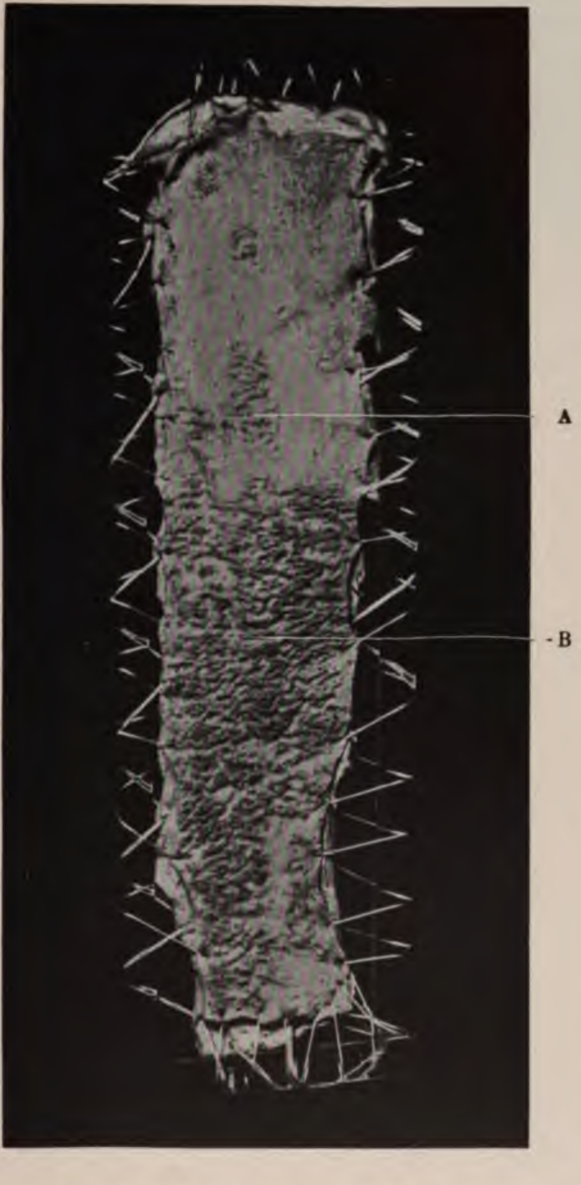
Follicular ulceration of the intestine in infancy, usually terminates fatally if the process is an extensive one. In less severe cases, recovery may take place, the ulcers healing by granulation and cicatrization in the course of from four to eight weeks.

Acute membranous ileo-colitis.—This is the most severe form of intestinal inflammation seen among children. The process differs quite materially from that described as occurring among adults. In only one of my own cases was it associated with membranous inflammation of any other mucous membrane, in that case with membranous gastritis. The most frequent type of membranous colitis is that with severe acute symptoms, both constitutional and local, with a duration of from six to fourteen days. In young infants its symptoms and course are very irregular, and it may be found at autopsy when no serious intestinal lesion has been suspected.

Gross appearances.—There is visible to the naked eye usually very little pseudo-membrane and no deep sloughing. The lesion affects the last two or three feet of the ileum and the entire colon, sometimes only the colon. It is exceedingly rare to meet with any marked lesions higher in the small intestine. The most marked changes are near the ileo-cæcal valve or in the sigmoid flexure and the rectum. In the ileum they may be quite as severe as in the colon (Plate X). The intestinal wall is firm and stiff, and is two or three times its normal thickness. It is not thrown into deep folds, as is the healthy intestine when empty. It is very rare to find false membrane that can be stripped off in patches of any considerable size. When membrane exists, the colour is a yellowish or grayish green, and the surface is often fissured, giving a lobulated appearance. In the parts where no pseudo-membrane can be seen, the surface is usually of an intense red colour and is rough and granular, in striking contrast to the normal glistening appearance. Here and there small extravasations of blood may be seen. In the regions most affected, the normal structures of the mucous membrane—the villi, Peyer's patches, and solitary follicles—can not be distinguished. In a single instance I found an exudation of fibrin on the peritoneal surface of the intestine for a short distance. Except in the lower ileum the small intestine shows no constant changes, and none are usually found in the stomach.

Microscopical changes.—These (Fig. 69) are much more uniform than the gross appearances. The most characteristic feature is the exudation of fibrin, which forms a distinct pseudo-membrane upon the surface of the intestine; it may infiltrate the mucosa, and even the submucosa. Fibrin is seen under the microscope in parts of the specimen, which to the naked eye show no distinct pseudo-membrane, but only a granular appearance. In rare cases a fibrinous exudation may be found upon the peritoneal covering of the intestine. The pseudo-membrane is made up of a fibrinous network containing small round cells, some red blood-cells, and numerous bacteria. The mucosa, and usually the submucosa, are densely infiltrated with small round cells, which in places may be so numerous as to efface the normal elements of the intestine. The tubular follicles are in some places quite destroyed, not a vestige of

PLATE X.



Membranous Inflammation of the Ileum.

A delicate child, eleven months old; mild diarrhœa for two weeks without fever; acute severe symptoms for twelve days; temperature, 100° to 102·5° F.; green and mucous stools; no blood.

The lesions involved the last foot of ileum and entire colon. Specimen is from lower ileum, and shows the abrupt termination of the lesion; the upper part shows normal small intestine; A is a Peyer's patch; B is the inflamed part of the intestine; it has a rough granular appearance and is much thickened.

them remaining. In other places they are compressed and distorted by the accumulation of cells. The great thickening of the intestine is due partly to the cell infiltration, partly to the fibrinous exudation, and partly to œdema. All the blood-vessels, both in the mucosa and submucosa, are

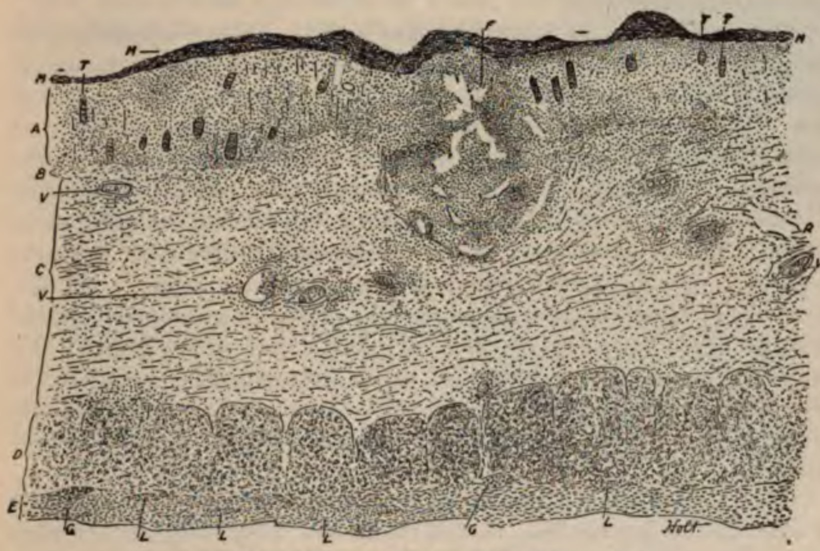


Fig. 69.—Membranous inflammation of the colon.

The intestine is covered with a pseudo-membrane, *M*, which is composed chiefly of granular fibrin; the mucosa, *A*, is densely packed with round cells, and the tubular follicles have almost disappeared, traces only being left at *T*, *T*. The submucosa, *C*, is greatly thickened, partly from cells, but chiefly from fibrin, which with a high power is seen to be everywhere in this coat, as well as the mucosa. Nests of cells are seen in the muscular coats at *L*, *L*. At *F* is a lymph nodule covered by pseudo-membrane, but breaking down at its centre. *V*, *V*, are small blood-vessels with nests of cells about them. *History.*—Fourteen months old; ill nine days; temperature 101° to 105° F.; all stools containing blood. Lesions found throughout colon and in lower ileum. Intestine greatly thickened. Specimen is from ascending colon, where lesion was especially severe.

gorged with blood, and many small extravasations are seen. A necrotic process with the formation of deep ulcers I have never seen associated with membranous colitis.

Associated lesions of ileo-colitis.—The most important one is broncho-pneumonia. It is found in quite a large proportion of the protracted cases, and not infrequently it is the cause of death. I think it is seldom due to an infection from the intestine, although such a thing is possible in septicæmic cases. It occurs rather as it does in any other protracted exhausting disease. In a study of sixty cases, Spiegelberg did not find bacteria in the pulmonary capillaries, and he regards infection through the blood as not yet proved. Pulmonary tuberculosis is not infrequently met with in hospital cases, having no relation to the intestinal disease. I once saw a pulmonary abscess complicating an attack of ulcerative colitis; it was at the apex, and was not associated with suppuration elsewhere. Peritonitis is infrequent. I have met with it but once or twice, and then it was localized and of the plastic variety. Inflammations of the other serous membranes—pleurisy, pericarditis, and meningitis—are all very rare.

The renal lesions of ileo-colitis have been the subject of considerable discussion,* some observers holding that nephritis is a frequent complication of the severer forms of diarrhœa, while others have held it to be rare. The lesions I have usually found in my own cases coincide with those described by others, and consist in marked degeneration of the epithelium of the tubes with but few glomerular or interstitial changes. In three or four instances only have I found well-marked lesions of acute diffuse nephritis at autopsy, or seen its symptoms clinically. I believe it to be a very infrequent though sometimes a most serious complication. The lesions mentioned as usually present are properly classed as acute degeneration rather than as inflammation of the kidney. Its causes are chiefly the irritation of toxins, intensified no doubt by the concentration of the urine. Degenerative changes may be found also in the heart muscle, the liver, spleen, and even in the central nervous system.

Considerable attention has been given lately to a study of the blood in intestinal inflammations, to determine how frequently and in what circumstances a general blood infection (septicæmia) from the intestines occurs. In the great majority of the cases studied under proper precautions the blood is sterile. It is most likely to become infected when there are serious ulcerative lesions; but even these may exist for a long time without producing such a result. It is not probable that the bacteria in the blood are an important factor in producing lesions in other organs.

Symptoms.—(1) *Catarrhal cases of moderate severity.*—The onset is usually sudden, often with vomiting, and for twelve, sometimes twenty-four hours the symptoms may be those of acute indigestion: vomiting, pain, fever, and frequent, thin, green or yellow stools, which are partly fæcal and contain undigested food. Later the discharges contain blood and mucus, are often preceded by pain and accompanied by tenesmus. The stools are very frequent, often every half hour and proportionately small, sometimes less than a tablespoonful being found upon the napkin after severe straining efforts. The mucus may be clear and jelly-like, or it may be mixed with fæcal matter. Blood is seen in some cases in almost every stool, but rarely in clots, usually streaking the mucus. These stools are almost odourless. After two or three days the blood usually disappears, or is seen only as traces in an occasional stool; but mucus is still present in large quantities. The colour of the discharges now becomes dark brown or brownish-green. Prolapsus ani is frequent, and may occur with nearly every stool. Abdominal pain is present, and is often quite intense just before the stool; and frequently there is tenderness along the colon. For the first twenty-four hours the temperature is usually high, from 102° to 104° F. During the greater part of the attack it ranges from 99° to 102° F. There is considerable prostration; the loss in weight is usually marked and continuous; appetite is lost; the tongue is coated and the general appearance of the children indicates serious illness, although no really grave symptoms are present. Convalescence is always slow, and it may be months before the child regains its lost weight (Fig. 70).

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* For a good *résumé* of the subject, see J. L. Morse, Archives of Pædiatrics, 1899, p. 649.

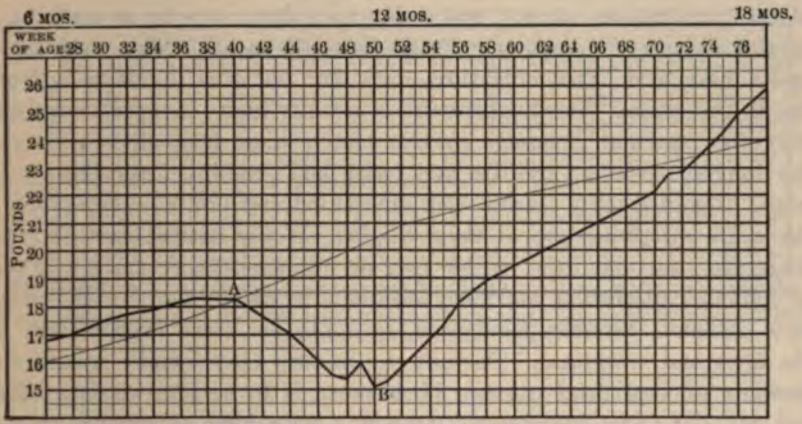

FIG. 70.—Weight curve showing loss from ileo-colitis.

Well-nourished infant; attack of measles at *A* (fortieth week), followed by ileo-colitis, which though not severe continued with exacerbations during September and October. At *B* all symptoms had disappeared except occasional mucus in the stools. Rapid improvement from this time, which was continued during the winter, the child being sent to a warm climate; it was, however, five and a half months before the weight reached the normal average line.

In the milder cases the symptoms point to inflammation of the lower part of the colon only. The constitutional symptoms are not at all marked. The temperature may not be above 101° F.; the tongue may remain clean and the appetite good; the child may be bright and active, and hardly seem at all ill, and yet have from six to eight small mucous and bloody stools a day.

The duration of the acute symptoms is usually about a week, and yet in such cases, even though the child was previously in good condition and properly treated, recovery is slow. The first symptom of improvement is generally the disappearance of blood from the stools, which at the same time become less frequent, and the pain and tenesmus cease. Gradually the stools assume more of a fæcal character, but mucus is likely to persist for two or three weeks; it may be seen in all stools, or only occasionally. In some cases both the mucus and blood disappear and the stools become thin, brown, or green, like those of an ordinary diarrhœa. Although the early stage of very acute symptoms may last but a few days, if there is a continuance for three or four weeks of the brown, mucous stools, with emaciation and slight fever, ulceration is probably present. This is likely to occur if the child is in poor condition, if its surroundings are bad, or if it is improperly treated at the outset. Relapses are readily excited, but cases like the above are rarely fatal except in delicate infants. This is the most common form of ileo-colitis which terminates in recovery.

(2) *The severe catarrhal form.*—This form of ileo-colitis, like that just described, is usually primary. The symptoms closely resemble those of the membranous variety, and a diagnosis from it is to be made only by the absence of pseudo-membrane from the stools. The most rapid case I have seen lasted only three days, but the usual duration is from one to two weeks. The temperature is steadily high; the stools continue very frequent and generally contain blood; there is great prostration, dry tongue, sordes on the lips and teeth, and prominent nervous symptoms. Death usually occurs from exhaustion and profound sepsis while the acute symptoms are at their height. If the patient survives this stage, the case may drag on for four or five weeks, very much like one of follicular ulceration, and then terminate in recovery or in death from slow asthenia, broncho-pneumonia, or from an acute exacerbation of the intestinal symptoms. The autopsy in such cases usually reveals the presence of superficial ulcers. If recovery is to be the outcome, after the symptoms have been nearly stationary for a long time, there is seen a gradual improvement first in the general and then in the local conditions. Convalescence is very slow, often interrupted by relapses, and it may be months before the patient is quite well. In some cases the child never regains its former vigour.

(3) *Follicular ulceration—ulcerative inflammation of the lymph nodules.*—Follicular ulceration is often preceded by other forms of intestinal disease. It is not very frequently met with in infants under six months of age. The great majority of those affected are in poor condition at the time of the attack.

To understand the symptoms of these cases, it must be remembered that follicular ulceration is a terminal process which may follow acute gastro-enteric intoxication. It may be preceded by one or more acute attacks, or by a protracted subacute attack. On account of the feeble resistance of the child or the continuance of the exciting cause, the pathological process gradually extends from the epithelium to the lymph nodules of the intestine, chiefly the colon, which, as already described, pass successively through the stages of swelling, softening, and ulceration. The onset of the illness may therefore be abrupt, with vomiting and high fever; or gradual, without vomiting and with very little fever. The patient may be ill for a week before the exact type which the disease is assuming can be positively determined. It is not possible to mark the transition from acute gastro-enteric intoxication to follicular ileo-colitis. Usually the latter may be assumed to exist whenever, after a very acute onset, there is a continued temperature above 101° F., and when the stools habitually contain large quantities of mucus without blood.

Vomiting is not a feature of these cases; but it is often present at the onset. Throughout the attack it is easily excited by injudicious feeding or medication. The temperature is seldom high, except at first; its usual range is from 99° to 101° F.; toward the close, even of fatal cases, it may be scarcely above the normal. The accompanying chart (Fig. 71) is a very good illustration of the course of the temperature in cases beginning abruptly and ending fatally.

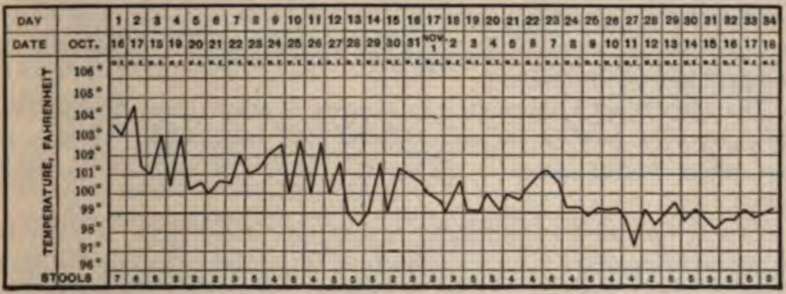

FIG. 71.—Temperature chart of ileo-colitis, fatal on thirty-fourth day. Autopsy showed follicular ulcers throughout the colon.

The stools are seldom very frequent, the number being from four to eight a day. The most constant feature is the presence of mucus, which is mixed with the stools and usually abundant. Blood is not generally present, and a large amount of blood is extremely rare. It was absent entirely in more than half of my cases in which the diagnosis was confirmed by autopsy. A small quantity of blood early in the attack is not uncommon, depending here upon congestion. Large hæmorrhages from ulcers I have never seen. The colour of the stools is most frequently dark green or brown. Fluid stools are seen only during exacerbations. The odour is usually offensive, particularly in protracted cases. The microscope shows epithelial cells in great numbers, and very often an abundance of small round cells, which may be looked upon as the most constant sign of ulceration.

The failure in nutrition and steady loss in weight are very constant in these cases. As emaciation goes on, the skin hangs in loose folds on the thighs; it becomes dry and scaly and loses its elasticity, and occasionally small petechial spots are seen upon the abdomen. The skin over the buttocks becomes excoriated, and bed-sores form over the heels, the sacrum, or the occiput. The abdomen may be moderately distended, or it may be relaxed and soft. Tenderness is not usually present. The appetite is lost, and in most cases great difficulty is experienced in getting children to take a proper amount of nourishment. Continued aversion to food is an unfavourable symptom. Occasionally, when there is fever, fluids are taken eagerly. A returning appetite is always an encouraging sign. The mouth is often dry, the tongue coated, sometimes dry and brown; there may be sordes upon the lips and teeth. Superficial ulcers form upon the mucous membrane of the mouth, and often thrush is seen. The urine is usually diminished, high-coloured, and loaded with urates. Albumin and casts are rarely present. In only two or three cases have I seen nephritis severe enough to be a factor in the result. Tenesmus and prolapsus ani are uncommon.

The average duration of the fatal cases is about three weeks; their course is often marked by exacerbations and remissions. If recovery takes place, convalescence is always very slow and relapses are easily excited.

Very few of these cases recover completely. Even those who survive the primary illness are likely to suffer from intestinal symptoms for many months. Fatal relapses are often brought on by injudicious feeding when the children are apparently almost well. The general health is usually so undermined that the patients continue to suffer from all the symptoms of malnutrition, and ultimately succumb to an attack of some intercurrent acute disease.

The diagnosis of ulceration is to be made from the case as a whole rather than from any special symptoms. If a delicate infant which has previously been prone to diarrhœal attacks, has green mucous stools with low fever, and these symptoms continue with unabated severity for ten or twelve days, ulceration is probable. If such symptoms continue for three or four weeks with steadily failing strength and loss of weight, the diagnosis is almost certain. If, on the contrary, after three or four days of acute symptoms there is improvement in the stools and occasionally some which are quite fæcal in character, even though it may be a week or more before the mucus disappears, we may be quite certain that no ulcers have formed.

(4) *The membranous form.*—This is the gravest form of inflammation of the intestines seen in children, and its symptoms are more often obscure than are those of any other variety. This is particularly true when it affects young infants. There may be at the onset and throughout the course of the disease severe local and constitutional symptoms; or with well-marked constitutional symptoms, the local symptoms may be

slight or of very doubtful character, so that it is often mistaken for some other disease.

In the first form it closely resembles the most severe cases of catarrhal inflammation. The disease begins abruptly, with vomiting, high temperature, and several large, fluid stools. The vomiting does not often continue after the first twenty-four hours. The temperature is at first from 102° to 105° F., and its course may be steadily high (Fig. 72), or remittent. The abdomen is often tender and sometimes swollen. There is severe pain, and at times tenesmus, with prolapse of the rectum. This is intensely congested, and sometimes shows patches of pseudo-membrane upon its surface, thus establishing the diagnosis.

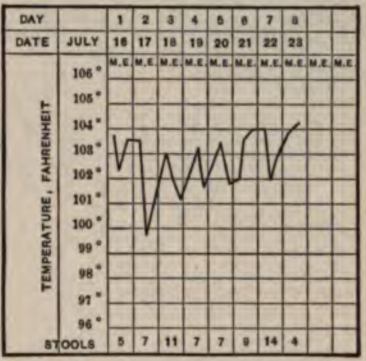

Fig. 72.—Temperature chart of membranous colitis; fatal.

The stools often resemble those of the catarrhal variety, except that blood is more constantly present and usually more abundant, but the only positive point of difference is the presence of shreds or flakes of pseudo-membrane. If the stools are thoroughly washed with water these may be seen as small gray opaque masses, which are then easily distinguished from the transparent mucus. Large shreds of membrane are seldom seen in children. Both blood and mucus sometimes disappear from the stools, which may consist only of dirty water. Under the microscope there may be seen epithelial cells, red blood-cells, and round cells in great numbers.

The presence of cerebral symptoms in these cases of membranous ileo-colitis may lead to great obscurity in the diagnosis. This is most frequently true at the onset. There may be high temperature, great prostration, vomiting, stupor, delirium, and even convulsions; and such symptoms may for two or three days completely mask the intestinal condition. As the case progresses, however, the intestinal symptoms come more and more into prominence, and the cerebral symptoms usually subside. But sometimes this is not the case. I once saw a case closely watched for two weeks by three physicians of large experience, who were agreed in the diagnosis of a cerebral lesion, but not as to its nature, which showed at autopsy only the lesions of membranous colitis. There was a continuous but irregular fever, stupor, retracted abdomen, opisthotonus, unequal pupils, and at times irregular respiration. Two or three days before death the first blood appeared in the stools, and at the same time, during extensive rectal prolapse, a false membrane was seen.

Membranous colitis is also obscure when it affects young infants. Every year a number of these cases are seen at the Babies' Hospital. The prominent symptoms are: rather high, continuous temperature, usually ranging between 101° and 104° F., but following no distinct curve (Fig. 73); wasting, which is not rapid but progressive; frequent stools, which have no constant or striking characteristics. They are usually thin, yellow or greenish in colour, often containing no mucus or blood. Occasionally for a day the stools may be almost normal in appearance. In number they average five or six a day, but often for days only two or three. Outside of a hospital where autopsies are regularly made these cases would pass as excellent examples of infantile typhoid. In many cases the diagnosis wavered between obscure pneumonia, tuberculosis, and typhoid, and was settled only at the autopsy.

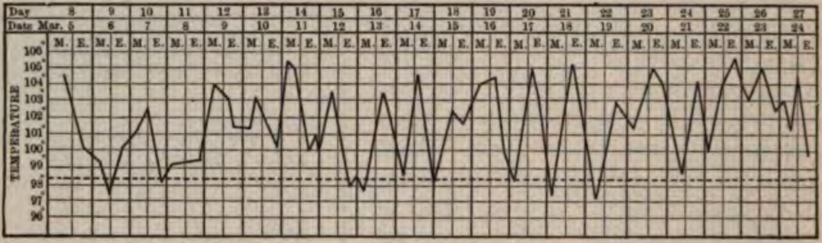

FIG. 73.—Temperature chart of membranous colitis.

Infant fourteen months old, Babies' Hospital. Symptoms for the first two weeks obscure, suggesting first pneumonia, afterward meningitis. Intestinal symptoms for the last two weeks only, never very severe; stools four to six daily, generally green, thin, with much mucus at times, and once or twice traces of blood. Culture four days before death showed streptococci and colon bacilli. Autopsy: No lesion of importance except membranous colitis involving entire colon; a slight catarrhal enteritis.

The duration of membranous ileo-colitis is usually from one to three weeks. Death takes place from sepsis, exhaustion, or from complications. It is probable that almost every case of the severity described terminates fatally when it occurs in an infant. In older children the prognosis is much better as to life, but in them the acute attack may be followed by the chronic form of the disease.

Diagnosis.—Ileo-colitis is to be distinguished chiefly from typhoid fever, intussusception, and meningitis. Typhoid (see chapter on Typhoid) is distinguished by the slower invasion, more constant temperature, enlargement of the spleen, tympanites, and most of all by the Widal reaction and the eruption. The fact that the disease is epidemic is also to be considered. Acute colitis should not be confounded with intussusception; yet the records of intussusception show that a very large proportion of the cases were regarded in the beginning as cases of dysentery. In intussusception, although we have a sudden onset with acute pain, tenesmus, vomiting, and marked prostration, there is rarely fever. The later symptoms—absolute constipation, tumour, tympanites, rising temperature, stercoraceous vomiting, and collapse—have nothing in common with colitis. The membranous form may be confounded with meningitis, and in some cases a differential diagnosis is impossible except by the course of the disease. Marked diarrhœa, even though the stools are not characteristic, should always make one doubt meningitis.

A diagnosis between the different varieties of ileo-colitis is not always possible. Follicular ulceration is distinguished by its lower temperature, rather subacute course, infrequency of blood in the stools, and by the fact that it is usually preceded by one or more attacks of acute gastro-enteric intoxication.

In the catarrhal form, the symptoms of an acute inflammation of the colon are usually manifest from the outset—bloody stools, pain, tenderness, tenesmus, and fever. In the membranous variety such symptoms are sometimes seen; but, as a rule, the local symptoms are less pronounced, while the constitutional symptoms, especially those relating to the nervous system, are usually marked. The course is usually shorter and more intense than in the follicular form.

An agglutination reaction of the *B. dysenteriæ* with the serum of affected children is usually present. But for general use in diagnosis this is not of great assistance. It is subject to considerable variation. Moreover, it is seldom present until the end of the first week of the disease, by which time the nature of the attack is evident by clinical symptoms. Agglutination in the higher dilutions is seen only with the particular type of organism with which the infant is infected.

Prognosis.—This is much worse in infants than in older children. It is especially bad in institutions, and is rendered unfavourable by previous rickets or malnutrition, and by the existence of any complication, especially broncho-pneumonia. Summer cases are never out of danger until the end of the hot season, on account of the great liability to relapses.

Prophylaxis.—What has been said regarding general prophylaxis in the previous chapter, applies equally well to cases of ileo-colitis.

Special emphasis should be placed upon the necessity of energetic early treatment of all the milder forms of diarrhœa, and particularly the cases of acute gastro-enteric intoxication, in order that the process may be arrested before serious anatomical changes have taken place—a thing which is often possible. Equal stress should be laid upon the importance of prompt and radical treatment at the very beginning of the cases with a sudden onset.

Hygienic Treatment.—The general plan recommended in the previous chapter should be followed here. A change of air is desirable for most cases as soon as the acute inflammatory symptoms have subsided. In the protracted cases which drag on a subacute course, this change will often do more than anything else. Plenty of pure fresh air is necessary in all cases. The indications for bathing are the same as in other cases of acute diarrhœa. It is undesirable to crowd these patients in institutions, as they always do better when separated.

The diet during the acute stage should be the same as in cases of acute gastro-enteric intoxication. In the protracted cases the diet presents great difficulties, as the children have little or no appetite, and soon come to refuse everything in the shape of food that is offered. In infancy, the articles which are most to be depended upon are skimmed milk which has been completely peptonized, animal broths, and liquid beef peptonoids. In some cases rice or barley water are well borne; in others, some of the malted foods, although these often increase the number of stools and have to be stopped on that account. Food which leaves little residue should always be chosen. Infants, when very ill, are much more likely to take too little than too much food. A careful record should be kept of the amount actually taken in each twenty-four hours. When this is much below the requirements of nutrition, gavage may be tried. Sometimes all food and stimulants may be advantageously given in this way. In no case should food be given oftener than every two hours, and usually the interval should be three hours, water and stimulants being allowed between the feedings. In older children the diet during the acute stage should be much the same as in infants. At a later period, raw beef, kumyss, or matzoon will be found useful, and during convalescence, eggs, boiled milk, or milk gruels made with rice or barley. Special care should be given to the diet for a long time. For months after an acute attack the intestines are very easily deranged. Relapses are excited by changes in the temperature, by great fatigue or exhaustion, but most of all by improper feeding. Especially in older children should such articles be avoided as oatmeal, potatoes, corn, tomatoes, and all fruits. I have seen a single peach given to a child two years old, excite a dangerous relapse, and a few raisins a fatal one.

Medicinal and Mechanical Treatment.—Cases, the early stage of which is marked by vomiting and thin diarrhœal stools, are to be managed at the outset according to the plan outlined in the previous chapter—viz., free purgation, irrigation of the colon, and stopping all food. When the symptoms of acute inflammation are evident from the outset, as shown by the frequent bloody and mucous stools with tenesmus and pain, the measures to be depended upon are castor oil or saline cathartics and irrigation of the colon, and later opium and bismuth by the mouth. Castor oil should be administered in a full dose at the outset—one drachm at six months, two drachms at one year, and half an ounce at four years. Its primary effect is to clear the intestines, and its secondary effect is soothing. The salines may be used as described in the previous chapter. If the stomach is at all irritable, calomel, one-fourth grain every hour for five or six doses, may be substituted.

Opium is usually required on account of the pain and tenesmus. The dose should be regulated by the severity of these symptoms and by the frequency of the stools. The deodorized tincture and paregoric are, I think, preferable to other preparations.

Repeated small doses are better than a single large dose. It is very important that opium should be withheld for at least twelve hours after the initial purgative. As the pathological process is principally in the colon, and most severe in the lower half of the colon, it can often be much more effectively treated by injections than by drugs given by the mouth. Irrigation of the colon is one of our most valuable means of treatment in these cases. For general purposes a saline solution at 100° to 104° F. should be employed. One or two quarts should be given at one time; it should be injected high into the colon through a long rectal tube, and early in the disease repeated at least twice a day. When the tenesmus is very great and blood abundant, small injections of either hot water (106° to 110° F.) or ice water may be used, and later astringent injections.

The most useful astringents are tannic acid and supra-renal extract; of the former one drachm, and of the latter two drachms, may be added to a pint of hot water. Whether injections are to be used continuously or not will depend much upon the patient.

If they are well borne, they may be given once or twice a day during the attack; but if at every attempt to give them the child struggles, screams, and resists, they may do more harm than good. Complete rest is a very important part of the treatment.

For cases not influenced by the measures mentioned, or those not seen at the outset, bismuth should be tried, but it is of no use whatever unless large doses are administered. One or two drachms of the subnitrate should be given in twenty-four hours to a child two years old, and proportionate doses to older children. This should be suspended in mucilage. Tenesmus and pain are sometimes relieved by the injection of three or four ounces of a starch solution to which from five to ten drops of laudanum are added. Severe tenesmus, when not controlled thus, and when associated with prolapsus ani, is sometimes immediately relieved by a suppository containing cocaine. Not more than one-fourth grain should be used for a child of three years.

Although a serum has been produced which protects animals against inoculation with the *B. dysenteriæ*, its use in the treatment of the various forms of ileo-colitis in children has not been followed by any very striking benefit.

Stimulants are needed in nearly all cases. There are no valid objections to their use even in the youngest infant. The feeble digestion and assimilation of these patients very frequently compel us to use alcohol. Stimulants are indicated by a weak pulse, cold extremities, and great general prostration, no matter at what stage in the disease these symptoms are seen. Old brandy is usually to be preferred. Generally not more than thirty drops every two hours are needed for an infant one year old, but for short periods a much larger quantity may be required. Brandy should always be diluted with at least eight parts of water.

In cases where symptoms have lasted two or three weeks, and the active symptoms have subsided, where the temperature is scarcely above 100° F., and the stools reduced to four or five a day, it is wise to stop all medication and attend only to food and stimulants, with irrigation of the colon every two or three days. One is often surprised at this stage to find that patients do better without drugs than with them. The prevailing tendency is to overdose cases of this type. Careful attention to diet, judicious stimulation, occasional irrigation of the bowel, with change of air, will do much more than any amount of medication.

During convalescence general tonics are required, such as arsenic, iron, nux vomica, and wine. Cod-liver oil should be deferred until the stomach and appetite are quite normal and the stools free from mucus. It should, however, be continued throughout the succeeding winter months.

CHRONIC ILEO-COLITIS.

The severe forms of chronic ileo-colitis follow acute ileo-colitis, usually the catarrhal or follicular form, as the membranous is so severe that the patients rarely survive the acute stage. There may be only a chronic catarrhal inflammation of the mucous membrane, or ulcers may be present.

The milder forms are usually the result of chronic intestinal indigestion.

Lesions.—*Catarrhal form.*—In its milder form it is quite common, but in its severe form it is exceedingly rare. There may be changes in a large part of the small intestine and in the stomach, as well as in the lower ileum and colon.

The gross appearance of the intestine often differs very little from the normal. The mucous membrane is usually of a dull gray or slate colour. Pigmentation may occur as striæ in the mucous membrane, but more frequently it is limited to Peyer's patches and the solitary lymph nodules; these, as well as the mesenteric lymph nodes, are generally swollen.

The microscopical changes are usually marked. The lesion is chiefly one of the mucosa (Fig. 74). The important features are a disappearance of very many of the tubular glands, and in the small intestine of the villi also. There is a very marked cell proliferation in the adenoid tissue of the mucosa, and if the disease has existed long enough there may be a production of new connective tissue. The solitary lymph nodules show usually nothing but cell hyperplasia. The lesions are not uniformly distributed, but occur in patches throughout the intestine. When present in the stomach, they are of the same kind as those described in the intestine, although rarely so severe. In milder cases the gross appearances may show very little change to the naked eye, except swelling of the lymph nodules. Under the microscope there may be found more or less extensive cell infiltration of the mucosa, but rarely any destructive changes or new connective tissue.

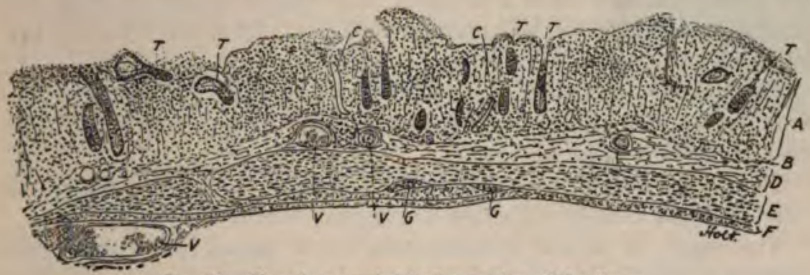

FIG. 74.—Chronic catarrhal inflammation of the ileum.

The lesions affect the mucosa, *A*, almost exclusively. It is somewhat thickened; there is extensive destruction of the tubular follicles, remains being seen at *T*, *T*; there is a great increase in the cells, and some new connective tissue in the mucosa. Large new blood-vessels are seen at *C*, *C*. *History.*—Delicate child, thirteen months old; diarrhœal symptoms for four months; during the first two weeks there was high fever; at death weighed eight pounds. The gross changes at the autopsy were very slight. The section is from the middle ileum.

Ulcerative form.—This is rather rare, for the reason that in infancy a very large proportion of the cases die during the acute stage.

The ulcers are nearly always of the follicular variety; occasionally they are broad and shallow. If the patient dies after an illness of from six to eight weeks, the appearances do not differ essentially from those described in acute cases. If life is prolonged from two to four months, ulcers are found in various stages of repair. Follicular ulcers require from one to three months for cicatrization, and the broad superficial ulcers even a longer time. It is very doubtful whether stricture ever results from these ulcers in children. The mucous membrane shows almost invariably evidences of more or less extensive chronic catarrhal inflammation. Among the very rare lesions are cysts of the colon. Fully developed cysts I have seen but once. The child had an attack of acute ileo-colitis, which became chronic, lasting about five months. He never regained his health, and died one year later from intercurrent disease. In the descending colon and rectum, about twenty cysts the size of a pea, and many smaller ones, were found. They had a thin, translucent covering. On section, a thick, transparent, gelatinous material escaped. They were situated in the submucosa, and were undoubtedly produced by the dilatation of some of the tubular glands whose orifices had been obliterated.

Associated lesions.—The important ones are in the lungs, the most common being hypostatic congestion, subacute or chronic broncho-pneumonia, more rarely pulmonary tuberculosis. It is rare to find the lungs perfectly healthy. The liver is often found extremely fatty in cases associated with great wasting, but in no case have I seen hepatic abscess. The kidneys usually show a more or less intense cloudy swelling, and sometimes there may be well-marked nephritis. Dropsical effusions into the serous cavities are very rare.

Symptoms.—In the milder cases there are only the symptoms of chronic intestinal indigestion with the constant presence of mucus in the stools, usually in large amount.

The severe cases are usually seen in autumn, and are generally the sequel of acute attacks occurring during the summer.

The signs of active inflammation have passed away; the temperature is usually normal; there is no pain or tenderness. There is, however, no improvement in the general condition, and either the weight remains stationary, or the child continues to lose slowly until it is little more than a skeleton. The face is pinched, the eyes sunken, and the cheeks hollow. The lips are pale, often fissured, and bleed readily. The fontanel is depressed. The body is so small that the head seems much too large. The skin hangs in loose folds on the thighs. The mouth is often the seat of thrush, of catarrhal, herpetic, or rarely of ulcerative stomatitis. The tongue may be heavily coated, but is more often dry, glazed, and red.

Although they seldom cry for food, as a rule these children will take nearly everything given them, and in almost unlimited amount. Notwithstanding that it is retained, the more they are fed the more rapid seems the wasting. Vomiting is not common, and seldom occurs except from overloading the stomach or during acute exacerbations.

The stools are rarely frequent, five or six a day being the average; often there may be only two or three a day for a week at a time. They are thinner than normal, but are not often fluid. They contain mucus of a green or brownish colour, usually in large quantity; but rarely blood. The stools are sometimes green, often greenish brown, sometimes a pale gray. They are always large in proportion to the amount of food taken. Undigested food is always present in quantity, and upon the diet depends very much the gross appearance of the stool, the odour of which is almost always offensive. Pus is often found under the microscope, but is rarely visible to the naked eye. Nothnagel and Baginsky have called attention to a form of stools which they believe to be characteristic of wide-spread inflammation of the mucous membrane with atrophy of the tubular glands: they are of nearly normal consistence, homogeneous, dark green or brown colour, and usually offensive; they sometimes alternate with stools of a watery character; under the microscope nuclei are found, but no unchanged epithelial cells; the food remains are sometimes unrecognisable, owing to decomposition.

Prolapsus ani is not so frequent as in the acute cases; but when it occurs it is generally more difficult to control. Flatulence and colic are prominent symptoms in some cases, but absent altogether in many others. As a rule, there is neither abdominal pain nor tenderness. The abdomen is usually distended, and in most cases the enlargement is uniform, but sometimes there is marked epigastric prominence, which is more often from dilatation of the transverse colon than of the stomach. Although the mesenteric glands are enlarged, they can not be felt through the abdominal walls. The skin is dry and scaly, and in the worst cases frequently covered with small petechiæ over the abdomen and lower extremities. About the anus, and over the sacrum, thighs, genitals, and sometimes feet, there are excoriations, and not infrequently ulcerations. The temperature is elevated only during exacerbations, or from inflammatory complications. A subnormal temperature is frequently met with. I have occasionally seen it 95° F. in the rectum. The urine often contains an excessive amount of indican. Dropsy is often present without albuminuria. The weight is stationary, or steadily falls to an almost incredible degree. I have seen one infant weighing but eight pounds at thirteen months; another, thirteen pounds at two years and four months. Ulcers of the cornea are not uncommon. Nervous symptoms are always present. The children are cross and irritable, sleep badly, and frequently have a low, whining cry, which is continued much of the time. Sometimes they are dull, apathetic, and quite indifferent to their surroundings. Persistent opisthotonus is occasionally seen; and there may be contractions of the extremities, but rarely general convulsions.

The duration of the disease is from two months to a year. Comparatively few patients survive more than four months. The progress is irregular, and marked by periods of improvement, during which for a time the patient may hold his own, or even gain in weight. Any trivial cause may excite a relapse, and the downward progress is rapid. Death often occurs during one of these exacerbations, or it may be due to broncho-pneumonia, tuberculosis, or slow asthenia.

Diagnosis.—It is important to distinguish the cases with marked cachexia and slow convalescence, although ultimately resulting in complete recovery, from those which present at a certain stage almost identical symptoms, and yet go on steadily downward, terminating fatally. The difference in these cases is really a difference in the character and extent of the lesions. The first group are probably cases of superficial catarrhal inflammation, or of follicular inflammation which has not gone on to ulceration, these lesions being capable of repair. The second group are the cases of ulceration, in which complete recovery from the lesions is impossible, and repair only partial, if indeed any occurs. In distinguishing between these groups the most important guide is the nature of the symptoms during the antecedent acute attack. The longer the acute symptoms have lasted and the higher the temperature, the greater is probably the extent of the lesions, and the more severe their character.

The diagnosis of chronic ileo-colitis from general tuberculosis is often difficult. Tuberculosis is more likely to be met with in institutions, among the poor of cities, and in children previously delicate and with a tuberculous family history. In chronic ileo-colitis the wasting and anæmia follow the intestinal symptoms, and are usually just in proportion to their severity. For the differential diagnosis of the pulmonary conditions, see the chapter on Tuberculosis. Fever is rarely absent in general tuberculosis or in tuberculous ulceration of the intestine if extensive, though it is not high and its course is very irregular. It is absent in chronic ileo-colitis, except from complications and from the occasional acute exacerbations.

Prognosis.—The prognosis depends upon the child's previous condition, upon the duration of the intestinal symptoms, upon our ability to carry out proper treatment, upon the presence of complications; but, most of all, upon the severity and extent of the intestinal lesions. The possibility of error always exists in estimating the gravity of the lesions, so that no case should be considered hopeless. The most unpromising cases sometimes end in complete recovery. If, however, continuous symptoms have existed for eight or ten weeks without any sign of improvement, recovery is extremely doubtful. The patient may linger for two or three months longer, but usually only to be carried off by the first acute disturbance which occurs.

Treatment.—No greater mistake is made than to give these children week after week the various diarrhœa-mixtures, with the expectation that ultimately the formula which exactly meets the particular case will be found. Drugs are to be used only for the relief of special symptoms. Thus a dose of opium may be needed when the movements are unusually frequent, or castor oil, or calomel occasionally when the stools are particularly offensive. The essential and important part of the treatment consists in injections, careful feeding, stimulation, and change of air. Astringent enemata, however, are of some value. They should not be given continuously, but from time to time should be omitted for a week or two to see what the condition of the stools is without them. I have seen several cases of the milder variety where the constant use of such injections seemed to be an important factor in keeping up the production of mucus. The colon should first be washed with a large amount of a tepid salt or borax solution, and then four or five ounces of the astringent solution injected, and held in place by compressing the buttocks for half an hour.

Alcoholic stimulants must be given in almost all cases, and they may be continued for a long time with advantage. Old port or sherry will sometimes do better than brandy or whisky. The diet mentioned in the later stages of the acute cases should be continued. The predigested foods are useful, especially completely peptonized milk; also are beef preparations as bovinine, and the liquid beef peptonoids, and in some cases raw scraped beef, also the whites of fresh eggs, partially cooked. Fats and starchy foods should be excluded entirely or given in very small quantities. It is usually better to give the carbohydrates in the form of the malted foods. Kumyss and matzoon and buttermilk are useful. The diet must be directed according to its effect upon the stools. Much information may be obtained by thoroughly washing the stools and examining the residue. Nutrition may be promoted by inunctions of cocoa butter, cod-liver oil, or some other form of fat.

The patient should first be put in the best possible surroundings; in no disease is a change of air more to be desired than in this. These cases are trying ones to the physician; for unless he can absolutely control the matter of diet, it is almost useless to attempt to do anything. Still, by careful study of the individual case and attention to minute details, success may sometimes be achieved even when the outlook seemed at the outset the most hopeless. The danger of relapses and second attacks continues long after the primary attack has subsided.

AMŒBIC COLITIS.

Amœbic colitis is rare in children; it is particularly so in infants, probably owing to the fact that nearly all the water taken at this age is boiled. Most of the cases in children thus far reported have been observed in warm climates, although Amberg * has recorded five which occurred in Baltimore, the youngest being two years and eight months old.

The symptoms in the few cases that have been reported in children have differed in no important particular from the disease as seen in adults. In exceptional cases the onset may be abrupt and the attack may run an acute course, terminating fatally in two to three weeks. Such cases are characterized by much abdominal pain and tenderness, frequent mucous and bloody stools containing amœbæ, and some fever, which, however, seldom reaches 102° F.

More frequently this acute onset is followed by a subacute or chronic form of the disease, or the disease may be subacute from the beginning. The protracted cases are the type of the disease most frequently seen. They are very obstinate to treatment. Periods of constipation and apparent recovery often alternate with exacerbations in which the bloody

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* See Bulletin of Johns Hopkins Hospital, December, 1901, for references to literature.

and mucous stools return, with pain, tenesmus, and slight fever. The duration may be from a few months to one or two years. Death may finally occur from exhaustion with extreme wasting, or from some complication, such as hæmorrhage, abscesses of the liver being very rare in children. The diagnosis from other forms of colitis is made only by the discovery of amœbæ in a freshly voided stool.

The general treatment is the same as for other forms of acute or subacute colitis. The special treatment for the purpose of destroying the amœbæ is the use of injections of quinine which may be employed in solutions varying in strength from 1 to 5,000 to 1 to 250.

AMYLOID DEGENERATION OF THE INTESTINES.

This is rarely met with in infants. It is not so infrequent in older children, where it is associated with amyloid changes in the liver, spleen, and kidneys, usually as a result of prolonged suppuration in connection with bone tuberculosis. It is sometimes met with in syphilis. The ileum is the part of the intestine most affected. The process begins in the walls of the arterioles and capillaries, particularly of the villi, and later involves the vessels of the submucosa; subsequently the epithelium may be affected. The mucous membrane in these cases is pale, rather translucent. The condition is recognised by the application of the iodine test; the affected villi become of a brownish-red or mahogany colour.

Amyloid degeneration produces no definite symptoms. Diarrhœa is frequent but by no means constant. The anæmia and waxy cachexia which are present are probably dependent much more upon the associated lesions of the liver and kidneys than upon the changes in the intestines.

TUBERCULOSIS OF THE INTESTINES AND MESENTERIC LYMPH NODES (MESENTERIC GLANDS).

These two conditions are usually, but not invariably, associated, and may be conveniently considered together.

Frequency.—In one series of 109 autopsies upon tuberculous cases from my own hospital records the intestines were involved in 37 per cent. In a second series of 103 autopsies they were involved in 54 per cent. The great majority of the patients were under three years of age. In 131 autopsies upon tuberculous cases published in the Pendlebury Hospital Reports, the intestines were involved in 50 per cent. These patients were mainly between four and fourteen years old. In 209 autopsies upon tuberculous children, chiefly infants, reported by Müller, the intestines were involved in 28 per cent. In 1,346 autopsies collected by Biedert there were intestinal lesions in 31·6 per cent. These figures show that tuberculosis of the intestines is not one of the most frequent forms in children, and that it is rather less frequent in infancy than at a later age. It is most common from the third to the eighth year. The mesenteric lymph nodes were tuberculous in about 50 per cent of my own autopsies, and in 59 per cent of the Pendlebury cases; occurring thus in both series with slightly greater frequency than tuberculosis of the intestines.

Etiology.—In the great majority of cases the mesenteric lymph nodes are infected from the intestines. It is possible, but I believe exceptional, for the infection to occur through the general circulation. With tuberculous ulcers of the intestine, the lymph nodes are, I think, invariably found by inoculation in animals to be tuberculous; although they may not yet be caseous. The infection of the intestinal mucous membrane is from bacilli in the canal. Much stress has been laid upon tuberculous milk as a means by which children are infected. There is little pathological support to be found for the view that children often contract the disease in this way. In 119 autopsies upon tuberculous children, chiefly infants, there was not found one in which the most advanced, and therefore presumably the primary, lesion was in the intestines or stomach. In 127 autopsies, also upon tuberculous infants, Northrup found the most advanced lesion in the intestines in but a single case. While infection from milk is possible, it is certainly extremely infrequent. In my own autopsies, intestinal lesions have been found, with but one exception, only in marked cases of generalized tuberculosis. In not more than one-fourth of the cases in which such lesions were present were they severe. They were usually associated with an advanced pulmonary process, and were doubtless due to swallowing tuberculous sputum.

Lesions.—*Intestines.*—The usual seat is the small intestine, chiefly the jejunum and lower ileum. With extensive disease the large intestine may also be involved, most frequently the cæcum, and exceptionally it alone may be affected. Tuberculous ulcers may be found in the appendix.

The early deposits appear as tiny yellow nodules, generally widely scattered and affecting Peyer's patches. Usually, however, ulcers are present, and often only ulcers are seen. Their size and number vary greatly; there may be only five or six tiny ulcers, or there may be forty or fifty, the largest being two or three inches in diameter. They very frequently involve Peyer's patches. The typical tuberculous ulcer is of irregular shape, with rounded borders and with its longest diameter at right angles to the intestinal axis. When large, it may nearly encircle the gut. The ulcers are excavated; they have overhanging, infiltrated edges of a deep red colour. The surface is covered with granulations. In those which have partially healed a distinct puckering of the intestine occurs, which is especially noticeable upon the peritoneal surface. The small ulcers involve the mucosa only; the larger and older ones the submucosa and the muscular coats, and not infrequently also the serous coat. Perforation may occur, but rarely into the general peritoneal cavity, as a localized plastic inflammation precedes it. There may be adhesions of adjacent intestinal coils, and fistulæ may form, owing to ulceration at their point of contact. With these severe cases there is always associated more or less extensive tuberculous peritonitis, frequently of the ulcerative variety. Like other tuberculous processes, the infiltration and ulceration may cease at any stage, and cicatrization follow. If the ulcers have been large ones, there is always some narrowing of the lumen of the intestine. Stricture rarely results, because the patients die from the general disease before it has had time to occur. Monti has reported a case of obstruction at the ileo-cæcal valve, due to an old tuberculous cicatrix, in an infant of twenty-one months.

Mesenteric lymph nodes.—Usually these tuberculous lymph nodes are from half an inch to an inch in diameter; occasionally they may reach the size of a hen's egg. From a fusion of several of them, tumours of considerable size may be formed. I have seen one such mass as large as the head of a child at birth.

The process is the same as that which occurs in other lymph nodes of the body. There is a tuberculous inflammation, followed by caseation, softening, and abscess, or by calcification. Localized peritonitis is found in all the marked cases; this is usually plastic, but may be suppurative when due to the rupture of an abscess. Pressure upon the vena cava may lead to dropsy in the lower extremities. Ollivier has reported a case in which thrombosis of the vena cava occurred. Pressure upon the portal vein may lead to ascites and dilatation of the superficial abdominal veins. There may be pressure upon the thoracic duct.

Symptoms.—The symptoms of intestinal tuberculosis are exceedingly irregular. Ulcers are very frequently found at autopsy when there have been no marked intestinal symptoms; this is especially true of the small ulcers usually seen in infants. On the other hand, diarrhœa is not uncommon in cases of advanced general tuberculosis where no ulcers are present. It is the most frequent symptom, and may be exceedingly obstinate. The stools do not differ essentially from those in chronic ileocolitis, except in the occurrence of hæmorrhages and in the presence of tubercle bacilli. Hæmorrhages are not very frequent, but they may be so large as to be the cause of death. This occurred in one of my cases, an infant nine months old, the blood coming from a single ulcer in the ileum. Hæmorrhage is more common in older children. In some cases localized abdominal pain or tenderness is present. In advanced cases the symptoms of intestinal ulceration are usually mingled with those of peritonitis, and there are also present the enlarged mesenteric lymph nodes, which may aid in the diagnosis. In the vast majority of cases, these nodes are recognised only by deep palpation. The tumours are generally felt as irregular nodular masses, lying close against the spine, not movable, and sometimes tender on pressure. Other tumours from deposits in the peritonæum may be present anywhere in the abdomen; they may be superficial or deep. The other symptoms are due to the complications already mentioned and to tuberculosis elsewhere.

Diagnosis.—The only positive evidence of intestinal tuberculosis is the discovery of the bacilli in the stools. In the absence of this evidence, the disease is differentiated from simple ileo-colitis, first, by the signs of tuberculosis elsewhere in the body, especially in the lungs, these being almost invariably involved; secondly, by the slow onset and gradual development of the symptoms, while in chronic ileo-colitis an acute attack has almost invariably preceded. Large hæmorrhages always suggest tuberculosis.

The large mesenteric glands are recognised only as abdominal tumours.

Prognosis.—This depends altogether upon the extent of the tuberculous disease elsewhere, as it is extremely rare for the intestinal lesion to be the cause of death. Once formed, the ulcers probably remain, cicatrization being very rare, and then only partial.

Treatment.—The only symptom which ordinarily demands treatment is the diarrhœa. When severe, this is to be managed much as in cases of ileo-colitis, except that irrigation of the colon is, of course, not called for. The chief reliance must be upon diet and internal medication. The drugs which are most useful are bismuth, opium, and creosote, which should be given in pills coated with shellac.

CHAPTER IX.

DISEASES OF THE INTESTINES.—(Continued.)

CHRONIC INTESTINAL INDIGESTION.

As the larger and more complex part of the process of digestion goes on in the intestine, so intestinal indigestion is a more common and more complicated disturbance than gastric indigestion. In many cases we find the two associated, but in perhaps the majority the symptoms relate entirely to the intestinal process. The conditions seen in young infants are so different from those in older children that the cases may be best considered separately.

IN YOUNG INFANTS.—The general causes are the same as those mentioned in connection with chronic gastric indigestion: they are constitutional debility, either congenital or acquired, unfavourable surroundings, and previous attacks of acute disease. Chronic intestinal indigestion is especially common during the first six months, and is seen both in nursing infants and in those who are artificially fed. In the case of breast-fed infants, the mother is often highly nervous, delicate, and anæmic, and may be taking large quantities of fluids of every description, for the purpose of maintaining an abundant flow of milk. Why it is that the milk causes so much disturbance can not always be discovered even by the most careful analysis. The difficulty seems to be most frequently with the proteids, which are often in excess. Sometimes, proteids differing in character from those normally present seem to be produced, as the stools show that they are not digested. The microscope in some cases reveals the presence of many colostrum corpuscles in the milk. In another group of cases, where the condition of the nurse is all that can be desired, the trouble is simply that the milk is too rich; it being then high both in fat and proteids. It may come, although rarely, from the fact that the child gets too much, being nursed either too frequently or for too long a time.

In infants who are being fed upon cow's milk, the most common cause is that the proteids are too high; this is usually the mistake when infants are fed upon plain milk which has been simply diluted. In other cases the fat or sugar may be excessive, as in many of the milk-and-cream mixtures in vogue. Next to this mistake in proportions, is that of overfeeding. Another very important cause is the use of farinaceous foods too early, and in excess.

Lesions.—Strictly speaking, chronic indigestion is a functional disorder without anatomical changes. When the condition has lasted for many weeks or months, as often happens, there may result a low grade of catarrhal inflammation in the colon, frequently attended by hyperplasia of the lymph nodules of the mucous membrane, and sometimes by a similar process in the mesenteric lymph nodes. Chronic indigestion may be the principal and the only symptom in cases of chronic ileocolitis which follow acute attacks.

Symptoms.—The general symptoms are those of malnutrition, or in the more severe form, those of marasmus. These have already been fully described, and need only be mentioned here. The most important are stationary or falling weight, anæmia, poor circulation, often subnormal temperature, almost constant fretfulness and crying, with very little quiet sleep. The tongue is usually coated and the appetite often good, these infants taking food whenever given, and in an almost unlimited quantity. There are few cases in which occasional vomiting does not occur, but it is rarely persistent. So far as the intestinal condition is concerned, the cases may be divided into those with diarrhœa and those with constipation. It may happen that the same child will suffer for a long time from diarrhœa and then from constipation, or the reverse; but usually one condition or the other is habitual. The diarrhœal stools are thin, green, and almost invariably contain curds, either in large lumps or small, flaky masses. They vary in number from three to ten in twenty-four hours. They are commonly passed without pain, although there may be flatulence. The stools have usually a sour, unpleasant odour, but they are rarely foul. They may be irritating to the skin, and cause troublesome excoriations or intertrigo. In some cases the stools contain but little solid matter, the character being that of yellowish-green water. In most of the cases, after the process has lasted two or three weeks, mucus is present, and may then become a constant feature.

If there is constipation, the stools are usually gray or white; they are smooth and pasty or like hard balls passed after much straining, often coated with mucus and sometimes streaked with blood. Often the bowels will not move for days except after the use of laxatives or enemata. The latter often have but little effect, as the rectum may be empty. Constipated cases are especially prone to suffer much from flatulence and colic, the attacks of which may be very severe.

The duration of these symptoms is indefinite. There is little or no tendency to spontaneous improvement, and they may drag on for several months or until the problem of diet is solved. The progress of these cases is marked by frequent exacerbations, during which there is vomiting, and usually fever. Such symptoms are generally dependent upon intestinal toxæmia. A low irregular fever may continue for days or even weeks. Although the general symptoms of failing nutrition are present in most cases, a mild degree of chronic intestinal indigestion with frequent loose movements may sometimes last for months, during which the patients may gain steadily in weight and give every indication of being well nourished. This is much more common in nursing infants than in those who are artificially fed.

Diagnosis.—It is not generally difficult to determine that an infant is suffering from chronic intestinal indigestion; but one should endeavour to go further in his diagnosis and discover which of the elements of the food is causing the chief disturbance. Thus, in an infant fed on cow's milk, we wish to know whether it is the proteids, the fat, or the sugar; or, in another case, whether it is the starch of some proprietary food. Much valuable information may be gained from a careful history of what has already been tried in the case; often some gross error can be detected in the formula used or in the preparation of the food. Difficulty with the proteids is usually shown by colic, constipation more often than diarrhœa, and by curds in the stools; often there is vomiting. Difficulty with the fat is often indicated by loose movements, usually of a yellow or yellowish-green colour and sour odour. Sometimes they are white, smooth and formed, with a peculiarly offensive odour; there may be vomiting or the regurgitation of food in small quantities. Difficulty with the sugar is less common than with either the proteids or the fat, but there may be flatulence, colic, and diarrhœa, with thin, sour, irritating stools. Difficulty with the starch leads to much flatulence and colic, diarrhœa alternating with constipation, and offensive stools. One may find the foregoing symptoms in any combination, for the trouble is rarely limited to a single element in the food. If one is feeding cow's milk, one should begin with what would be a proper formula for a healthy infant somewhat younger, and watch the stools closely for two or three days. The proportion of the offending element should then be reduced until the symptoms it is causing disappear. By carefully modifying milk in this way, a diagnosis of the type of disease can usually be reached.

Prognosis.—This depends almost entirely upon how early the cases come under treatment and how they are managed. There is very little tendency to spontaneous improvement or recovery. The existence of chronic intestinal indigestion is one of the most important predisposing causes of more serious forms of intestinal disease.

Treatment.—Drugs have no part in the treatment of these cases, except now and then for particular symptoms, such as diarrhœa, constipation, or colic. These infants are cured by proper dietetic and hygienic measures, and by these alone. The diet has already been discussed in the chapter on Infant Feeding, and the general management, not less important, in the chapter on Malnutrition.

IN OLDER CHILDREN.—Chronic intestinal indigestion is especially common in children from the first to the fifth year. With the younger children, solid food has generally been used too early and in too large quantities. The articles from which most trouble is seen are imperfectly cooked cereals, vegetables of all kinds, but especially potato. Often the diet is composed almost entirely of farinaceous foods and bread. Children suffering from rickets are particularly liable to be affected. The condition is seen in all grades of society.

Symptoms.—The clinical picture which these cases present is a very common one, and the symptoms are quite uniform. The patients are generally very thin, with very small extremities, a small amount of fat, and a large protuberant abdomen (Fig. 75). There is much flatulence, and usually there is marked tympanites. Such children are pale, anæmic, and sallow in complexion; they have dark rings under the eyes; they are easily fatigued on slight exertion; they are very cross, irritable, and emotional to an unnatural degree. They are hard to amuse, hard to control, and altogether exceedingly difficult patients to deal with. Their growth is retarded if the symptoms have lasted long. They are much below the average in height and weight, but mentally often quite precocious. The sleep is always unnatural and disturbed; and at night they toss about their cribs, waking frequently, crying out and often grinding their teeth, this sometimes leading to the diagnosis of intestinal worms. They perspire very readily, and suffer from cold extremities.

The bowels are usually constipated, the stools being of a light gray colour or perfectly white. They are always formed and generally lumpy.

The odour from the discharges is usually extremely foul. In other cases there is chronic diarrhœa. The stools are not very frequent, rarely exceeding four or five a day, but they are large, thin, gray, green, or brown in colour, often frothy, sometimes offensive, and always contain undigested food. They are often excited by the taking of food. From time to time, in many patients, large quantities of mucus are passed from the intestine; in some cases this comes to be a constant feature of the disease. It results from an intestinal catarrh, which has been set up by the irritation from the hard fæcal masses or from the chronic functional derangement. Large quantities of gas are expelled *per anum*. Pain is not a very common symptom in most cases. The appetite is capricious and usually poor, though some patients will eat everything offered. The tongue may be coated; but unless the stomach is also affected it is usually clean and the breath is not offensive.

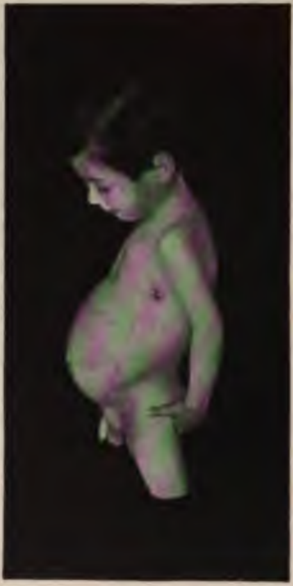

FIG. 75.—Chronic intestinal indigestion.

Patient four years old; symptoms of three years' duration, following attack of acute ileo-colitis. Height, 34 inches; circumference of abdomen, 22¼ inches; weight, 24 pounds.

The nervous symptoms which these patients present are exceedingly varied, and often of the most puzzling character. In many cases they are so severe and so persistent as to lead to the diagnosis of organic disease of the brain. In addition to the condition of general nervous irritability, there may be opisthotonus, tetany, fainting attacks resembling somewhat the seizures of *petit mal,* exaggerated reflexes, attacks of dulness or sometimes stupor, with retracted abdomen, irregular pulse and respiration, and other symptoms strongly suggestive of tuberculous meningitis. Convulsions are not very uncommon. They are usually accompanied by fever, and may be repeated at intervals of a few minutes. Headache and frequent attacks of vomiting which are perhaps to be interpreted as instances of migraine, are occasionally seen. In fact, there is almost no end to the complexity of these cases and the combinations of nervous symptoms which they may present. Most of these are toxic in their origin. The skin shows frequently eruptions of erythema or of urticaria.

Slight fever, also of toxic origin, is sometimes present for many weeks, the temperature usually varying between 99° and 100·5° F. Sometimes for several days it may be normal, and occasionally may rise to 102° or 103° F. during a slight exacerbation in the symptoms. The urine of most of these patients contains a great excess of indican; the amount present indicates very accurately the degree of intestinal putrefaction present, and often fluctuates regularly with the nervous symptoms.

Intercurrent attacks of acute indigestion, with diarrhœa and vomiting, are common and quite easily excited. The course and duration of these symptoms are indefinite. In the most severe forms, if untreated, the patients gradually waste until they die from exhaustion, or fall easy victims of any acute disease which they may happen to contract. There is but little tendency to spontaneous recovery.

Prognosis.—This depends upon the duration of the symptoms, the general condition of the patient at the time treatment is begun, and upon how thoroughly it can be carried out. The symptoms, in the great majority of cases, have existed for several months at the time the case comes under observation. Generally, the greater the mistakes in feeding have been, and the greater the violation of hygienic and dietetic rules, the better the prognosis. A child who has developed chronic intestinal indigestion of a severe type, in spite of the fact that the hygienic surroundings were good, and where the dietetic errors were not flagrant, is not nearly so hopeful a subject for treatment as one whose hygienic surroundings have been poor and whose diet has been especially bad. In cases like the latter, a removal of the causes and the institution of proper methods of treatment almost invariably result in immediate and striking improvement, unless the general vitality of the patient has been reduced to a very low point. In the other cases, where the mistakes have been less marked, and the condition is due more to constitutional than to local causes, the improvement is slower and less striking. Thus, as a rule, hospital patients improve more rapidly than those seen in private practice, because their previous treatment has been so much worse.

Treatment.—In no class of cases that the physician is called upon to treat are results more satisfactory than in many of those of chronic intestinal indigestion, where the intelligent co-operation of the parents or a trained nurse can be secured. If the parents themselves are lax in discipline, and are unable to control the child, an efficient trained nurse should be secured, into whose hands the exclusive management of the child should be placed. The essential part of the treatment is diet and general management. In the second and third years the most important thing is to stop all starchy food for a considerable time, and put the patient upon an exclusive diet of rare beef or beef juice and milk. The milk for many of the patients must be peptonized, as the casein of cow's milk is often very difficult of digestion even for children three years old. By some the fat also can not be digested, and skimmed milk should then be used; in very obstinate cases it should be peptonized for two hours;

in the majority of cases, however, it is sufficient to peptonize it from fifteen to twenty minutes. After a few weeks some carbohydrates may be added, preferably in the form of one of the malted foods, which may be continued until the child can digest some form of starch. The number of feedings should not be more than four a day during the second year, and three or four a day for children during the third and fourth years. These should always be at regular intervals, and nothing whatever given between meals. The meat should be rare scraped beefsteak or mutton; from one to three tablespoonfuls may be allowed once a day. The juice of fresh fruit, especially oranges, may after a time be given once a day, one hour before meals. Kumyss and matzoon are often of very great value in children who are not fond of milk, or who become tired of the diet. Although at first they are taken with difficulty, in many cases a fondness for them is very soon acquired.

After improvement has been going on for two months, bread may be added, at first in small quantities and once a day. This should preferably be stale bread, cut thin and dried in the oven until it is crisp, and given without butter. Two or three times a week raw oysters may be tried. Mutton, chicken, or beef broth, without vegetables, may be given occasionally in the place of one of the milk feedings. After this diet has been kept up for three or four months, if improvement continues, one of the green vegetables may be added once a day, preferably either spinach, stewed celery, or asparagus. After two or three months more of continued improvement, thoroughly cooked rice or macaroni may be given twice a week. With these articles of diet one can get along very comfortably for a year, and no larger variety should be given until all the symptoms have disappeared. When starchy food is first allowed, it should be only in small quantities, and usually with some preparation of diastase. Potato and oatmeal should be forbidden for a long time.

Intestinal irrigation is useful in some cases in which there is much mucus passed. But it should not be forgotten that continued irrigation often keeps up the production of mucus. Astringents should not be used, but only a warm saline solution, and this not regularly. It should be omitted from time to time to see whether the discharge of mucus is not less without it. It is of most value during exacerbations.

The constipation can sometimes be controlled by the diet. Calomel frequently seems to exert a very marked influence, even when the constipation is not severe. It is often wise to administer a full dose every five or six days. In some patients castor oil acts more satisfactorily. It is sometimes objectionable, however, from its tendency to aggravate the constipation. As laxatives in this condition I have found the greatest satisfaction from the use of preparations of cascara and the compound licorice powder. Abdominal massage is also useful.

Drugs directed against the process of putrefaction are extremely unsatisfactory even in older children, but sometimes diminution in the amount of flatulence follows the use of subgallate of bismuth, carbonate of creosote, salol, or salicylate of soda. General tonics are required, and may add materially to the improvement of the patients. Altogether the best one is nux vomica. It may be given in combination with the bitter wine of iron just before meals three times a day. This increases the appetite and acts favourably upon the constipation. Cod-liver oil, particularly in the early stage, is badly borne. It should be withheld in all cases until very marked improvement in the condition of the digestion is assured.

Relapses are easily excited by indiscretion in diet, and parents should be impressed at the very beginning with the necessity of adhering rigidly to the diet prescribed, for a long period. It very often happens that the improvement which is seen after one or two months of careful treatment is so marked as to lead the parents to the belief that a cure has been accomplished, so that they relax their vigilance and allow improper articles of food which are almost certain to induce a relapse. If the case is an aggravated one, and the symptoms of long standing, it is wise to tell parents at the outset that a year's treatment is the minimum in which anything permanent can be accomplished.

The general treatment of the patient must not be overlooked. Proper clothing, regular exercise in the open air, cool sleeping rooms, massage, sponging every morning with cold water, are all of very great importance, and contribute almost as much to the results obtained as the special measures adopted. (See chapter on Malnutrition.)

The improvement in the nervous symptoms of the patient is one of the first things noticed, and is often marked in a few days after the beginning of treatment. From an irritable, fretful, peevish child the patient is sometimes totally changed in disposition in a few weeks, so as to become quiet, affectionate, docile, and playful.

INTESTINAL COLIC.

The term *colic* is applied to any severe paroxysmal pain occurring in the intestines. It may be due to many causes. The colic of lead and arsenic poisoning are both very rare in children; but colicky pains are present in appendicitis, intussusception, ileo-colitis, and, in fact, in all the severe forms of intestinal inflammation. Colic may be due to swallowing certain substances, especially foreign bodies and the seeds of fruits; and in rare cases it may be excited by the presence of roundworms when they are numerous. In all the conditions mentioned, colic is only one of the symptoms, although it may be a very prominent one.

The special and peculiar colic of infancy is that which is associated with flatulence, and is due to indigestion. Here it is a symptom only, but may be a most troublesome one. This form of colic belongs essentially to the first six months of life, and is more frequent during the first three months. It may be seen at any time when digestion is very feeble. Many young infants suffer from colic a large part of the time; others have only occasional attacks, which are often repeated at a certain time in the day, usually toward evening.

The flatulence to which the colic is usually due may be from decomposition in the food or intestinal secretions, or in both. It is seen quite as often in nursing infants as in those who are artificially fed. Any of the elements of the milk may be a cause of colic, but in fully four-fifths of the cases it is the proteids. The colic of nursing infants is nearly always due to the fact that the milk is excessive in proteids, or else that these are digested with special difficulty. If cow's milk is the food, it is the proteids which are usually at fault. It is rare that the quantity of sugar present in cow's milk is sufficient to be a cause of colic; but this may happen when sugar has been added, more frequently with cane sugar than with milk sugar. It is extremely rare for the fat to be a cause of colic. In infants whose food consists largely of farinaceous substances, colic is also very common.

As a result of the decomposition taking place in the intestine, gas accumulates, and, the intestines lacking sufficient muscular force to expel it, distention follows. To this in part the pain is due. But spasm of the muscular walls of the intestine is also an element in producing the pain. In some of the most severe cases it is possible that the spasm may be accompanied by a slight transient intussusception. Colic may occur without flatulence, as in cases when it follows cold feet or chilling the surface. In these cases also, muscular spasm appears to be the principal factor in causing the pain. Intestinal colic may occur alone, or it may alternate with or accompany gastric colic.

Symptoms.—These are in most cases so typical as to be easily recognised. They are always more severe in delicate and highly nervous children. In the severe attacks there is contraction of the features, the loud paroxysmal cry, subsiding for a few moments and then beginning with renewed intensity, drawing up of the lower extremities, and in male infants contraction of the scrotum. With these symptoms the abdomen is usually found tense and hard. With the expulsion of the gas, the symptoms subside at once, and the child usually falls asleep. In the most severe attacks there may be considerable prostration, cold extremities, and perspiration. When the symptoms are less severe there is only continual fretfulness, and the child can not sleep. When colic is habitual there are very few hours in the twenty-four when the child seems to be entirely comfortable. In nursing infants there may at times be difficulty in distinguishing the cry of colic from that of hunger, as infants suffering from colic will usually take food eagerly, and this is often followed by temporary relief. In colic, however, the pain soon returns, and often is more severe than before. The cry of colic is usually violent and paroxysmal; that of hunger is apt to be prolonged and continuous, and is not accompanied by the other symptoms mentioned as indicating abdominal pain. In older children the less frequent causes of colic mentioned at the beginning of this article, especially appendicitis, should be borne in mind.

Treatment.—When colic is due to flatulence of the intestine, nothing given by the mouth has much effect in relieving the symptoms. Certainly food should not be given. The purpose of treatment during the attack is to assist the child to get rid of the gas; as this is usually in the colon, the most efficient means is by massage or enemata. At first an injection of four or five ounces of lukewarm water should be used. If this is not successful, two ounces of cold water with half a teaspoonful of glycerin may be tried. This rarely fails to start peristalsis and expel the gas. In conjunction with these measures, dry heat should be applied to the abdomen by means of hot flannels or a hot-water bag, and the feet should be well warmed. In cases of colic not associated with flatulence, where the pain is probably the result of muscular spasm, opium in some form is required in addition to heat or counter-irritation. The treatment between the attacks and the treatment of habitual colic should be directed toward the indigestion, upon which they depend.

CHRONIC CONSTIPATION.

Constipation may be said to exist whenever the stools are less frequent, harder, and drier than normal. During the first six months infants usually have two movements a day. Many, however, have only one; but if this is normal in character the child is not constipated. In other cases, although there are two and even three stools a day, they may all be small, dry, and hard, having all the characters of constipated stools, and the case should be treated accordingly.

Etiology.—The causes of chronic constipation are many and far-reaching. It may be due to a diminution in the secretion of the intestinal glands or of the liver. The movements are then hard, dry, very light-coloured, and are associated with much flatulence and other signs of intestinal indigestion. Very often the principal factor in constipation is insufficient muscular contraction in the intestine. The fæcal masses are then propelled so slowly and remain so long in the intestine that the fluid portion is absorbed, the residue becoming, in consequence, so dry and hard that it is difficult to expel. In other cases constipation depends upon the fact that there is insufficient volume to the stools, as may be the case when the food given leaves very little residue. Constipation may depend upon local causes, as, for example, where an evacuation of the bowels is resisted on account of pain from fissure of the anus or from hæmorrhoids. Although not the primary cause, this condition may be sufficient to keep up the constipation indefinitely. It may, in rare cases, be due to a congenital condition, such as a narrowing of the large intestine at some point. The most important causes of constipation may be grouped under two heads: diet, and conditions giving rise to muscular atony.

Diet.—In breast-fed infants the trouble is usually a lack of fat and an excess of proteids in the milk. In those who are artificially fed it is often because the fat is too low, and sometimes because both the fat and the proteids are too low, the stool lacking volume. In other cases the cause of constipation is indigestion, in still others the use of " sterilized " milk. During the second and third years the cause may be too much cow's milk, particularly that which has been boiled, or the use of an excessive amount of starchy food. As during the first year, the trouble with cow's milk is that it contains too much casein, the digestibility of which has often been rendered more difficult by the boiling. In older children the cause may be an excess of starchy food and a lack of sufficient green vegetables, meat, and fruit.

Muscular atony.—The most common cause of muscular atony is habit; in a large number of cases lack of proper training is the principal etiological factor. If the inclination to have a stool is regularly disregarded it soon ceases to be felt. The ordinary irritation from fæcal masses produces no response whatever. The longer such a condition continues the more obstinate does it become. This is an important factor in all cases. Another potent cause of muscular atony is rickets. In this disease the muscular walls of the intestine suffer like the muscles of the extremities, and become incapable of doing their work. Again, any form of malnutrition in which there is feeble muscular tone may cause or aggravate constipation. It is often seen as a sequel to acute attacks of diarrhœal diseases, particularly when these have been prolonged. Want of sufficient muscular exercise is a frequent cause. There are many children who rarely suffer from constipation in summer when they have plenty of out-of-door exercise, who very often do so in winter when such exercise is wanting. A loss of muscular tone is not an infrequent result of the prolonged and indiscriminate use of purgative drugs or enemata.

Symptoms.—In many cases no symptoms are present except the local ones, the general health being excellent and the nutrition in no way disturbed. In the majority, however, there are symptoms of greater or less severity, depending somewhat upon the cause of the constipation. There may be simply flatulence and colicky pains, or the irritation of the hardened fæcal masses may produce a slight catarrhal inflammation of the sigmoid flexure and the rectum, so that mucus and sometimes traces of blood may be passed with the stool. Hæmorrhoids may develop even in infancy, and frequently the constant straining leads to the production of hernia. In many cases there are from time to time nervous symptoms resulting from the absorption of various toxic materials from the intestine. There may be headache, dulness, fretfulness, disturbed sleep, and often associated signs of intestinal indigestion. The urine often contains indican in excess, and there may be slight fever.

Diagnosis.—This includes the discovery of the cause and the principal seat of the constipation. To arrive at the former the most careful and thorough investigation should be made of the child's diet and habits. It is desirable to determine whether the seat of trouble is the rectum, the upper part of the colon, or the small intestine. If a suppository is almost immediately followed by a normal stool, one may be sure that the rectum only is at fault, and that it needs but a little extra stimulus to make it do its work. This is common in infants who are too young to make any voluntary efforts. In such cases there are no other symptoms present. In others, the white or gray stools, marked flatulence, offensive breath, and general irritability, leave no doubt of the fact that the trouble is in the small intestine and depends upon indigestion.

Treatment.—This is always difficult, and in obstinate cases must be continued for a long time. The co-operation of an intelligent mother or nurse is absolutely indispensable. To establish the habit of regular stools should be the first step, for without this regularity nothing can be done. Even in infants only a few months old proper habits are often easily formed if the child is put upon the chamber or chair invariably at the same hour. When a local stimulus is required in addition an oiled glass rod or a glutin suppository may be inserted. An older child must be taught to heed the first impulse to evacuate the bowel. Regular habits can hardly be formed unless the same time each day is chosen for the movement. That to be preferred is soon after the morning meal, as taking food into the stomach usually starts a peristaltic wave which is continued throughout the intestine. With older children breakfast should be early enough to allow ample time for this duty before the other engagements of the day; and nurses should be impressed with the importance of the early formation of proper habits on the part of their charges. Stretching the sphincter under an anæsthetic is sometimes of great benefit, especially where tonic spasm is present.

Food.—With nursing infants who get good breast-milk constipation is rare. Where the milk is low in fat and high in proteids, constipation is not uncommon. For the measures by which such milk can be improved, see chapter on Breast Feeding.

In feeding cow's milk, constipation is overcome by getting the exact proportions of proteids and fat which are suited to the infant. With most infants during the early weeks from 2 to 3 per cent fat and 1 per cent proteids succeed best; with those a little older, from 3 to 4 per cent fat and 1·5 per cent proteids. During the last half of the first year 4 per cent fat and from 2 to 3 per cent proteids will be found satisfactory. (See Infant Feeding.) To feed an infant two or three months old upon 2 per cent fat and 2 per cent proteids—which is what is usually given when cow's milk is simply diluted once with water—almost invariably produces constipation. With most infants during the first year, constipation may be, if not cured, at least prevented by proper milk modification.

During the second year, children who suffer from constipation should have both cream and water added to the milk, to reduce the proteids without lowering the fat. Suitable proportions can be obtained by adding two tablespoonfuls of cream to two-thirds of a glass of milk, and filling up the glass with water. Very great improvement may often be brought about by substituting malted foods for farinaceous foods. Meat broth and beef-juice are quite laxative on account of their extractives and salts. Fruits are valuable in all these cases; but only the juice should be given until a child is eighteen or twenty months old. That of almost any fresh fruit may be employed. After two years pulpy fruits may be given; baked apples, oranges, stewed prunes, and in summer, fresh peaches, plums, and pears, may be given in small quantities; but all fruits with seeds should be avoided.

For older children who are on a mixed diet the amount of starchy food should be moderate, oatmeal being perhaps the best cereal. Milk should be given rather sparingly, and even then may be advantageously modified as for the second year. It is sometimes advisable to stop milk altogether and give only cream, from four to six ounces of which may be allowed daily. It may be used with the breakfast cereal, mixed with potato or rice, added to soups or broths, and taken in various other ways. All bread should be made from whole wheat or unbolted flour. Meat and broth may be allowed freely, also green vegetables, one of which should be given every day. All fruits allowed infants may be used, but in larger quantities, and in addition raw apples. Of the dried fruits, only dates, prunes, and figs are admissible, and these are better stewed than raw. Fresh fruit is preferably given in the morning, oranges being especially useful when taken on rising.

Either hot or cold water, when taken an hour before breakfast, may be of considerable benefit to older children. The sparkling waters, like Vichy or Apollinaris, are often better than plain water.

Massage, when properly employed, is useful in conjunction with other measures, but rarely succeeds alone. It should be given for five or ten minutes after retiring and just before rising. The hand must be warm, but no oil used, the purpose being not to make friction upon the skin, but to move the skin and abdominal walls upon the intestines. This should be done with a circular motion, changing the point from time to time until the whole abdomen has been thoroughly covered. In addition to this a general kneading of the abdomen may be employed. Only slight pressure should be made until the child becomes accustomed to the process, when quite deep pressure will be tolerated. The intestinal coils may often be felt contracting under the hand during massage.* In general torpor of the intestines massage is useful, and when properly done may affect the small as well as the large intestine.

A proper amount of active muscular exercise is necessary and should be made a part of the treatment in every case. Yale (New York) has called attention to the importance of posture during the stool, he having found that in many cases a cure was effected simply by substituting a low seat on a nursery chair or closet for the high one previously used. The low seat afforded the child an opportunity to strain to some purpose, while the higher one with the legs dangling, made this almost impossible.

Suppositories.—In many cases, particularly in young infants who are not old enough to initiate the muscular effort, a slight stimulus to the rectum is all that is required. The cone of oiled paper has a great reputation in domestic practice and is not objectionable. It may be of assistance in establishing the habit of a daily movement at a regular time. Soap suppositories produce a more marked irritation; although their immediate effect is quite satisfactory, they should not be continued indefinitely. They are, however, less objectionable than glycerin suppositories. The latter, for an immediate effect, are convenient and usually efficient; but their prolonged use, especially in infants, is likely to set up a catarrhal proctitis. The gluten suppositories produce less irritation and are consequently slower in their effect, but they have not the disadvantages of the soap or glycerin. Medicated suppositories are certainly one of our most efficient measures; if drugs must be employed, they are perhaps open to the fewest objections when used in this way. The following are the best drugs for this purpose, the dose being that for a child of two or three years: ext. nux vomica, gr. $\frac{1}{12}$; ext. belladonna, gr. $\frac{1}{24}$; ext. hyoscyamus, gr. $\frac{1}{24}$; sulphur, gr. ij; purified aloes, gr. $\frac{1}{4}$; aloin, gr. $\frac{1}{24}$. A good combination is aloin, gr. $\frac{1}{24}$; ext. belladonna, gr. $\frac{1}{24}$; ext. nux vomica, gr. $\frac{1}{12}$; ol. theobrom., gr. x. In obstinate cases this may be used night and morning, and later at night only. After some improvement has occurred the aloin may be omitted. Many of the proprietary suppositories contain the ingredients mentioned, particularly belladonna, the dose of which is often considerably larger than should be given. Suppositories are chiefly useful when the trouble is the rectum and lower colon; but very little is to be expected from them when it is higher in the intestine.

Enemata.—These should be restricted to cases in which only temporary relief is desired. An injection of an ounce of sweet oil may facilitate the passage of very hard and dry stools, and larger injections of soap and water may be used to break up hard fæcal accumulations. For immediate effect an injection of one drachm of glycerin in half an ounce of water is perhaps the most efficient means at our command. Cases of fæcal impaction are rarely met with in children. They are to be managed as in adults, by repeated injections of warm water or of ox-gall, and sometimes by mechanical removal. For continuous use enemata are not to be advised, for larger and larger quantities are required to produce the effect.

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* See Karnitzky, Archiv für Kinderheilkunde, Bd. xii, p. 66.

Medicinal treatment.—This is the least important part of the management of chronic constipation. No plan is worse than to give some active purgative every third or fourth day and trust matters to take care of themselves the rest of the time. The most valuable drugs are those which stimulate the muscular walls of the intestine, such as cascara, nux vomica, belladonna, and hyoscyamus. These are particularly useful in atonic constipation associated with rickets and following diarrhœal diseases, but they are valuable in all cases. With most drugs the prolonged use of small doses is better than the occasional use of large ones. Calomel is indicated in cases attended with dry, very white stools and marked flatulence; one fourth to one half grain of the tablet triturates may be given for two or three successive nights in conjunction with other means. Cascara may be used either in the form of the elixir, dose from one half to one drachm, or the fluid extract, from one to five drops. Rhubarb, either in the form of the syrup or the mixture of rhubarb and soda, may be given occasionally, but it is not adapted to continuous use. Of salines, phosphate of soda is best for continuous use in infants. All the preparations of malt possess slight laxative properties, and are useful in conjunction with dietetic and other medicinal means; either Trommer's extract of malt or maltine may be employed. Castor oil should seldom be given for chronic constipation. The frequent use of small quantities of olive oil is often a good means of treatment in the case of young infants, the oil being added to the food.

Summary.—The treatment of constipation is palliative and curative. The palliative measures are drugs, suppositories, injections, and enemata. Cure is accomplished only by diet, massage, exercise, and the formation of regular habits. An average case of chronic constipation in a child four years old may be managed as follows: Massage for eight minutes, morning and night; the juice of half an orange and a glass of Vichy immediately upon rising; a breakfast of oatmeal with one ounce of cream, dried bread with butter, an egg, half a glass of milk with cream and water added; a dinner of soup, one starchy vegetable—e. g., potato with cream, and one green vegetable, beef-steak, baked apple or prunes, dried bread and butter, and water to drink; for supper, cream-toast, egg, dried bread and butter, or Graham crackers, half a glass of milk with cream and water added; a suppository containing nux vomica and hyoscyamus given at bedtime.

Hypertrophy and Dilatation of the Colon.—It is probable that in many cases of chronic constipation, especially among rachitic infants, a considerable degree of dilatation of the colon occurs. However, it seems to be but a temporary condition, disappearing by the third or fourth year.

There is another form of dilatation which may be permanent; it is associated with a marked degree of hypertrophy of the muscular walls of the colon. The reported cases thus far are few in number, but have been observed both in infants (Hirschsprung,* Mya †) and in older children (Osler, Hughes ‡). The prominent symptoms are two: obstinate constipation, which in most of the cases has continued from early infancy, and is sometimes so severe that the patients have gone for two weeks without a movement of the bowels; and distention of the abdomen, which may be extreme, but which may disappear and the abdomen become perfectly flat after the fæces and flatus have been discharged. There is usually emaciation, and from time to time there may be diarrhœa. Death may occur in infancy, or the patients may live to adult life.

In the cases which have come to autopsy there has been found an enormous dilatation of the large intestine, chiefly of the transverse colon and the sigmoid flexure. In one case (Hughes'), in a boy of three years, the colon was four inches in diameter, and held fourteen pints of water. In none of the cases was there stricture at any point. The mucous membrane has invariably been found ulcerated, this clearly being a secondary process. The muscular walls have been greatly hypertrophied. The condition is without doubt a congenital one. Treatment is palliative only. In some of the cases the condition seems to have been aggravated by the use of large enemata.

INTUSSUSCEPTION.

Intussusception consists in the invagination of one portion of the intestine into another. It occurs most frequently in infancy, being at this age the most common cause of acute intestinal obstruction. The accident is not a common one, but the life of the patient generally depends upon its prompt recognition.

Varieties.—Usually the upper part of the intestine is invaginated into the lower, although the reverse is occasionally seen. Intussusceptions may occur at any point in the intestinal tract. Those of the small intestine are called *enteric*; those of the colon, *colic*; and those occurring at the ileo-cæcal valve, *ileo-cæcal* (Fig. 76). Of 90 cases under ten years of age, in which the variety was determined by autopsy or operation, 75 were ileo-cæcal, 9 colic, and 6 enteric. In the ileo-cæcal form a few inches of the ileum pass through the ileo-cæcal valve, and then invagination of the colon occurs. Cases in which the ileum passes through the valve, but without invagination of the colon, are sometimes classed separately as an *ileo-colic variety.*

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* Hirschsprung, Jahrbuch für Kinderh., Bd. xxvii, p. 1.

† Mya, Revue Mensuelle des Maladies de l'Enfance, vol. xii, p. 633.

‡ Osler, Archives of Pædiatrics, vol. xi, p. 112.

Intussusceptions of the dying, as they have been called, are met with in my experience in about eight per cent of all autopsies made upon infants; they are not often found in children over two years of age. They are descending, enteric, easily reducible, and multiple—usually from eight to twelve invaginations being present. They are more frequently in the jejunum than in the ileum. They usually involve but two or three inches of the intestine, but may include ten or twelve inches. They are found in autopsies upon patients dying of all varieties of disease, and are probably produced in the death agony. These intussusceptions are without symptoms, and are of no clinical importance.

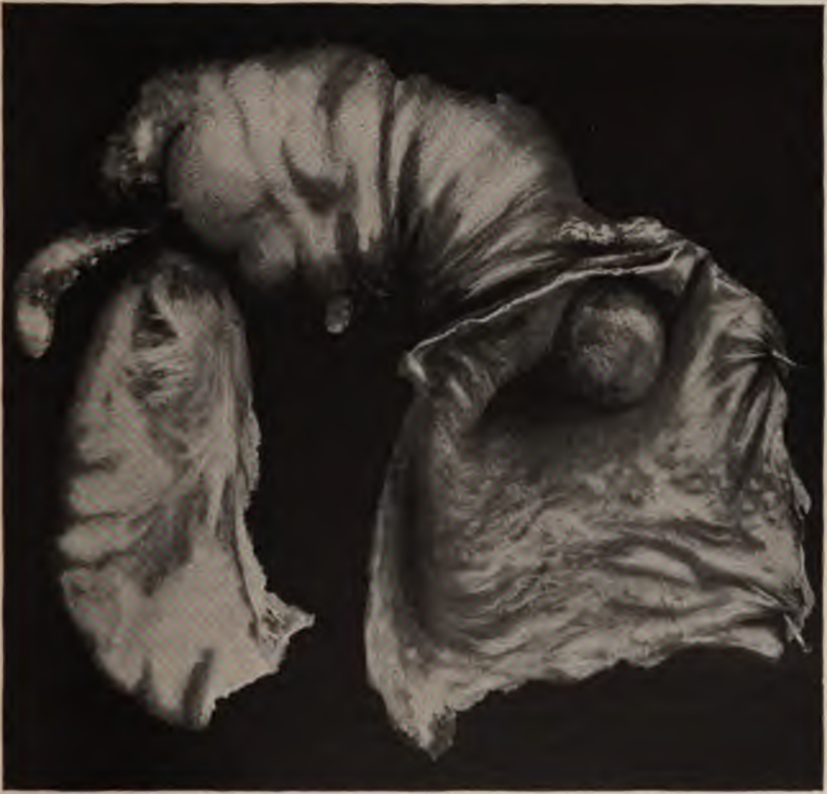

FIG. 76.—Ileo-cæcal intussusception. A specimen removed from a child in the New York Infant Asylum.

Etiology.—Of 358 collected cases under ten years, the following are the ages reported: under four months, 28 cases; from four to six months, 113; seven to nine months, 71; ten to twelve months, 18; one to two years, 32; two to ten years, 96. Three fourths of the cases which occur in childhood are, therefore, in the first two years, and one half of them between the fourth and ninth months. The greater frequency in infancy is attributed to the thinness of the intestinal walls, the greater mobility of the cæcum and ascending colon, and the presence of other intestinal derangements at this age.

Males are more often affected than females. Of 268 cases in which the sex was mentioned, there were 174 males and 94 females. For this fact there is no explanation. The exciting causes of an attack are extremely obscure. The great majority of cases occur in children who were apparently in perfect health. Some previous intestinal disorder was present in about three per cent of the cases I have collected—diarrhœa, dysentery, colic, chronic indigestion, and constipation, all being mentioned. In four cases the intussusception was ascribed to injury of the abdomen. The association with the general diseases is too infrequent to be of any importance.

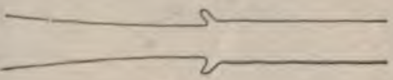

FIG. 77, A.

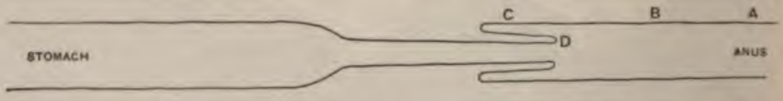

FIG. 77, B.—Mechanism of intussusception. (Treves.)

Lesions.—Nothnagel's vivisection experiments * have shown conclusively that intussusceptions are formed by the irregular action of the muscular walls of the intestine. They can be produced or released at will by varying the application of the electrical current. In the artificial intussusception there is first a contraction of a certain part of the intestine, and if this ceases abruptly the normal gut below this point turns upward and folds over upon the contracted portion, thus forming a minute intussusception (Fig. 77, A). When once begun, the intussusception increases solely at the expense of the external layer (Fig. 77, B). Thus, while the apex of the tumour D remains unchanged, the part of the sheath at A passes to B and then to C, so that the lower part of the intestine is drawn over the upper, rather than the upper crowded into the lower. The mechanism of the invagination was apparently the same when a part of the intestine was first paralyzed by crushing, as in the cases in which a spasm of the intestine was first produced.

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* Beiträge zur Physiologie und Pathologie des Darms, Berlin, 1884. A full abstract is to be found in Treves's Intestinal Obstruction, London, 1884, to which I am indebted for many points in this article.

There is no doubt that pathological intussusceptions are produced in the same way as in these experiments. As the invagination takes place, the mesentery is drawn in with the bowel, and always lies between the sheath and the inner layer. To allow intussusception to occur, the mesentery must be unduly long, stretched, or lacerated. Its attachment to the spine causes the intussusception to describe an arc of a circle, the concavity of which is always toward the spine. It also causes a puckering of the tumour. Invagination does not necessarily produce either obstruction or strangulation, but usually both are present, and are the chief causes of the symptoms. Traction upon the mesentery leads to obstruction in its vessels, causing congestion, œdema, hæmorrhages, and even gangrene. Obstruction is chiefly due to swelling. It may be due to dragging of the mesentery, which brings the apex of the tumour against the side of the gut, or to bending of the intussusception.

The great cause of irreducibility in the first two or three days is swelling. I have several times seen at autopsy or operation the intussusception easily reduced, except the last two or three inches of the cæcum or ileum, which was swollen to the thickness of from a fourth to half an inch. Adhesions may prevent reduction, but rarely before the fourth day; they are often absent as late as the sixth or seventh day. They are usually between the internal and middle layers of the intussusceptum, and are due to local peritonitis. In chronic cases, however, they form the principal obstacle to reduction. Other causes of irreducibility are twisting of the tumour and pinching of the prolapsed intestine, especially of the ileum by the ileo-cæcal valve.

Gangrene and sloughing of the gangrenous portion of the intestine occur much more often in acute than in chronic cases. Portions of intestine were passed *per anum* in 24 of 362 cases under ten years, or about six per cent; but only two of these were in infants. Toward the end of the second week is the time when the separation of the sloughs is to be looked for. The amount of intestine discharged, varies from a few inches to several feet. Two cases are on record in which the entire colon was passed, the patients recovering, but dying several months later from other causes. At the autopsies the ileum was found attached to the lower part of the rectum just above the anus. In acute cases gangrene occurs about the upper end of the tumour, and the intestine usually comes away in one large mass. In chronic cases shreds of intestine may be discharged for several weeks.

Symptoms.—The clinical picture of a case of intussusception is a striking one, and when acute the symptoms are so uniform that, once seen, it can scarcely be overlooked a second time. The patient, usually between six and twelve months of age, is taken suddenly ill with severe pain and vomiting; the pain recurring paroxysmally every few minutes, and the vomiting being first of the contents of the stomach, and afterward bilious. There may be one or two loose fæcal stools, then only blood or blood and mucus are passed without any admixture of fæces. The general symptoms are those of great prostration, or even collapse—pallor, feeble pulse, apathy, and normal or subnormal temperature. The abdomen is relaxed. A tumour is present in the left iliac fossa, or it is felt *per rectum*. Later there is tympanites; the vomiting and pain continue; there is a steady increase in the prostration, and toward the end a rapidly rising temperature, which may reach 105° or 106° F. before death occurs from collapse. If the symptoms continue longer the signs of peritonitis are added. In subacute cases the onset is less abrupt, and pain, vomiting, and constipation less constant and less severe; but the same symptoms are present. In chronic cases the onset is with vague, indefinite intestinal symptoms; pain, vomiting and bloody discharges are usually wanting; there are progressive wasting and more or less diarrhœa, but only the presence of the tumour leads to the recognition of the condition.

Onset.—Of 193 cases under ten years in which data upon this point could be obtained, the onset was sudden in 181 and gradual in 12 cases. By far the most frequent symptoms of onset are pain and vomiting. In a smaller number of cases the initial symptom is diarrhœa or a discharge of blood and mucus.

Pain.—This is rarely continuous, but is intermittent, recurring in paroxysms like those of ordinary colic, but of great severity. No pain in infancy is to be compared with it. The child often shrieks so as to be heard all over the house. Pain is a prominent symptom in over three fourths of the cases, and is very rarely absent. It is generally more marked for the first two days, but may continue throughout the attack. In a few cases the pain is localized, being usually referred to the region of the umbilicus.

Vomiting is more marked at the onset, but may continue throughout the attack. Like pain, it is more frequent in the acute cases. It is due to intestinal obstruction. Vomiting is present in fully four fifths of all cases. Usually it is persistent and uncontrollable; it is often projectile. If food is given, vomiting often occurs as soon as it reaches the stomach. Stercoraceous vomiting occurs in about fifteen per cent of the cases in children under ten years, but is not common in infancy. It is rarely present before the third or fourth day. Although a bad sign, it is not by any means a fatal one, as nearly one half the cases in which it has been noted have recovered; it is to be regarded as indicating complete intestinal obstruction rather than strangulation.

Tumour.—This is one of the most important symptoms for diagnosis because of its frequency and its peculiar character. It is present early in the disease, often in a few hours after the initial symptoms. The following table shows the frequency with which a tumour was present in the different varieties, and the position which it occupied in each. The anatomical variety was determined either by autopsy or operation.

The Relation between the Tumour and the Different Varieties of Intussusception in 188 Cases under Ten Years.

SEAT OF TUMOUR.	SEAT OF INTUSSUSCEPTION.					
	Ileo-cæcal.	Ileo-colic.	Colic.	Enteric.	Not stated.	Total.
Region of cæcum	..	3	..	1	7	11
" " ascending colon	1	..	..	..	12	13
" " transverse colon	3	..	..	..	13	16
" " descending colon	3	..	..	..	18	21
" " sigmoid flexure	4	..	1	..	8	13
Rectal	25	1	7	..	28	61
Protruding from anus	9	..	1	..	12	22
Umbilical region	..	..	..	1	..	1
Movable	..	..	..	1	2	3
Site unknown	1	..	..	..	..	1
Total	46	4	9	3	100	162
No tumour felt	10	2	..	1	13	26

Tumour was thus made out during life in eighty-six per cent of the cases; and in the great majority of these it was discovered at the first careful examination.

It will be noted that in one half of the cases the tumour was either felt in the rectum or protruded from the anus, and that in over two thirds it had advanced as far as the descending colon or beyond. The tumour may reach the rectum in a surprisingly short time, even when the invagination begins at the ileo-cæcal valve. In one of my own cases it was felt in the rectum in less than twelve hours from the onset. The usual description, "sausage-shaped," is accurate when the invagination is large, the tumour then being from four to six inches long and about an inch and a half in diameter. It is often curved.

During manipulation, or during an attack of pain, the tumour may become more prominent and may be distinctly erectile. To the touch the rectal tumour closely resembles the os uteri, the central opening being the apex of the intussusception. When protruding from the body, the tumour is rarely more than two inches long. It is usually of a deep purplish colour, and may be gangrenous. It has been mistaken for prolapsus ani, polypus, and even hæmorrhoids. In a case which came subsequently under my observation, the tumour was discovered by the mother before the physician had suspected the condition.

Condition of the bowels.—Bloody stools are a very constant symptom. Of 186 cases under ten years in which this condition of the bowels was noted, blood in the stools was present in seventy-six per cent. There are very often two or three thin, diarrhœal movements, and then only blood and mucus are passed with no trace of fæces and with no fæcal odour. The amount of blood varies from a quantity sufficient to stain the mucus to an ounce of semifluid blood. It rarely occurs without some mucus. Such discharges frequently follow attacks of severe colicky pain, and may occur several times in an hour. They may continue, or after a day or two they may be succeeded by absolute stoppage. Diarrhœa throughout the attack is rare in children, particularly so in infants. It belongs generally to chronic cases. Constipation is complete in most of the acute cases, neither gas nor fæces being passed; a fact which the discharge of blood and mucus may lead one to overlook.

Tenesmus is very common if the tumour is rectal. Relaxation of the sphincter is met with in a considerable proportion of the cases when the tumour is in the sigmoid flexure, or rectum.

During the first twenty-four or forty-eight hours the *abdominal walls* are soft and relaxed, and may even be retracted. Usually there is then little resistance to abdominal palpation. After the second or third day there is usually tympanites; but this does not necessarily mean that peritonitis exists. Localized tenderness is a symptom of some importance when a tumour is absent. Scanty urine has been noted in a few cases, but is of no special value in showing the seat of obstruction.

In the acute cases the *general symptoms* are very striking. They are the ordinary ones of severe shock—marked prostration, pallor with an anxious expression of the face, general muscular relaxation, cold extremities, cold perspiration, and often a subnormal temperature. Early there is marked restlessness, and even convulsions may occur. Later there are apathy, dulness, and semi-stupor. The temperature during the first twenty-four hours is usually not elevated, and is frequently subnormal. Toward the close of the disease it rises rapidly to 103°, 104° F., or even higher, quite independently of peritonitis. A rapidly rising temperature is always a bad symptom, and usually betokens death within twenty-four hours. Wasting is seen in the chronic cases, and may be quite rapid.

Course, Duration and Termination.—Of 198 cases under ten years, 155 were classed as acute, lasting less than seven days; 33 as subacute, lasting from one to four weeks; 10 were chronic, lasting over four weeks. Nearly all the cases occurring in infancy are acute. The duration of the disease in 92 fatal cases was as follows: less than twenty-four hours, 2 cases; two to four days, 44 cases; five to seven days, 22 cases; one to two weeks, 18 cases; two to three weeks, 6 cases. Thus one half the cases died upon the third, fourth, or fifth day. Of 57 cases terminating in recovery, 66 per cent were reduced in the first or second day. (See table, page 436.)

Spontaneous reduction is, without doubt, possible in intussusception.

Treves and others are of the opinion that this happens much more frequently than is generally supposed, and that many cases of severe colic are really cases of slight intussusception. There are seen in both conditions the tendency to vomit, the paroxysmal pain, the constitutional depression, and often the sudden cessation of the symptoms, especially under the influence of opium; but to make a positive diagnosis of invagination in such cases is impossible. Intussusception may be cured spontaneously by sloughing of the invaginated part, the continuity of the intestine being preserved by adhesions. Such a result is rare at all ages, and is almost never seen in infancy. Even though recovery from the attack takes place, complete restoration to health is very rare.

The most frequent cause of death in acute cases is shock. Peritonitis is not found at autopsy or operation so often as might be expected. In 58 autopsies, it was seen but twenty times, and in seven of these it was limited to the intussusception. In but 7 cases was there perforation. In chronic cases death is usually from exhaustion or complications.

Diagnosis.—This usually presents no difficulty in acute cases provided the physician has the condition in mind. The great majority of such cases present nearly all the classical symptoms—viz., sudden onset, recurring colicky pains, frequent vomiting, bloody and mucous stools without fæcal matter, general prostration or collapse, and low temperature. The records show that the most common error is to regard the case for the first few days as one of gastro-enteritis or ileo-colitis, the physician's attention being engrossed by the vomiting and bloody stools. Given the other usual symptoms, the presence of the characteristic tumour is conclusive evidence of intussusception. Unless the patient is very much relaxed, a satisfactory examination is possible only under full anæsthesia. In any case of acute obstruction in infants, intussusception should first be considered. Chronic cases present no diagnostic symptoms except the tumour. In both acute and chronic cases the rectal examination is most important for diagnosis, and often settles the question at once.

Prognosis.—The prognosis of intussusception depends upon the age of the patient, upon the variety of the disease—whether acute, subacute, or chronic—and upon the time when proper treatment is begun.

There were collected by Pilz* in 1870, 94 cases under one year, the mortality being 84 per cent. Of 135 cases of the same age reported between 1870 and 1891 the mortality was 59 per cent. In Pilz's table, of 51 cases between one and ten years of age, the mortality was 68 per cent; while of 82 cases between one and ten years of age, from 1873 to 1891, the mortality was but 46 per cent. Formerly recovery was rare, except in cases of sloughing; but with earlier diagnosis and a better understanding of the proper methods of treatment, the mortality has been very much reduced. Combining the figures of Pilz with my own, there are 362 cases with 231 deaths, or 63·5 per cent.

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* Jahrbuch für Kinderh., Bd. iii, p. 6.

Gibson (New York) in 1900 collected 187 operations for intussusception, with a general mortality of 51 per cent; in 126 cases, in which the tumour was reducible, it was but 36 per cent; in 61, in which it was irreducible or gangrenous, it was 80 per cent. The table gives the mortality in relation to time of operation:

TIME OF OPERATION.	Number of operations.	Number reducible.	Mortality. Per cent.
First day	35	33	37
Second "	36	30	39
Third "	33	20	61
Fourth "	15	6	67
Fifth "	..	..	73
Sixth "	..	..	75

After the second day the chances of success are greatly reduced.

Treatment.—One should first attempt reduction by inflation or injections with the assistance of taxis, and, this failing, resort early to laparotomy.

Inflation should always be done under an anæsthetic, unless there is extreme relaxation. Occasional inversion of the child may be practised, to get the assistance of traction of the intestine above upon the seat of invagination. An ordinary hand bellows with a catheter attached is the best apparatus; air should be injected very slowly, and prevented from escaping by pressing the buttocks tightly together. The best guide to the amount introduced is the tension of the abdominal walls. A thorough trial of this method should not occupy more than fifteen or twenty minutes.

Reduction is sometimes indicated by rumbling sounds, and by the abdomen resuming its normal contour because the whole of the colon is filled, in place of the unequal distention before present. In some cases a gush of fluid fæces has followed disinvagination. Not infrequently all such decisive symptoms are absent, and the physician may be in doubt whether or not reduction has taken place. The air is allowed to escape and the abdomen examined while the patient is still under chloroform. The right iliac fossa should be examined with the greatest care, as it often happens that all the tumour except the last few inches has been reduced. The question of reduction must be frequently decided by the general symptoms. If vomiting continues, if no gas or fæces pass the bowels, if there is no improvement in the pulse or the general condition, and, besides, if the temperature rises, it is almost certain that reduction has not been effected. In a very acute case a few hours' delay may turn the scales against the patient. The abdomen should be opened if the child is strong enough to bear the operation. Even in cases not so acute, it is not admissible to postpone operation more than a few hours, since all delay adds to the difficulty of reduction and diminishes the chances of success.

Injections of fluids.—A saline solution may be used, milk and water, or thin gruel. The temperature should be from 100° to 105° F. for the relaxing effect. The fluid is placed in a fountain syringe suspended four or five feet above the patient's bed. The injections should be made through a catheter, the escape of the fluid being prevented as in inflation. From time to time the patient should be inverted. It may be desirable to increase the pressure by raising the syringe to the height of five or six feet, but more is rarely advisable. After from ten to twenty minutes the water is allowed to escape and the abdomen examined.

The choice between inflation and injection depends somewhat upon individual experience. The danger of rupturing the intestine belongs alike to both; but that it is not likely to occur with either is conclusively shown by the fact that in a series of 225 collected cases, all in children, and including nearly all those reported between 1870 and 1891, this accident has been recorded only once. In rare cases the symptoms may continue after reduction. Pick records such a case in which laparotomy was done with the belief that reduction had not been effected. No intussusception was found, and the continuance of the symptoms was attributed to intestinal paralysis.

After reduction the patient should be kept absolutely quiet and moderately under the influence of opium for two or three days. The diet should be very light. Cathartics especially should be avoided for several days.

Recurrence of the invagination is not uncommon. It was noted in 13, or about six per cent, of my collected cases under ten years; of this number nine recovered and four died. Recurrence is more likely to happen in the first twenty-four hours after reduction; this was the time in nine of the thirteen cases. It may, however, be as late as a month, rarely later. In one half the cases there was but a single recurrence, but three, four, and even six recurrences in the course of a few weeks have been seen. Ludwig reports a case in an infant eight months old in whom twenty-two recurrences were seen in one month. This was of the colic variety; it could hardly happen in any other form.

Laparotomy is indicated as soon as a thorough trial of reduction by inflation or injection has been made without success. In the very acute cases the operation should not be delayed an hour after such failure is evident. Needless delays have caused death in many instances. The operation should not be looked upon as a last resort in hopeless cases, but as a measure which, if employed reasonably early, offers a fair prospect of success where disinvagination can not be accomplished by any other means. All statistics show that the result depends more upon the time when the operation is done than upon any other single factor. With earlier diagnosis and more prompt resort to operation in case of failure of reduction by mechanical means, the mortality from intussusception has during the past ten years been steadily falling. A large proportion of the infants who suffer from this accident may be saved if they receive proper treatment in season.

CHAPTER X.

DISEASES OF THE INTESTINES.—(*Continued.*)

APPENDICITIS.

THE terms *typhlitis*, *perityphlitis*, and *perityphlitic abscess* were formerly much used to denote certain forms of inflammation occurring in the right iliac fossa. Of late these terms are but little employed, as it has been shown that these conditions are almost invariably due to disease of the vermiform appendix. The existence of typhlitis as a separate and independent disease is exceedingly rare, if indeed it ever occurs except as a result of fæcal impaction.

Etiology.—The predominance of the male sex holds even in childhood. Of 101 cases under fifteen years, 72 were males and 29 were females. Appendicitis is exceedingly rare in infancy, the condition having never once been found in about 2,000 autopsies, nearly all upon children under two years old, in three institutions with which I have been connected. It does, however, occasionally occur even in very young infants. The youngest cases that have come under my observation were infants of nine and fourteen months respectively. Goyen's case was in an infant only six weeks old; Shaw's, seven weeks; Demme's, seven weeks; and Savage's, nine weeks old.

Appendicitis is rather more frequent in children who have suffered from digestive disturbances, particularly chronic constipation, than in others. Regarding the exciting cause of an attack but little is yet definitely known. In only a very small proportion of the cases is a foreign body discovered in the appendix. In one of my own a pin was found, and a number of similar cases are on record. There is, however, almost invariably a fæcal concretion which is moulded into the shape of a foreign body, and formerly often regarded as such. This probably has some relation to the attack by causing disturbances of circulation and increasing the chances of infection. The bacteria most frequently found in abscesses from appendicitis are streptococci usually associated with colon bacilli.

Lesions.—The position of the appendix is extremely variable. It may be found low in the pelvis, as high as the liver, in front of the kidney, and sometimes near the umbilicus. This anatomical peculiarity accounts for the variation seen in the situation of the abscesses due to appendicitis. Inflammation of the appendix may be acute catarrhal, suppurative, or gangrenous, and it may be recurrent or chronic.

Catarrhal appendicitis.—In this form there is an inflammation of the mucous membrane with swelling of the follicles and infiltration of the mucosa with round cells; the process may extend to the muscular and possibly also to the serous coat. As a result, the appendix is thickened and stiffer than normal. It may become distended with mucus or muco-pus to the size of the thumb or even larger. The inflammation sometimes results in the formation of superficial ulcers involving the mucous membrane. Catarrhal appendicitis may subside without any serious consequences, and complete recovery follow. In most cases, however, some changes remain; there may be adhesions; the lumen may be constricted at any point; and sometimes communication with the cæcum may be shut off entirely. Catarrhal appendicitis may be followed by a chronic form of inflammation or by the suppurative form.

Suppurative appendicitis.—This may follow one or more attacks of the catarrhal form, or the inflammation may be of the suppurative type from the beginning. In this variety the inflammation of the mucosa is much more extensive; the infiltration of the muscular layer is more marked, and the serous coat is usually involved. As a result, the appendix usually becomes distended with a foul, purulent fluid. This process may terminate in several ways. Drainage into the intestine may be reestablished and the pus escape in this way, the inflammation of the coats of the appendix undergoing resolution, but leaving some thickening and adhesions. This termination is not common. A more frequent course is for perforation to take place either by ulceration or localized gangrene. Perforation may be followed by a general septic peritonitis, or the inflammation may be circumscribed by adhesions and result in a localized peritoneal abscess. Such an abscess may subsequently burst into the general peritoneal cavity, or spontaneous opening may occur into the intestine, the bladder, or the vagina; or the abscess may burrow for a long distance. Secondary lesions are occasionally seen in children; there may be suppurative pylephlebitis, abscess of the liver, empyema, pneumonia, or general pyæmia.

Gangrenous appendicitis.—Gangrene of the appendix may be localized, in which case it is usually one of the forms of termination of the suppurative inflammation; or it may be general, in some cases involving the entire appendix, in others only the distal portion. Such a process is the result of some cause which completely arrests the circulation. The rupture of a gangrenous appendix is usually followed by a general septic peritonitis which develops with great rapidity; less frequently the peritoneal inflammation is localized and there develops a peritoneal abscess.

Chronic appendicitis.—This usually follows one or more attacks of the catarrhal form. It results in thickening, adhesions, constrictions, and more or less interference with the communication with the cæcum, the appendix being sometimes distended with mucus or muco-pus.

Symptoms.—*Catarrhal appendicitis* is often not recognised, and in many cases a diagnosis is impossible. The milder attacks are usually passed over as acute indigestion. The only suspicious symptoms are acute abdominal pain and tenderness. In a very large proportion of the cases the pain is not in the region of the appendix. It may be referred to almost any part of the abdomen, and is frequently about the umbilicus. When the abdomen is carefully examined, by making pressure with the finger point, there is generally found well-defined localized tenderness, in the right iliac fossa, one or two inches from the spine of the ileum on an arc described with the spine as a centre. The onset is often with vomiting, and there is some fever, though rarely over 101:5° F. The bowels are usually constipated, although occasionally diarrhœa is present. The disease gradually subsides in the course of four or five days, the local symptoms being the last to disappear.

In the more severe attacks the pain and tenderness are much more marked. There is never any area of induration, but the swollen appendix may sometimes be felt if the abdominal walls are thin and relaxed. The onset is usually more severe than in the cases first described; the vomiting may be repeated several times, and constipation is often marked. The early temperature frequently reaches 102° or 102·5° F.; but it soon falls to 100° or 101°, and in two or three days may be normal, and the symptoms gradually subside, the whole duration being usually less than a weak. Subsequent attacks, however, occur in the great majority of cases.

Suppurative appendicitis.—The onset resembles the more severe attacks of catarrhal appendicitis, but both the local and the general symptoms are apt to be more acute. The disease may follow one of three courses, according as the termination is a localized plastic peritonitis, a peritoneal abscess, or general peritonitis.

1. With localized plastic peritonitis.—The symptoms in this variety usually last about ten days. They are severe only for the first two or three days, and then gradually subside. There is present, in addition to the symptoms described in the catarrhal variety, a distinct inflammatory induration in the region of the appendix. At first this is somewhat diffuse, but later it becomes more and more circumscribed, until after three or four days a small mass not much larger than an egg remains, which after another week can scarcely be felt. In such cases there is a suppurative inflammation of the wall of the appendix with localized plastic

peritonitis, or a slow perforation occurs which is immediately surrounded by an exudate of lymph protecting the general peritoneal cavity.

2. With peritoneal abscess.—In some of the cases with an acute onset there is a continuance of the high fever, pain, and tenderness, with the rapid formation of an abscess. A distinct tumour may be noticed at the end of two or three days, and pus may be found at operation as early as the third day from the onset. At other times the course in the early stage resembles that of the cases which terminate in resolution. Marked improvement takes place after four or five days of rather severe symptoms. The temperature does not, however, quite reach normal. After a variable period of quiescence, lasting from two or three days to as many weeks, the temperature gradually rises; the pain and tenderness become more severe and are felt over a larger area; the induration, which has been stationary, enlarges and becomes more prominent, and the existence of abscess is unmistakable. In a small number of the cases terminating in abscess the onset is very gradual, without any of the acute symptoms mentioned. It may be accompanied by slight pain only, retraction of the right thigh, and moderate fever. Whether the formation of the abscess is rapid or slow, the subsequent course may be the same. The sac is gradually distended with pus, which may accumulate in immense quantities; as much as five pints have been evacuated. At the present time but few abscesses are allowed to open externally, incision being commonly made before that time. The situation of the abscess depends upon the position of the appendix. It may be in the pelvis, in the lumbar region, and occasionally just below the liver. Pelvic abscess may be recognised by rectal examination. The termination in a single abscess is a favourable one, for with proper surgical treatment these cases almost invariably recover.

3. With general peritonitis.—This may occur early in the disease with a rapidly spreading inflammation of the suppurative variety terminating in perforation; or it may develop late, being caused by the rupture of an abscess into the general peritoneal cavity. It is seen more frequently with gangrenous appendicitis, with which its symptoms are described below.

Gangrenous appendicitis.—At the outset this form of appendicitis is not characterized by any distinctive symptoms. For two, three, or even four days, things may go so smoothly as to excite no apprehension, neither the general nor local symptoms indicating anything more serious than an ordinary attack of catarrhal appendicitis of moderate severity; when suddenly without warning a marked change for the worse occurs, as perforation into the general peritoneal cavity takes place. Sometimes there are no early symptoms which are recognised, the signs of perforation being the first to attract attention to the abdomen.

In the most severe cases the symptoms immediately become alarmingly worse, and death may occur within twenty-four hours. Rupture of a gangrenous appendix is usually indicated by a sudden attack of vomiting, very severe abdominal pain, followed by great prostration or even collapse. The temperature varies greatly in the different cases, and is no guide to the gravity of the condition. It may rise rapidly to 105° or 106° F., or it may be subnormal. The pulse is uniformly rapid, small, and compressible. The expression of the face is anxious and the features are drawn, and usually the forehead is covered with a cold perspiration. The abdomen soon becomes tense and tympanitic. In the most severe cases there is no reaction, and prostration deepens with the occurrence of stercoraceous vomiting, hiccough, clammy skin, collapse, and death.

In other cases, after the first shock of perforation, there is some reaction, and the usual symptoms of general septic peritonitis develop, with which the child may live for from two to five days. The temperature is not usually very high, generally averaging from 102° to 104° F.; vomiting is almost invariably present, and is of greenish material, indicating regurgitation from the small intestine into the stomach; pain and tenderness are acute and rapidly extend over all or the greater part of the abdomen. The other important symptoms are, absolute constipation, tympanites, a rapid, feeble pulse, and general prostration. There is mental dulness or apathy, and occasionally convulsions. The case usually goes on steadily from bad to worse; sometimes, after the first intense onset, there may be a lull in the symptoms for a day or two, to be followed by a recurrence of the severe pain, vomiting, and collapse. Such a course indicates that the first perforation has been followed by some limiting adhesions, which subsequently give way, causing all the symptoms of a new perforation.

When general peritonitis occurs from perforation due to ulceration its symptoms are rather less violent in their onset, less intense in their development, and slower in their progress, the usual duration being from five to fourteen days. When the peritonitis is the result of an abscess which has ruptured into the general peritoneal cavity the symptoms are like those of a sudden perforation. This accident may come as late in the disease as the second or third week.

Course and Termination.—Few diseases differ more widely in their course than does appendicitis. So often do cases apparently mild suddenly develop most severe symptoms that all such patients should be very carefully watched from the outset in order to determine what the course of the disease is likely to be.

It is hard to state in figures the relative frequency of the different terminations. Of 102 cases in children under fourteen years old, in which this was definitely known, 11 ended in resolution, 52 in abscess, and 40 in general peritonitis. These figures probably do not represent correctly the proportion of those terminating in resolution, for many such are doubtless overlooked or wrongly diagnosticated. Of the 52 cases which terminated in abscess, all but 6 were operated upon; 4 of the latter opened into the rectum with a favourable result; 1 opened externally, and 1 ruptured into the general peritonæum, causing death. From these statistics it would appear that general peritonitis is a more frequent termination in children than in adults, and this is, I think, borne out by general surgical experience.

Prognosis.—The prognosis in young children is not good; but in those over seven years old it is rather better than in adults. The results depend much upon early diagnosis and proper treatment. General peritonitis is the cause of death in about 80 per cent of the cases, pyæmia being next in frequency. Of 43 fatal cases, nearly all of them from general peritonitis, only 6 died during the first three days, 19 from the fourth to the seventh day, 13 in the second week, and 5 in the third week. Cases terminating in the formation of a single abscess usually recover when properly treated. If general peritonitis occurs, whether early or late, the chances of recovery are small; but it has occasionally followed when general peritonitis existed at the time of operation.

Diagnosis.—The diagnostic symptoms of appendicitis are a sudden onset with vomiting, sharp pain in the abdomen, and persistent acute localized tenderness in the right iliac fossa. Rigidity of any or all of the abdominal muscles is also significant. Constipation is much more frequent than diarrhœa. There is almost invariably some elevation of temperature, but not often high fever. The different forms can seldom be distinguished from each other at the outset. In some of the catarrhal cases the onset may be acute and severe; while, on the other hand, perforation or rupture may take place without any preceding characteristic symptoms. Abscesses out of the usual situation, due to an abnormal position of the appendix, often lead to mistakes in diagnosis.

Appendicitis may be confounded with colic, indigestion, and in infants with intussusception; in older children with abscesses due to psoitis. Colic is distinguished by the absence of localized tenderness and fever, by its short duration, and by the fact that the pain is generally less intense. Severe colic with fever in children over three years old should, however, always be regarded with suspicion. From acute indigestion the diagnosis of appendicitis is difficult at the onset, and it may be impossible for twenty-four hours. However, the pain of indigestion is rarely so severe while the fever is usually higher. It should be remembered that the pain in appendicitis is not always localized, nor is the tumour always in the right iliac fossa. The presence of pain, vomiting, and localized tenderness, and the greater severity of the constitutional symptoms, indicate appendicitis. I have twice known pneumonia at the right base to be mistaken for appendicitis. There was severe localized pain in the iliac fossa, which was evidently to be explained by pleurisy involving the lower intercostal nerves. Intussusception, with its pain, colic, and vomiting, may suggest appendicitis, but is very rare except in infants. Acute or subacute suppuration in the right iliac fossa is almost invariably due to appendicitis.

The leucocyte count may be of considerable assistance in differentiating appendicitis from colic, ileo-colitis, intussusception; also in distinguishing the catarrhal from the suppurative form. As between the two conditions last mentioned, it is not only the actual number of leucocytes present, but their rapid increase, which indicates the presence of suppuration. It should, however, be remembered that in some of the gravest cases the leucocytosis may be slight or there may be none at all. On the whole, while the presence of marked leucocytosis—i. e., above 20,000—may be of considerable assistance in the diagnosis, no inference can be drawn from a normal count or a slight leucocytosis.

Whenever, in children over two years old, there are symptoms pointing to acute peritonitis, no matter what their combination or variety, appendicitis should always be suspected.

Treatment.—Absolute rest in bed can not be too strongly insisted upon whenever appendicitis has been diagnosticated or is suspected, no matter how mild the attack may appear. As a local application the ice-bag is to be preferred. Morphine often does harm by obscuring important symptoms and increasing constipation. The colon should be kept empty by the daily use of enemata. After a thorough clearing of the bowels in the beginning, preferably by a saline, cathartics are to be avoided.

Appendicitis is a surgical disease, and surgical advice should be sought early. In deciding as to the time of operative interference, it should be remembered that the natural course of the disease in children is less likely to be favourable than in older patients. In general the statement may be made, that the younger the child the less the local and constitutional resistance, the more rapid the progress, and the greater the chances that the general peritonæum will be invaded.

If the symptoms are sufficiently clear to admit of a positive diagnosis being made early, while the disease is still limited to the appendix and before rupture has taken place, immediate operation should be urged. At this time the operation is simple, practically free from danger, and prompt recovery is almost certain to follow. No doubt some such cases might recover without it; but against this argument should be placed the great risks which are assumed when the disease is allowed to follow its natural course, and the probability, amounting almost to certainty, of subsequent attacks.

If the patient is not seen early, or if a positive diagnosis has not been possible until considerable local inflammation has developed, the decision as to operation should depend upon the course of the symptoms in the individual case. If the disease is progressing favourably—i. e., the inflammatory area not increasing and the constitutional symptoms steadily subsiding—one may often wait, with advantage, for abscess to form before interfering. If suppuration does not occur and the case ends in resolution, operation may be deferred until the acute attack is over. It should, however, be remembered that the gravest symptoms not infrequently develop with great suddenness in cases which, to all appearances, have been progressing favourably, and sometimes in waiting to secure a more favourable time for operation, the only favourable time has been lost. All these cases should be very closely watched, being seen every few hours, and the surgeon should stand ready to operate immediately should the inflammation take an unfavourable turn, as when symptoms point to a rapid extension of the disease or to perforation into the general peritoneal cavity.

On the whole, in very young children, the earlier the operation is done the better. The risks of waiting are great and a comparatively small proportion of the cases can be expected to terminate in resolution.

INTESTINAL WORMS.

Judging by published reports, intestinal worms are much more common in Europe than in this country. In 10,000 patients treated for medical diseases in my dispensary service, there was positive evidence of worms in but 79 cases. Of these, 9 had tapeworms, 40 roundworms, 27 threadworms, and 3 both round and threadworms. In private practice among the better classes, worms are certainly rare.

Cestodes—Tapeworms.—Cestodes are usually introduced into the body by the ingestion of some form of food containing larvæ (cysticerci). The larva of the *tænia solium* is most frequently found in pork; that of the *tænia mediocanellata* in beef; that of the *bothriocephalus latus* in fish; that of the *tænia cucumerina* inhabits dog or cat lice, being introduced into the intestinal tract accidentally by the hands.

In the intestine the larvæ develop into the mature tapeworms, usually in from three to three and a half months; after which the terminal segments becoming mature, separate, and are discharged in the fæces, sometimes singly, sometimes connected. New segments continually form next to the head as the terminal ones are cast off, so that the length of the worm is not diminished. The duration of life of the worm is estimated to be from ten to thirty years. Each mature segment is provided with both male and female sexual organs, and contains ova in great numbers. The ova escape after the rupture of the segment outside the body. They find their way into the stomach usually of herbivorous animals with their food. Here the thick shells of the ova are dissolved by the gastric juice and the embryo set free. By means of the hooklets with which it is provided, it migrates from the stomach or intestine and may be found in the muscles or in any organ of the body, even the brain and eye. When it reaches its final resting place it loses its hooks and gradually becomes transformed into a vesicle, from the inner surface of which there projects something resembling the head of the future tapeworm. In this stage it is known as the bladderworm or cysticercus. The cysticerci of the *tænia solium* are sometimes found in man, but the other varieties very rarely. For the further development of the larval form it must be taken into the stomach of man or some carnivorous animal. This occurs when pork, beef, or fish containing cysticerci is eaten. The vesicle wall is now dissolved, and the head passing into the intestine develops into the mature tapeworm. Several varieties of tænia are found in the human intestine:

Tænia Saginata or Mediocanellata—Beef Tapeworm (Fig. 78). This is the most frequent form found in children, all others being rare. Infection results from eating raw or partially cooked beef containing cysticerci. The worm is from twelve to twenty feet in length, and has a square pigmented head without hooks but provided with four suckers. The full-sized segments are from one half to three fourths of an inch long and about half as wide.

Tænia Solium—Pork Tapeworm (Fig. 79). This is a rare form in children, and comes from eating raw or partially cooked pork or sausage. It is from six to ten feet in length, the segments being nearly square. The head is about the size of a mustard seed and is pigmented. It also is provided with four suckers and a proboscis, surrounding which is a circle of about twenty-six hooks.

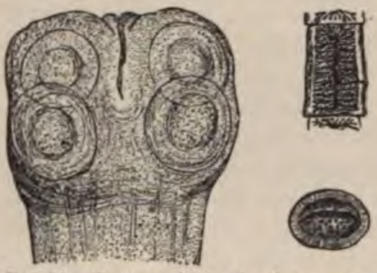

FIG. 78.—Tænia saginata; head, segment, and egg. (Jaksch.)

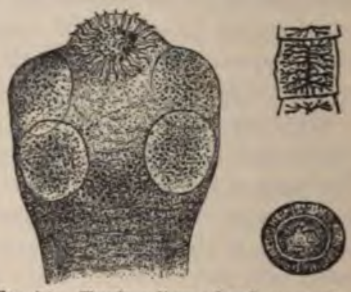

FIG. 79.—Tænia solium; head, segment, and egg. (Jaksch.)

Tænia Cucumerina or Elliptica (Fig. 80). The larvæ of this form develop in a louse found on the skin of dogs and cats. Children who play with infected animals are the ones affected, the parasite being conveyed to the mouth usually by means of the hands; it may thus be found even in young infants. Most of the tapeworms in infants are of this variety. This form of tænia is much smaller than either of the preceding varieties, the full length being only from six to twelve inches.

Bothriocephalus Latus (Fig. 81). This is a rare form except in the sea countries of northern Europe and Switzerland, where it is said to be very common. The larvæ are harboured by certain fish, through which they are introduced into the body. The full-grown worm is from twenty-five to thirty feet in length.

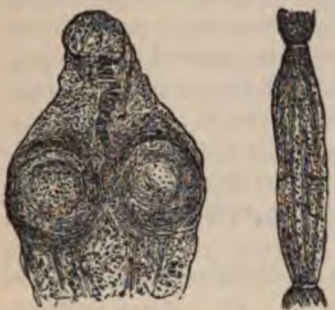

Fig. 80.—Head and segment of tænia cucumerina. (Jaksch.)

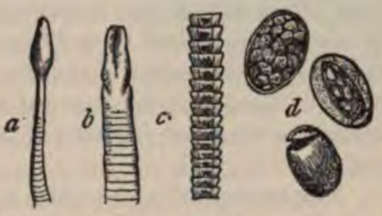

Fig. 81.—Bothriocephalus latus; *a*, *b*, front and side views of head; *c*, segments; *d*, eggs. (Jaksch.)

Tænia Nana and Tænia Flava Punctata. These are two rare varieties that have been found in children in a few instances.

Usually but a single worm is present, although as many as five or six have been found. Rarely tæniæ have been associated with roundworms and also with threadworms.

Symptoms.—The only positive evidence of tapeworm is the discharge of the separated segments, either singly or in groups. Occasionally worms pass into the stomach and are vomited. Various abdominal symptoms may be associated with worms, but most of these are very indefinite in character and are more often due to other causes. The most frequent symptoms are bad breath, various annoying sensations, colicky attacks, inordinate or capricious appetite, and diarrhœa. Usually, if the patient is in good health, no constitutional symptoms are seen. Sometimes, particularly with the bothriocephalus latus, there is a very grave degree of anæmia. Many cases are now on record, some of them in children, in which the symptoms of pernicious anæmia have been present and have disappeared after the expulsion of the tapeworm. Nervous symptoms are not so often seen as with roundworms, and will be discussed in connection with them.

Treatment.—Prophylaxis requires the cooking of meat to a sufficient degree to destroy the cysticerci. There is especial danger in eating raw pork or sausage; that from rare beef is much less. The list of drugs used for the expulsion of the worm is a long one; probably the most satisfactory is the oleoresin of male fern, which should be given in capsule, in ♏xv doses to a child of ten years, four capsules usually being administered at hourly intervals. The vermifuge should be preceded by several hours' fasting, and the bowels should be previously opened by a laxative.

The following plan of administration has been found satisfactory: A light supper of milk, and in the morning a saline laxative on rising, but no breakfast; after the saline has acted freely the capsules are to be given, one every hour, and following the last one, half an ounce of castor oil or some other active purge. The effect of the cathartic is aided by an injection. Only milk should be given that day. The fragments passed should be carefully examined to see if the head has been expelled, as the worm is very likely to be broken at the neck. If this occurs it will grow again, and in about three months segments will appear in the stools. Other drugs useful for tænia are infusion of pomegranate root, turpentine, and chloroform.

NEMATODES.—Two varieties are found in the intestinal canal, the *ascaris lumbricoides* and the *oxyuris vermicularis*.

Ascaris Lumbricoides—Roundworm.—This worm occupies the small intestine. It is much more frequently met with in children than is the tapeworm. It is exceedingly rare in infancy, but is usually seen between the third and tenth years. In over one thousand autopsies upon infants I have only once found a roundworm in the intestine.

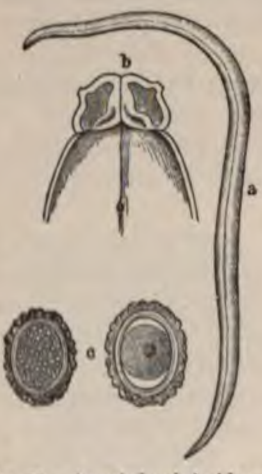

FIG. 82.—Ascaris lumbricoides; *a*, entire worm; *b*, head; *c*, eggs. (Jaksch.)

The roundworm is from five to ten inches long, the female being longer than the male. It is of a light gray colour with a slightly pinkish tint, cylindrical, and tapering toward the extremities (Fig. 82). The eggs are oval in form, about $\frac{1}{400}$ inch in diameter, and are numbered by millions. These worms rarely exist singly; usually from two to ten are present, but there may be hundreds. When very numerous they coil up and form large masses, which may cause intestinal obstruction.

The life history of the roundworm is not yet perfectly understood. Epstein cultivated outside of the body eggs taken from the stools, and found that under favourable conditions of sun and air five weeks were required for the development of the embryo. These were then fed to children. In three months the ova appeared in the stools, and after the administration of santonin many worms were discharged. From these experiments it would appear that no intermediate host is required, although this was previously supposed to be the case. It was believed that the ova were swallowed by some worm or insect, and in this form were taken into the intestinal canal with green vegetables, fruit, or drinking water.

The migration of these worms is curious, and in some instances truly remarkable. They frequently enter the stomach and are vomited. Occasionally one may appear in the nose. They have been known to pass through the Eustachian tube into the middle ear and to appear in the external meatus. Entering the larynx they have produced fatal asphyxia. It is not very rare for them to enter the common bile duct and produce jaundice. They may even enter in great numbers the smaller bile ducts and produce hepatic abscesses. They have been found in the pancreatic duct, in the vermiform appendix, and in the splenic vein. It has long been known that they would perforate an intestine which was the seat of ulceration, but well authenticated cases have been reported in which they have perforated an intestine previously healthy, setting up a fatal peritonitis. In Archambault's case they perforated the stomach. In cases of a persistent Meckel's diverticulum, worms have been discharged from an umbilical fistula. They have been found in umbilical abscesses. Considering, however, the frequency of roundworms, migrations are rare.

Symptoms.—The symptoms of roundworms are of the most indefinite kind; often there are none until the worm is discovered in the stools. It is then fair to assume that other worms are also present. The most frequent abdominal symptoms are colic, tympanites, and other symptoms of indigestion, loss of appetite, restless, disturbed sleep, grinding of the teeth at night, and picking the nose. These symptoms are much more frequently due to other causes than to worms, but when all are present the existence of worms should be suspected.

A great variety of nervous symptoms may be associated with intestinal worms. They are more often seen with lumbricoids than with either of the other varieties. The symptoms may be of the most puzzling character, and may simulate very closely those of serious organic disease. There may be chills, headache, vertigo, hallucinations, hysterical seizures, epileptiform attacks, convulsions, tetany, transient paralyses such as strabismus, and even hemiplegia and aphasia. All these have been observed in connection with intestinal worms, and from the fact that the symptoms disappeared completely after the worms were expelled there seems to be but little doubt that they were the cause of the symptoms. As in the case of the abdominal symptoms, however, intestinal worms are only one of the causes of such nervous disturbances, and certainly not the most frequent; but the possibility that they may depend upon worms should not be overlooked.

The only positive evidence of the existence of roundworms is the discharge of a worm from the body, or the discovery of the ova in the stools. A microscopic examination of the stools is a valuable means of diagnosis, and one that is too infrequently employed. When worms are present the ova may be found in great numbers. Their continued presence after the discharge of one worm, indicates that other worms remain.

Treatment.—Altogether the most efficient agent for the removal of the worms is santonin. The same plan of administration may be followed as in the case of the tapeworm—viz., to give the drug on an empty stomach, preceded by a laxative. Santonin is best given in powdered form mixed with sugar. For a child of five years three grains are usually required. This amount should be given in three doses at intervals of four hours, soon followed by a purge of calomel or castor oil.

Oxyuris Vermicularis—Pinworm—Threadworm.—The oxyuris (Fig. 83) resembles a short piece of white thread. The female is about one-third of an inch long, the male about one-half that length, but is less frequently seen. The worm tapers toward the tail. The ova are of slightly irregular size, and are considerably smaller than those of the round-worm.

The oxyuris inhabits chiefly the rectum and lower colon; less frequently it may be found as high as the cæcum. These worms have been seen in the stomach, and even in the mouth. If present in the rectum they are usually discovered by separating the folds of the anus. The number of worms is usually large. The irritation to which they give rise, causes a great production of mucus, and frequently leads to a chronic catarrh of the colon of considerable severity. The worms are imbedded in the mucus; often they form with it small balls. According to Leuckart, they are incapable of multiplying *in situ.* For development, the ova must be swallowed. The ova as well as the worms are passed in enormous numbers with the stools. They attach themselves to the folds of the skin, the hairs about the anus, and even to the genitals. The patient may, through lack of cleanliness of the parts, continually re-infect himself. After discharge from the body, the ova may be carried by flies and deposited upon fruits, vegetables, or in drinking water.

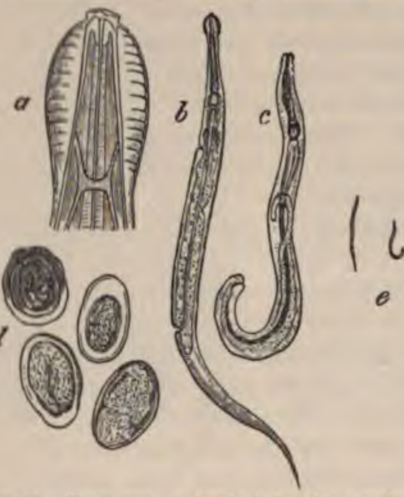

FIG. 83.—Pinworms. *a*, head; *b*, female; *c*, male; *e*, female and male, natural size; *d*, ova. (Jaksch.)

Symptoms.—The principal symptom caused by the oxyuris is itching of the anus or the genitals. This is caused by the migration of the worms from the bowel, and usually comes on at about the same hour at night, generally soon after the patient has retired. It is sometimes so intense as to be almost intolerable. It leads to frequent micturition, to incontinence of urine, in the male to balanitis, and in the female to vaginitis or vulvitis, and in both, but especially in the latter, it may be the cause of masturbation. Owing to the catarrhal colitis which is excited, there is discharged a large quantity of mucus. The irritation may lead to prolapsus ani. Nervous symptoms are not so frequently associated as with the other varieties of worms, although I have seen at least one case of chorea in which they were almost certainly the cause. They have been known to excite convulsions.

Treatment.—This is usually spoken of as a very simple matter, and no doubt in recent cases, or where the number of worms is small, this is true; but where the number is large, and considerable catarrhal inflammation of the colon is present, it is often a matter of the greatest difficulty to rid the bowel of these parasites. Cases often resist the most approved methods of treatment for months, even though carefully and thoroughly applied. The reason for this difficulty is, that the whole colon is doubtless infected, and that the upper part is very imperfectly reached by injections. While, therefore, injections are important and indeed invaluable, they can not be relied upon exclusively. The most scrupulous attention to cleanliness is an absolute necessity as the first step in the treatment of all cases. It is well to bathe the parts about the anus after each stool, and even two or three times a day, with a bichloride solution, 1 to 10,000. Itching is best controlled by the application of mercurial ointment to the folds of the anus at bedtime, this effectually preventing the escape of the worms from the bowel. The local application of cold will sometimes have the same effect. The most efficient of the injections is probably the bichloride. The colon should first be thoroughly cleansed by an injection of lukewarm water containing one teaspoonful of borax to the pint, in order to remove the mucus. When this has been discharged, half a pint of the bichloride solution mentioned should be injected high into the bowel through a catheter, and retained as long as possible. This should be repeated every second or third night. On other nights a simple saline injection may be employed. The infusion of quassia, asafœtida, aloes, and garlic are also useful.

When the worms are high in the colon, drugs by the mouth must be combined with injections. The worms must be dislodged by the use of saline cathartics, and simple bitters, especially quassia and gentian, should be given by the mouth. I have known one case, which resisted for over two years everything which had been tried, to be cured in two or three weeks by injections of a decoction of garlic, in connection with which garlic was given in large quantities by the mouth.

CHAPTER XI.

DISEASES OF THE RECTUM.

PROLAPSUS ANI.

UNDER this term are included two conditions. In the first, or partial prolapse, there is simply an eversion of the mucous membrane which protrudes beyond the sphincter. In the second, or complete prolapse, there is invagination of the rectal wall for a variable distance, usually two or three inches.

Etiology.—Prolapse is most common in children during the second and third years. Its frequency in early life is partly due to the lack of support furnished by the levator-ani muscles. It also occurs very readily when the ischio-rectal fat is scanty; it is therefore often seen in children suffering from marasmus. The exciting cause may be anything which provokes severe and prolonged straining. This may be either the tenesmus accompanying inflammation of the rectal mucous membrane or chronic constipation. It may come from phimosis or stricture of the urethra, and it is a very frequent symptom of stone in the bladder.

Symptoms.—Prolapse usually occurs during the act of defecation. It is generally easily reduced, but shows a great disposition to return with every stool. In obstinate cases the bowel comes down at other times. The appearance of the tumour varies with its size. In the slighter form there is simply a ring composed of a fold of mucous membrane surrounding the anus. In the more severe form there is a flattened, corrugated tumour, usually about the size of a small tomato (Fig. 84). The mucous membrane covering the tumour is of a deep purplish-red colour, and bleeds readily. It may be the seat of catarrhal or membranous inflammation. The diagnosis in most cases is easy, although the tumour has been confounded with polypus and intussusception.

Treatment.—In most cases reduction is easily accomplished by laying the child upon its face across the lap, and making gentle pressure upon the tumour with oiled fingers. The application of cold, either by means of ice or cold cloths, is of assistance in cases which are not at once reduced by pressure. After reduction, in the milder cases the child should be kept upon its back for at least an hour. Where the tumour tends to come down with every stool, special attention should be given at this time. If an infant, the bowels should always move while the child lies upon its back, and during defecation the buttocks should be pressed together by a nurse. Older children should use an inclined seat placed at an angle of about forty-five degrees, but should never sit upon a low chair or assume any position in which straining is easy. After defecation the patient should lie down for at least half an hour. Where there is constipation, the bowels should be kept free by means of laxatives. If there is a diarrhœa, tenesmus may be overcome by frequent sponging with ice water, or by the use of small injections of ice water and tannic acid, in the proportion of twenty grains to the ounce. In more severe cases it may be controlled by the use of suppositories of opium or cocaine. Where the bowel tends to come down frequently, this may be prevented by the use of an adhesive strap two or three inches wide, placed tightly across the buttocks. This is better in the milder cases than a T-bandage. The great majority of the cases are cured by these means in the course of a few weeks.

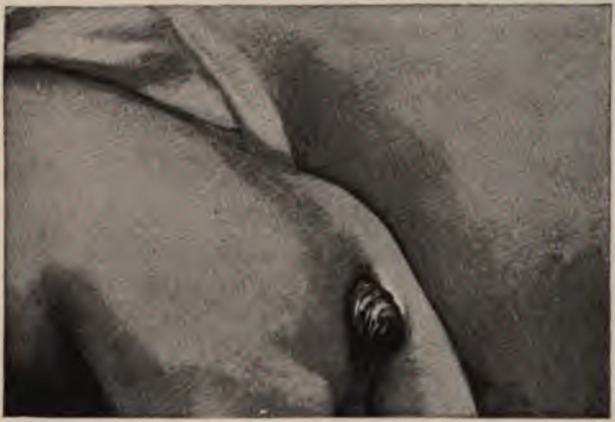

FIG. 84.—Prolapsus ani.

In the most severe cases the bowel not only protrudes during defecation, but also in the interval, and it may be down for weeks at a time. Such cases are rarely seen except in infants who have very flabby muscles, and but little adipose tissue at the floor of the pelvis. Reduction is sometimes difficult in cases where the prolapse has lasted a long time. It is often facilitated by painting the protruding part with a 4-per-cent solution of cocaine, and then dilating the sphincter by passing the finger into the central opening of the tumour. After reduction, suppositories containing from one fourth to one grain of cocaine may be inserted. They are more efficient than those containing opium or belladonna. A firm pad should be applied over the anus, held in position by a T-bandage. The tone of the levator and sphincter-ani muscles is often greatly improved by local injections of strychnia. For a child two years old $\frac{1}{100}$ grain may be used twice a day. Where these measures fail, the protruding part may be touched with the Paquelin cautery, linear markings being made at intervals of an inch. Amputation or excision is not required in children.

FISSURE OF THE ANUS.

This is not a very uncommon condition in children. The most frequent cause is the passage of a large, hard, fæcal mass. Sometimes it results from traumatism inflicted with the nozzle of a syringe while giving an enema. It may be produced by the scratching excited by pinworms. In the beginning there is a simple tear at the margin of the anus. The laceration which is produced usually heals promptly; but if the cause is repeated, healing is prevented, and there is finally produced a linear ulcer, or a true fissure, which may last for some time and be a source of great annoyance.

A fresh fissure has the appearance of any other tear at a muco-cutaneous orifice. One of longer standing has a gray base, slightly indurated edges, often discharges a small amount of pus, and bleeds a drop or two with nearly every movement of the bowels. The most constant symptom is pain, which usually occurs with the act of defecation, and continues for some time afterward. It is most severe when the fissure is just at the margin of the sphincter, and leads the child to resist every inclination to have the bowels move, so that it becomes a cause of chronic constipation, which condition again greatly aggravates the fissure. The pain is often referred to other parts in the neighbourhood.

The treatment is simple and usually efficient. It consists in cleanliness, overcoming the constipation, and touching the fissure with nitrate of silver, preferably with the solid stick. If the case is not speedily relieved by such measures, the sphincter should be stretched as in adult patients.

PROCTITIS.

Proctitis, or inflammation of the rectum, usually occurs with inflammation of the rest of the large intestine, but it may occur alone. It is to the cases in which only the rectum is involved that the term is generally applied.

The causes are for the most part local. A frequent one in infants is the use of irritating injections or suppositories, either for the relief of constipation or as a means of administering certain drugs. I have seen one obstinate case in an infant a year old, following the prolonged use of glycerin suppositories. It is sometimes caused by traumatism, especially by the careless giving of an enema. It accompanies pinworms. In certain cases it may result from direct infection through the anus. This may be from a gonorrhœal inflammation extending from the vagina or urethra, or from an infection due to other bacteria, particularly in cases of measles, scarlet fever, and diphtheria; or finally, it may be due to syphilis. The varieties of inflammation are the same as in the rest of the intestine. Proctitis may thus be catarrhal, membranous, or ulcerative.

Catarrhal Proctitis.—The pathological conditions are the same as in ordinary catarrhal inflammation of the intestinal mucous membrane. By the introduction of a speculum, or by simply everting the mucous membrane, it is seen to be reddened, swollen, and bleeds easily. There is a copious secretion of mucus. In cases of long standing there may be superficial ulceration appearing as a white or yellowish-white surface, usually just inside the sphincter.

The symptoms are chiefly local, although a condition of general irritability may result from the local condition. There is heightened reflex action, so that the stool often comes with a squirt. There is pain with defecation, and mucus is discharged, usually as a clear, jelly-like mass, and sometimes in the form of a cast, but not generally mixed with the stool. There are usually traces of blood, sometimes quite large hæmorrhages. In the most acute cases, tenesmus is present both during and after the stool. There may be prolapsus ani. The skin in the vicinity is irritated by the discharges, most frequently so in infants. If the cause is pin-worms, there may be intense itching. The duration of the disease is indefinite, depending upon the cause. It may be a few days or many months. The inflammation may extend from the rectum to neighbouring parts, leading to ischio-rectal abscess.

Membranous Proctitis.—It has been customary to describe this as a complication of diphtheria, usually occurring with diphtheria of the external genitals. As very few of these cases have been studied bacteriologically, it is impossible to say what proportion of them, if any, are to be regarded as true diphtheria. It is probable that the great majority are due to infection by streptococci. When the infection is from the intestine above, the rectum is never affected alone. When it is from below, this may be the case. The lesions are the same as in membranous inflammation occurring higher in the colon. The symptoms resemble those of the catarrhal variety, with the addition that the stools contain pieces of pseudo-membrane. This can be made out only by repeatedly washing the discharges with water. If accompanied by prolapse, the pseudomembrane may be seen. Membranous proctitis may be complicated by a membranous inflammation of the genitals or the perinæum. Although it is usually acute, it may last for weeks.

Ulcerative Proctitis.—Ulcers of the rectum may be the result of a catarrhal inflammation; these, however, are usually superficial, affecting the mucous membrane only, and in most cases heal rapidly. Sometimes they extend more deeply into the submucous or even the muscular coat. They are then chronic, often very obstinate, and may last indefinitely. Follicular ulcers of the rectum are nearly always associated with the same condition in the sigmoid flexure. These are always multiple and usually small, rarely being more than a quarter of an inch in diameter. Sometimes the small ones coalesce, producing much larger ulcers. Membranous proctitis is rarely followed by ulceration, although this is a possible result where sloughing has occurred. Single ulcers may be of tuberculous origin. Steffen reports two cases of tuberculous ulcer of the rectum in children of seven months and three years respectively. I have seen one such ulcer in a young infant, which was fully three-fourths of an inch in diameter, and was not associated with other tuberculous disease of the large intestine. Syphilitic ulcers are extremely rare in children.

The symptoms of ulcer of the rectum are mainly two—pain and hæmorrhage. The pain is of variable intensity, and may be referred to the coccyx, or to any of the neighbouring parts. The amount of bleeding may be small, the blood coming in clots, or it may be fluid and in so large a quantity as to produce general symptoms. It usually accompanies every stool. In addition the stool contains more or less pus, particularly in chronic cases. When the ulcer is low down, tenesmus is present and may be a prominent symptom. A positive diagnosis of ulcer can be made only by examination with a speculum.

Treatment.—In cases of acute catarrhal proctitis injections of some bland fluid should be employed, such as a starch-water, limewater, a mixture of oil and limewater, or a warm one-per-cent saline solution. The local cause, if one is present, should be removed. Where the stools are excessively acid, alkalies may be given by the mouth. The disordered digestion, when present, is to be treated according to its special symptoms. In the most acute cases the patient should be kept in bed. Where the tenesmus is severe, suppositories of opium or cocaine may be used. In the more chronic cases saline injections should be given, and followed by a mild astringent like tannic acid, ten grains to the ounce, or a one-per-cent solution of hamamelis. Cases associated with pinworms are especially obstinate. Here the treatment is first to be directed to the worms, and afterward to the proctitis.

In the membranous cases the same measures are to be employed, and in addition the injection of a warm boric-acid solution two or three times a day.

Cases of ulcer require the most careful treatment. In many there is but little tendency to spontaneous recovery. An examination with the speculum should be insisted upon in all cases of chronic proctitis, to make sure of the diagnosis. Rest in bed is essential to a rapid improvement. The patient should be put upon a bland diet, especially of milk, and the bowels kept freely open by the use of laxatives, and injections twice a day of a saturated boric-acid solution. Locally there should be applied a solution of nitrate of silver, one grain to the ounce, the bowel having previously been washed with tepid water. If a stronger solution than this is used, it should be neutralized after half a minute by the injection of a salt solution.

ISCHIO-RECTAL ABSCESS.

This is not a very rare condition even in infancy. Infection from the rectum, usually through the lymph channels, seems to be the most common cause, although sometimes the abscess may be traced directly to traumatism. In a single year I have seen six cases. All but two were small, circumscribed abscesses and quite superficial, apparently starting as an acute inflammation of the lymph glands of the region. They are analogous to a similar process in the lymph glands of the neck, seen in infancy. These cases healed promptly after incision. In other instances there is seen a disposition to burrow, as in adults. Only once have I met with diffuse suppuration in the ischio-rectal region, terminating in sloughing and death, and this was in an infant only three months old.

Essentially the same varieties of inflammation are seen in early life as in adults. Most of these cases recover promptly after simple incision and cleanliness, fistula being a rare sequel.

HÆMORRHOIDS.

These, fortunately, are not often seen in children, although they occur in those as young as three or four years, and in some cases may even be congenital. The principal cause is chronic constipation, rarely diarrhœa. The tumours are generally small and external, the chief symptom complained of being pain on defecation. Bleeding sometimes accompanies the pain, but the hæmorrhages are usually small. The treatment is to be directed toward the underlying cause. In most of the cases this suffices to cure the condition. I have rarely seen in a young child a case requiring operation, although neglect may make this procedure necessary.

INCONTINENCE OF FÆCES.

Inability to control the fæcal evacuations is seen in certain cases of paraplegia due to myelitis, in injury of the lumbar portion of the spinal cord, and in spina bifida. It is also seen in the coma of meningitis, and occasionally in the typhoid condition and in extreme adynamia, no matter in the course of what diseases they develop. In all these conditions incontinence of fæces is a symptom giving rise to much annoyance and needing careful attention. Uncleanliness with reference to excreta, seen in idiocy, can hardly be classed as incontinence.

Besides these familiar forms, the condition is sometimes seen from causes somewhat resembling those of incontinence of urine. The tone of the sphincter becomes so feeble that it does not resist even the slightest impulse to evacuate the rectum. The discharge may take place with but little warning, and may occur either by day or night. In some cases a local cause exists, such as stretching of the sphincter by a rectal prolapse or by impaction of fæces; more frequently, however, the causes relate to the general nervous condition of the patient. Fowler* (New York) has reported two very typical cases of this variety, and I have seen one. They are, however, very rarely met with in practice. Of the cases reported in literature, the majority have occurred in highly nervous, anæmic children. Fowler's cases were cured by the use of ergot given by the mouth and by suppository. In cases not relieved by this treatment, strychnia should be injected locally as described under Prolapsus Ani. In all cases the general condition should receive careful attention.

CHAPTER XII.

DISEASES OF THE LIVER.

Aside from the different forms of degeneration which are seen in the various infectious diseases, the liver is not often the seat of serious disease in infancy and early childhood. In later childhood nearly all the forms seen in adult life are occasionally met with, although even then they are quite rare.

Size and Position.—The weight of the liver in the newly-born child, from one hundred and seven observations of Birch-Hirschfeld, is 4·5 ounces (127 grammes), or about 4·2 per cent of the body weight. The following table gives the results of one hundred and seventy-four observations upon the liver in infancy in the autopsy room of the New York Infant Asylum:

Weight of the Liver in Infancy.

AGE.	AVERAGE.		Per cent of body weight.
	Ounces.	Grammes.	
3 months	6·3	180	3·1
6 "	7·5	212	3·0
12 "	11·0	311	3·40
2 years	14·0	397	3·37
3 "	16·0	453	3·26

In adults, according to Frerichs, the weight of the liver is about 2·5 per cent of the weight of the body.

The upper border of the liver is best made out by percussion. In the child, the upper limit of the liver dulness in the mammary line is found in the fifth intercostal space; in the axillary line, in the seventh space; posteriorly, in the ninth space. The lower border is best determined by palpation. This, as a rule, in the mammary line is found about one half an inch below the free border of the ribs. According to Steffen, the left lobe is relatively larger in the child than in the adult. The liver may be displaced downward by contraction of the chest, as in rickets, or by an accumulation of fluid in the pleural cavity. It is frequently found lower than normal in conditions of great emaciation, owing to relaxation of the abdominal walls and its ligamentous supports. Upward displacement is much less frequent, and depends usually upon ascites or abdominal tumours.

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* American Journal of Obstetrics and Diseases of Children, October, 1882.

Malformations and Malpositions.—Congenital malformations relate chiefly to the bile ducts. These have been considered in the chapter devoted to Icterus in the Newly Born (page 78).

The liver may be found upon the left side in cases of general transposition of the viscera. In fissure of the diaphragm it has been found in the thoracic cavity.

ICTERUS.

Icterus, or jaundice, occurs in children, as in adults, from two general classes of causes. The first includes those cases in which there is some obstruction to the flow of bile from the liver into the intestine, or obstructive jaundice. In the second group, in which the jaundice is classed as non-obstructive, it depends upon certain changes in the blood itself. This is seen in the physiological jaundice of the newly born, in that associated with septic conditions and as the result of certain poisons.

Obstructive jaundice from pressure upon the bile ducts is extremely rare in children. Obstruction by a roundworm entering the common duct has been recorded, but is also very rare. The principal form of obstructive jaundice seen in early life, is catarrhal. This has already been considered in connection with Gastro-duodenitis.

FUNCTIONAL DISORDERS.

Functional derangements of the liver are undoubtedly exceedingly common in childhood. They are as yet but little understood, and it is almost impossible to separate them from the other symptoms of intestinal indigestion with which they are associated. These are described in the chapter upon Chronic Intestinal Indigestion. Some of these symptoms depend upon a diminution in the quantity, or the impoverished quality of the biliary secretion. There are gray or white stools, flatulence, and other evidences of increased intestinal putrefaction. These in all probability depend upon imperfect absorption in consequence of the absence of bile, rather than upon the absence of some antiseptic property, as recent experiments seem to show that the bile is not an intestinal antiseptic. The other functional disturbances of the liver relate to its effect upon the proteid substances which undergo destructive metamorphosis in this organ. The nature of this change, and the symptoms which result from this disturbance are as yet but imperfectly understood. It is quite probable that many of the nervous functional disorders of children—for example, attacks of migraine or of cyclic vomiting—may depend upon such a cause.

NEW GROWTHS.

New growths of the liver are rare in children and are usually secondary to deposits elsewhere, most frequently in the kidney. They are generally sarcomatous. Primary sarcoma of the liver has, however, been observed, and at so early an age as to make it practically certain that the condition was a congenital one. A single example of primary adenosarcoma of the liver has fallen under my observation. This was in an infant only seven months old. In a report of this case I collected from literature ten cases of sarcoma of various types in infants under one year.* In most of the cases there is simply a slowly increasing abdominal tumour and progressive asthenia.

ACUTE YELLOW ATROPHY.

This form of hepatic disease, although rare in adults, is still more rare in children. Greves has reported a well-marked case in an infant of twenty months, and has collected seventeen other cases under ten years of age; the youngest was in an infant three months old. The symptoms and course of the disease are essentially the same as in adults.

CONGESTION OF THE LIVER.

Congestion of the liver occurs from the same causes in children as in adults. Acute congestion is not often seen. Chronic congestion is more common, and is usually secondary to general venous obstruction dependent upon congenital or acquired heart disease, atelectasis, or other pulmonary conditions, particularly chronic pleurisy, chronic interstitial pneumonia, and emphysema. Chronic congestion of the liver causes no characteristic symptoms except a moderate enlargement of the organ. In acute congestion, there may be in addition some localized pain or tenderness. The treatment is that of the primary disease.

ABSCESS OF THE LIVER—SUPPURATIVE HEPATITIS.

In 1890 Musser found but thirty-four recorded cases of abscess in children under thirteen years. Since that time a few additional cases have been reported. In the above collection, there have not been included cases of suppurative hepatitis occurring in the newly born.

As in adults, abscess of the liver may result from traumatism, or it may be secondary to suppurative pylephlebitis, which depends upon a focus of infection in the umbilical vein, or in some part of the abdomen from which the branches of the portal vein arise. Pylephlebitis may follow appendicitis (Bernard's case), it may follow typhoid fever directly (Asch's case), or be due to suppuration of the mesenteric glands or peritonitis following typhoid. In seven of the cases collected by Musser the disease was due to migration of round worms from the intestine into the hepatic ducts. Menger (Texas) has reported one case following dysentery, the only one, I think, on record in this country. In quite a number of cases no adequate cause can be found.

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* Archives of Pædiatrics, April, 1905.

In the cases occurring in pyæmia and in those associated with pylephlebitis there are usually several abscesses; in traumatic cases generally but one. If untreated, the majority of cases prove fatal either from exhaustion or from rupture into the pleura or peritonæum. In Asch's case spontaneous cure took place by rupture into the intestine.

Symptoms.—Occasionally abscess in the liver is latent, but in most of the cases the symptoms are marked and sufficiently characteristic to make the diagnosis a matter of no great difficulty. The most constant general symptoms are chills, which may be single, but are usually repeated; fever, which is commonly of the hectic variety and followed by sweating; prostration, vomiting, diarrhœa, and cachexia. Jaundice is present in less than half the cases, and is rarely intense. The liver is almost invariably sufficiently enlarged to be easily made out by palpation or by percussion; the enlargement in most cases is chiefly downward. Tumours on the surface of the liver are often present; these may be recognised as abscesses by the presence of fluctuation. Pain is quite constant, and frequently intense, but not always in the region of the liver. It may be in the epigastrium, at the umbilicus, in the lower part of the abdomen, and occasionally in the right shoulder. Tenderness over the liver is usually present. A positive diagnosis of hepatic abscess is to be made only by aspiration and the withdrawal of a fluid having the characteristics of "liver pus." Pulmonary symptoms usually exist with an abscess occupying the convexity of the right lobe. There may be cough and dyspnœa from pressure, or pleurisy from extension of the inflammation through the diaphragm, or from rupture into the pleural cavity. The usual duration of abscess of the liver after the beginning of the symptoms is from one to two months. The prognosis will depend upon the cause of the disease. The pyæmic cases are usually fatal. In Musser's collection, the proportion of recoveries was about thirty per cent. At the present time, with improved methods of treatment and earlier diagnosis, the outlook is somewhat better than this.

Treatment.—This is purely surgical. Without operation the chances of recovery are very slight. A small number of cases have been cured by aspiration, but in the vast majority only incision and drainage are to be depended upon, and, if the abscess is accessible, should be resorted to as soon as the diagnosis is established.

CIRRHOSIS.

Cirrhosis of the liver is exceedingly rare in early life, although quite a number of cases are now on record between the ages of seven and fourteen years. Sixty-five have been collected by Howard * and fifty-three by Laure and Honorat.† Nearly all the cases in these collections were between nine and fifteen years old. Cirrhosis in infancy is usually of syphilitic origin. Two-thirds of those in Howard's collection were males. The etiology in most of the cases is obscure; in over half of those reported no cause could be discovered. Fifteen per cent of Howard's cases were traced to alcoholism, eleven per cent to syphilis, and eleven per cent to tuberculosis. Laure and Honorat believe that the eruptive fevers sometimes play an important part as an etiological factor, and that at other times the cause is possibly malaria.

The anatomical features of cirrhosis in early life are essentially the same as in adults. The liver is sometimes enlarged, but usually it is smaller than normal. The connective tissue may be distributed around the lobules, along the bile ducts, in irregular patches, or in striations through the organ. Associated with this there is atrophy and fatty degeneration of the liver cells. In some of the cases reported there has been also a similar increase in the connective tissue of the spleen and kidneys.

Symptoms.—These are very much the same as in adult life. In the beginning there are the indefinite disturbances referable to the digestive organs, and the liver may be found to be slightly enlarged; later there is ascites, enlargement of the spleen, and dilatation of the abdominal veins. Ascites is a pretty constant symptom, and is generally marked. Slight icterus is often present, but a marked amount is rare. There may be hæmorrhages from the stomach, from the nose, or from other organs; in a few cases there is slight fever. The late symptoms are a small liver, marked ascites with the consequent embarrassment of respiration, cachexia, and sometimes general dropsy. Diarrhœa is a much more constant symptom than in adults. Death usually takes place from exhaustion. The course of cirrhosis in children is commonly more rapid than in adults, and the progress is steadily downward.

Treatment.—Medicinal treatment is of avail only in cases which are syphilitic. These should be put upon mercury and large doses of the iodides. The treatment in other respects is symptomatic and palliative. As largely as possible patients should be kept upon a milk diet. The ascites may require aspiration or puncture, as in adults.

AMYLOID DEGENERATION (WAXY, LARDACEOUS LIVER).

From the experiments of Krawkow, Davidsohn, and others there seems now little doubt that amyloid degeneration is produced by the prolonged action of the toxins of the staphylococcus pyogenes aureus.

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* American Journal of the Medical Sciences, 1887, p. 350.

† Revue Mensuelle des Maladies de l'Enfance, 1887, pp. 97, 159.

Amyloid degeneration of the liver is associated with similar changes in the spleen and kidneys, and sometimes in the villi of the small intestine, and is usually seen in children after long-continued suppuration in chronic bone or joint disease, empyema, tuberculosis, or syphilis.

The liver is generally very much enlarged; in extreme cases a weight of six or seven pounds may be reached. It is of a glistening, waxy appearance, very firm and hard. With a solution of iodine, a mahogany-brown reaction is obtained. The amyloid degeneration affects first the arterioles, and finally the hepatic cells.

Amyloid liver *per se* produces few symptoms. Ascites is rarely present except in cases in which the liver is very large, and jaundice does not occur. In addition to the symptoms of the original disease in the course of which the amyloid degeneration occurs, there is the peculiar waxy cachexia which is seen in no other condition, but resembles somewhat that belonging to malignant disease. The face has the appearance of alabaster, and the skin has a singular translucency. The liver may be so large as to form a tumour, sometimes nearly filling the abdominal cavity. Not infrequently it extends to the umbilicus, and even to the crest of the ilium. The surface is smooth and hard, and the edges usually rounded. There is no localized pain or tenderness. The spleen is invariably enlarged. As a result of the associated amyloid degeneration of the kidney, there may be dropsy and albuminuria. Dropsy may occur from pressure of the large liver upon the vena cava, apart from the condition of the kidney.

Amyloid changes usually take place slowly, the whole course of the disease being marked by years, the patient dying from slow asthenia, from nephritis, or from some acute intercurrent disease. As a rule, cases go on steadily from bad to worse; but sometimes, after the disease has reached a certain point, the condition is stationary for a long time.

The prognosis is always bad, although in a few cases improvement, and even cure, are stated to have occurred after the excision of the diseased joints upon which the amyloid degeneration depended. When due to syphilis, the usual anti-syphilitic remedies should be given.

FATTY LIVER.

Fatty infiltration of the liver is generally a secondary condition in early life, and causes no symptoms by which it can be positively recognised. Considerable discussion has of late arisen regarding its frequency in infants. From our records at the Babies' Hospital, Dr. Martha Wollstein has tabulated 345 consecutive autopsies in which the condition of the liver was carefully noted. The liver was fatty in 201, or 58 per cent. Of these autopsies, 63 were cases of tuberculosis, in 43 of which, or 68 per cent, the liver was fatty.

The general nutrition of the 345 infants was as follows:

Wasted..................188: liver fatty, 104, or 55 per cent—very fatty in 17.
Fairly nourished........ 80: " " 52, " 65 " " " " " 9.
Well nourished.......... 77: " " 45, " 59 " " " " " 20.

These figures coincide very closely with the observations of Freeman at the New York Foundling Hospital, and indicate that fatty liver is not, as has been so often asserted, much more frequent in wasted infants than in others. The cause of this change in the liver is as yet but little understood.

The liver is moderately enlarged, smooth, with rounded edges, of a yellowish-red or a lemon-yellow colour, and can be indented with the finger. A warm knife becomes coated with oil after cutting. Microscopically there is seen an accumulation of fat in the liver cells, usually irregularly distributed. Jaundice, ascites, and the other peculiar symptoms of hepatic disease, are absent. The liver is moderately increased in size and its functions are interfered with, but not in such a way as to be recognised by the symptoms. The treatment is that of the original disease.

HYDATIDS.

Echinococcus disease of the liver, while rare among adults in this country, is almost unknown in children. I have been able to find but two recorded cases in America. From twenty-two European cases collected by Pontou (Paris, 1867), it appears that unilocular cysts are especially frequent in young subjects. If the upper surface is affected, pulmonary symptoms, cough and dyspnœa, are usually present; if the under surface of the organ, there is pressure upon the portal vein, the vena cava, bile ducts, stomach, and intestines. This pressure may cause icterus, dilatation of the superficial abdominal veins, and sometimes ascites. The local signs are enlargement of the liver with a tumour, which is easily recognised in children because of the thin abdominal walls. The hydatid fremitus is usually obtained. By aspiration a clear fluid is withdrawn, showing under the microscope the presence of the hooklets, which establishes the diagnosis. Occasionally cure may take place by spontaneous rupture or suppuration of the cyst, but in most cases, when left to itself, the disease proves fatal. The treatment is surgical, and consists in aspiration or in incision, and the evacuation of the cyst.

BILIARY CALCULI.

Up to the age of puberty calculi are extremely rare. Still (Transactions London Pathological Society, 1899) was able to collect but twenty cases from medical literature, eleven of which occurred in newly born infants or else gave symptoms during the first month of life. The prominent symptom was intense and persistent jaundice. Nearly all died within the first month, the autopsy usually showing multiple calculi in the common duct.

The cases in older children do not differ from those in adults.

CHAPTER XIII.

DISEASES OF THE PERITONÆUM.

INFLAMMATION of the peritonæum is not very frequent in childhood, because at this time most of the causes which are operative in later life either do not exist at all or are infrequent. An analysis of 187 collected cases of peritonitis—not including those associated with appendicitis—gave the following results, which are of some interest as showing the relative frequency of the different forms in early life:

	Acute.	Chronic.	Total.
Fibrinous	22	10	32
Serous	22	15	37
Purulent	46	16	62
Tuberculous	18	38	56
Total	108	79	187

We shall consider separately acute, chronic, and tuberculous peritonitis.

ACUTE PERITONITIS.

Acute peritonitis may occur at any period of infancy or childhood. It may even exist in intra-uterine life. In the newly born, peritonitis is quite frequent. After this time it is exceedingly rare during infancy, only four cases, including all varieties, being met with in 726 consecutive autopsies in the New York Infant Asylum. After the fifth year the disease is relatively much more common. Of the 187 cases above referred to, 25 per cent occurred in the newly born, 21 per cent between one and five years, and 54 per cent between the fifth and the sixteenth years.

Etiology.—In the newly born, peritonitis is seen as one of the most frequent lesions of acute pyogenic infection (page 83). It is usually due to direct infection through the umbilical vessels. In infancy and childhood, peritonitis occurs both as a primary and secondary inflammation. The primary form is rare. It may be due to traumatism, such as falls or blows, or to surgical operations upon the abdomen; it has occurred after an injection for the cure of a congenital hydrocele. In a very small number of cases the inflammation seems to have been excited by cold or exposure, and it may follow severe burns.

The secondary form is more common. The most frequent of all causes is appendicitis, which should always be suspected in acute peritonitis occurring without definite cause. Extension of inflammation from the viscera to the peritonæum is very much less frequent in children than in adults. I have seen it but once in autopsies in acute intestinal diseases. It is also rare in typhoid fever, being noted but twice among my collected cases. It is occasionally due to abscess of the liver, ulcer of the stomach, acute intestinal obstruction from internal strangulation, intussusception, volvulus, or congenital atresia. It may extend from inflammation of the pleura. This may be in the form of empyema which burrows through the diaphragm, or, without burrowing, the infection may take place through the lymph channels. It is not very infrequently due to infection through the female genital tract, especially in gonorrhœal vulvo-vaginitis in young girls. Extension of inflammation from the male genital organs is not common. In one case at the New York Infant Asylum, fatal peritonitis in an infant started from a suppurative inflammation of the tunica vaginalis of unknown origin, the infection extending into the peritonæum through the inguinal canal. Any abscess in the neighborhood may rupture into the peritonæum and excite peritonitis. The most frequent in children are those connected with Pott's disease, perinephritis, and cellulitis of the abdominal wall.

Of the acute infectious diseases, peritonitis is most frequently seen with pneumonia and scarlet fever, occasionally with influenza. In four cases occurring in the New York Infant Asylum, the disease was twice secondary to pneumonia, in both complicated by extensive pleurisy. It may be accompanied by pericarditis, and even by meningitis.

The bacteria most frequently associated with acute peritonitis in children are: the streptococcus, especially in the newly born; the micrococcus lanceolatus (pneumococcus), in cases complicating pneumonia or empyema; and the bacterium coli commune in those following intestinal perforation. Those mentioned may be associated with other pyogenic bacteria, or less frequently the latter may occur alone.

Lesions.—In the fibrinous form we have changes similar to those occurring in inflammation of the pleura and the other serous membranes. The peritonæum is injected and lymph is thrown out in considerable quantity, usually accompanied by a small amount of serum. The process may be localized or general. It is more frequently general in the child than in the adult. The peritonæum lining the abdominal wall, as well as that covering the coils of intestine and the solid viscera, is covered by patches of yellowish-gray lymph, causing adhesions between the various viscera and often matting the intestines together. In recent cases these adhesions are soft, and easily broken down; in old cases they are quite firm, and they may result in the formation of connective-tissue bands which are the source of subsequent trouble.

In the serous form there is a moderate amount of lymph, generally less than in the plastic variety, and, in addition, an outpouring of serum in considerable quantity. This is usually clear, but may be turbid from flakes of lymph, or it may even be bloody. In most cases the amount is not very large, usually varying from half a pint to two pints. In cases going on to recovery the serum is absorbed, but there may result adhesions as in the preceding variety.

In the purulent form the products are serum, lymph, and pus. When peritonitis results from perforation it is, as a rule, purulent from the outset, and the pus is foul and stinking. The amount of pus is generally larger than in adult cases. When the disease proves fatal in a few days there is found an extensive exudation of plastic lymph, with the formation of small pockets containing pus, among the coils of intestine. Occasionally there may be larger collections of pus in the peritoneal cavity. In cases which have lasted a longer time—generally those of localized inflammation—the process results in the formation of a peritoneal abscess. This consists in a collection of pus in some part of the peritoneal cavity, the situation depending upon the cause, but it is usually in one iliac fossa or in the pelvis. The abscess is shut off from the rest of the peritoneal cavity by a thick wall of fibrin. If left alone, such abscesses may open into the rectum, vagina, bladder, pelvis of the kidney, or externally, usually at the umbilicus. After the discharge of pus the cavity may contract and fill up by granulations, and the patient recover.

Inflammations of the other serous membranes, especially the pleura, are often associated with peritonitis.

Symptoms.—The symptoms of acute peritonitis in older children, as in adults, are usually well marked and sufficiently characteristic to enable one to recognise the disease easily; but not so in the case of infants. In them the symptoms are often obscure, and the disease may be found at autopsy when not suspected during life. The onset is nearly always abrupt, with fever and vomiting. As a rule, the temperature is high—from 103° to 105° F. Vomiting may be only at the onset, but it often continues; vomited matters are usually green. Older children complain of pain, which may be localized or general; and in younger ones this is indicated by crying and fretfulness. The abdomen very soon becomes swollen and tympanitic, this being one of the most constant features of the disease. The distention is generally uniform, but it may be irregular. It is very rare in acute cases that there is a sufficient amount of fluid present to give the sensation of fluctuation. There is tenderness on pressure, and usually marked rigidity of the abdominal walls. The position assumed by the patient is generally dorsal, with the thighs flexed. The bowels are in most cases constipated, but diarrhœa is by no means rare. The abdominal distention causes dyspnœa and thoracic breathing. There may be retention of urine or frequent micturition.

The general symptoms, almost from the beginning, are those of a serious disease. The pulse is small, rapid, and compressible. The prostration is great, from the very outset. The face is pinched, the mouth is drawn, and the features indicate pain. In severe cases there may be hiccough, cold extremities, clammy perspiration, and collapse. The mind is usually clear. In infants there may be convulsions.

In the most severe forms of general peritonitis the course is short and intense, and the disease goes on rapidly from bad to worse until death occurs. In infants this is often on the third or fourth day. The most severe forms of general peritonitis in older children run the same rapid course. In other cases the course is slower, lasting a week or ten days. If the patient lives longer than this the case is more hopeful, because the process is more apt to be localized. The development of peritoneal abscess is indicated by the continuance of the temperature, which may assume a hectic type, and be accompanied by chills and sweating. There are the local signs of an abdominal tumour.

Prognosis.—Acute general peritonitis, whatever its cause, is a very serious disease in childhood. Of eighty cases of all varieties under sixteen years of age, sixty-nine per cent died. In the newly born and in infancy the disease is almost invariably fatal. In older children the outlook is not quite so hopeless, and depends upon the exciting cause. It is better in localized than in general inflammation; also in the fibrinous than in the purulent form; but the most favourable cases are those with a sero-fibrinous exudation.

Treatment.—The *medical treatment* of acute general peritonitis in children is extremely unsatisfactory, as the disease is usually fatal unless it can be relieved surgically. Opium is indicated only for the relief of the single symptom, pain; according to its severity, the size of the dose and the frequency of its repetition should be determined. On account of vomiting it is well to administer it hypodermically. The only other medical measures deserving much consideration are catharsis by salines, and saline injections. Used early, and in sufficient amount, free purgation by salines seems to produce a derivative effect upon the peritoneal inflammation, which is sometimes very marked. Either the sulphate or the citrate of magnesia may be used, often advantageously preceded by calomel. Much larger doses than in most conditions are necessary on account of the constipation which belongs to the disease, this being one reason why so little effect is sometimes seen. High saline injections are useful in aiding the elimination of poisonous products from the intestinal tract. A normal salt solution should be given at a little above the body temperature, at least one quart being employed for a single injection, to be repeated two or three times a day if the effect upon the general condition is favourable.

As a local application cold is usually to be preferred. It may be applied either by an ice-bag or by a Leiter's coil. If children rebel against the use of cold, heat must be substituted. Turpentine stupes may aid in relieving tympanites.

Feeding is always a difficult matter on account of the strong tendency to vomiting; this is due to the regurgitation from the intestine into the stomach, which in some cases is almost continuous. In such conditions I have found great benefit from washing the stomach shortly before feeding, repeating this several times each day. In this way vomiting may often be controlled and the stomach made ready for food. The diet should be peptonized milk, broth, or kumyss. As stimulants, brandy with ice, or if this is vomited, champagne may be given.

Surgical treatment.—In every clear case of acute peritonitis of doubtful origin, an early exploratory operation should be done if the child's general condition will permit. Appendicitis is often found to be the cause when least expected; besides, in most other conditions this gives the only chance for recovery. Acute perforative peritonitis in a child is usually fatal under any treatment; but immediate laparotomy should be tried. Operation is also indicated in peritoneal abscesses.

CHRONIC (NON-TUBERCULOUS) PERITONITIS.

Peritonitis may occur in fœtal life with the production of extensive adhesions, which may interfere with the development of the intestine and result in various malformations. These cases have been ascribed by Silbermann * to syphilis.

Chronic peritonitis may follow the acute form, in which there are left adhesions which slowly increase owing to the production of new connective tissue. Such cases are sometimes chronic from the beginning.

The peritoneal abscesses which follow the suppurative form may run a chronic course. Chronic localized peritonitis may occur in connection with disease of any of the organs covered by the peritonæum.

Chronic Peritonitis with Ascites.—In most cases this is chronic from the outset and independent of the causes above mentioned. By far the most frequent form of inflammation is that due to tuberculosis, and by some writers the opinion is still held that this form is always tuberculous. After the observations reported by Henoch, Vierordt, Fiedler, and others, there seems to be no longer any room for doubt regarding the existence of a chronic non-tuberculous form of peritonitis with ascites, although it must be considered a rare disease. In its pathological and clinical aspects it is to be compared to subacute or chronic pleurisy with effusion.

Etiology.—Nearly all the cases thus far reported have occurred in children over six years old. The causes are for the most part obscure. The disease has been attributed to exposure, rheumatism, and injury. In a few instances it has followed measles. It may be associated with disease of the intestines or the solid viscera of the abdomen, especially with new growths of the kidney, liver, etc.

Lesions.—The post-mortem observations thus far have been few. In the reported cases there has been found a large amount of greenish serum in the general peritoneal cavity, with a very moderate amount of fibrin and adhesions, which are sometimes few and sometimes very numerous. Chronic pleurisy may be associated.

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* Jahrbuch für Kinderh., Bd. xviii, 420.

Symptoms.—The early symptoms are of a very indefinite character, such as a decline in the general health, or dyspeptic symptoms; but often nothing whatever is noticed until the swelling of the abdomen begins. The enlargement comes on rather gradually in the course of a few weeks. Pain is slight, or wanting altogether. There may be some abdominal tenderness, but this is rarely marked. The bowels are irregular; sometimes there is diarrhœa and sometimes constipation. The abdomen is usually distended with fluid, the umbilicus protruding, and the superficial veins prominent. The enlargement is generally regular and symmetrical, and the wave of fluctuation is readily obtained. The general symptoms are very few. In some cases there is a slight evening rise of temperature of one or two degrees. There may be general weakness, loss of appetite, and moderate anæmia.

The usual course of the disease is for the fluid to remain for a time and then undergo slow absorption, the case going on to complete recovery. Occasionally relapses are seen. The results are not always so favourable, for in some instances there is no tendency to absorption of the fluid, the general health is gradually undermined, and the patients die from exhaustion or from some intercurrent disease. The diagnosis rests upon the presence of ascites, developing gradually without any signs or symptoms of disease in the heart, liver, or other organs. The points which distinguish it from tuberculous peritonitis are considered under that disease. In the cases which recover, the fact that no other signs of tuberculosis subsequently develop is an important point in diagnosis. The prognosis is in most cases favourable, but must be guarded on account of the difficulty in making a positive diagnosis from the tuberculous form. Recovery is usually complete and permanent.

Treatment.—It is important that the patient should be kept at rest, preferably confined to bed. The best results are usually obtained by the adoption of a general tonic plan of treatment. If absorption of the fluid does not begin with such means, saline diuretics should be given and the amount of fluid allowed the patient limited. When there is no tendency to absorption after a thorough trial of the above measures, and especially when the patient's general health begins to suffer, the fluid should be removed by aspiration. If it continues to accumulate after repeated aspirations, laparotomy may be performed, for in some cases this has the same beneficial effect as in tuberculous peritonitis.

TUBERCULOUS PERITONITIS.

The peritonæum is quite frequently the seat of tuberculous inflammation in early life; but not so often in infants as in older children. Of 56 collected cases, 7 were under three years of age, 26 from three to eight years, and 23 from eight to sixteen years. In 119 autopsies upon tuberculous patients, most of them under three years old, of which I have records, the peritonæum was involved in 8·5 per cent. In 105 autopsies, for the most part upon older tuberculous children, Ashby found the peritonæum involved in 36 per cent. In 883 collected autopsies upon tuberculous children of all ages, Biedert * found the peritonæum involved in 18·3 per cent. These figures do not represent the number of cases of tuberculous peritonitis, as in many of them only a few miliary tubercles were present.

It is no doubt possible for peritonitis to occur as the primary lesion of tuberculosis, but in the great majority of cases it is secondary. It may, however, appear as the most important tuberculous lesion in the body. The peritonæum may be infected directly from the intestine, the mesenteric glands, or the pleura, or from more distant parts, such as the lungs, the bronchial glands, the cervical, or other external glands. In a small number of cases there is a history of some local exciting cause, such as a fall or blow upon the abdomen. The disease may follow exposure, or occur as a sequel to one of the exanthemata.

Tuberculous peritonitis may be acute or chronic. It presents several varieties, quite distinct from one another, both in their pathological and clinical features.

1. **Miliary Tuberculosis of the Peritonæum accompanying General Tuberculosis.**—The peritonæum may be involved as one of the lesions in acute or subacute general miliary tuberculosis. This is the most common form seen in infants. The lesions consist in a deposit of miliary tubercles, which are generally rather sparsely scattered over the peritonæum. The evidences of inflammation are very slight, or they may be absent altogether. These cases do not come under observation as cases of peritonitis, as there are no abdominal symptoms.

2. **Miliary Tuberculosis of the Peritonæum with Ascites.**—Although not the most common variety in children, these cases form an important group. The peritonæum is thickly sown with miliary tubercles, both discrete and in conglomerate masses. They are found in the omentum and the mesentery, upon the surface of the intestines and the solid viscera. The peritonæum shows in varying degrees the changes of acute or subacute inflammation. There is congestion, with the production of a moderate amount of fibrin and a large amount of serum. In the most acute cases the fluid is in the general peritoneal cavity. In those of longer duration it may be sacculated. The fluid is usually abundant, but not excessive. It is most commonly an olive-coloured serum, but it may be seropurulent, and even bloody. There are commonly other lesions of tuberculosis in the body, but they are less marked than those of the peritonæum.

These ascitic cases generally run an acute or subacute course, the usual duration being from one to four months. Clinically they present the symptoms of a moderate grade of peritoneal inflammation with ascites. The onset is rather gradual, with indefinite general symptoms. There is usually some fever—100° to 101·5° F. There are general weakness, prostration, and loss of flesh, but not rapid emaciation. Vomiting is not prominent, and pain and tenderness are rarely very marked. There may be nothing distinctive until distention of the abdomen is seen. This at first is due to gas, but later to fluid, which may accumulate in sufficient quantity to fill the general peritoneal cavity. The bowels are constipated, or there may be diarrhœa.

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* Jahrbuch für Kinderh., xxi, 178; see also Osler, Johns Hopkins Hospital Reports, vol. ii.

The usual course, when untreated, is for the disease to go on to a fatal termination from exhaustion. Less frequently the fluid is absorbed, and the case becomes one of the fibrous type, with a tendency to relapses; rarely it is followed by the ulcerative form.

3. **The Fibrous Form.**—This, in its general characters, may be compared to the fibroid form of pulmonary tuberculosis. There is a tuberculous inflammation, the products of which have undergone transformation into fibrous tissue. This may in a certain sense be regarded as a method of cure. The essential feature of the lesion in these cases is the production of extensive organized adhesions between the intestinal coils, and between the intestines and the abdominal walls. The intestines may be compressed against the spine by bands. Ascites may be present, but it is frequently absent altogether. If there is fluid, it may be in the general peritoneal cavity, or it may be sacculated. The fluid may consist either of serum or of sero-pus. There is no tendency to caseation or breaking down.

Clinically these cases are distinguished by their slow, irregular course. They are the most chronic of all the forms. The disease may be chronic from the outset, or it may follow the variety previously mentioned. The onset is generally insidious; fever is slight, or entirely absent. There is rarely vomiting. The bowels may be constipated or loose. For a long time the general health may remain good. The only characteristic symptom is the enlargement of the abdomen. In the early part of the disease this is chiefly from the tympanites, but later it may depend wholly or in part upon an accumulation of fluid. Ascites usually develops very slowly, but may be abundant. The adhesions of the intestines may give rise to irregularities in the outline of the abdomen. Ascites may be present for a time and then disappear spontaneously, and the general health may so improve that the patient is considered quite well. There may even be a permanent cure. In other cases, after symptoms have been absent for some time, relapses occur, and more fluid is poured out. In addition to these symptoms, others are present depending upon the mechanical effects of pressure from the contracting adhesions. There may be more or less constriction of the intestine, pressure upon the vena cava, the renal or portal veins, the thoracic duct or its branches, or upon the stomach. These may give rise to dyspeptic symptoms, emaciation, œdema of the lower extremities, and albuminuria.

In some cases the disease is entirely latent, and it is discovered at autopsy when there have been either no abdominal symptoms during life, or only colicky pains of an indefinite character. The course of this form of peritonitis is slow and irregular; it generally lasts for from three to twelve months, although with intermissions and exacerbations it may extend over several years. The fatal result may be due to an acute exacerbation, to exhaustion, or to the development of tuberculosis elsewhere.

4. **The Ulcerative Form.**—This is an inflammation associated with large tuberculous deposits which go on to caseation and softening. It may be compared to ulcerative phthisis. In point of chronicity it stands midway between the two preceding varieties. It is one of the most frequent forms seen in children, and, while it may be localized, it is usually general.

There is commonly a very abundant fibrinous exudate, matting the coils of intestine together and causing them to adhere to the solid viscera and to the abdominal walls. In this exudate there are seen tuberculous deposits consisting of small, yellow nodules and larger caseous masses, often broken down at the centre. These caseous deposits are also found in the mesentery and in the omentum, which may be very greatly thickened. Pockets are formed by the adhesions which sometimes contain clear serum, but more frequently pus or a brownish fluid. The tuberculous deposits are found upon the peritoneal surface of the intestine, and infiltrate the intestinal walls, often leading to perforation, and sometimes to fistulous communications between adherent intestinal coils. There may also be tuberculous infiltration of the abdominal walls, accompanied by cellulitis, resulting in abscesses, which may open externally, usually in the neighbourhood of the umbilicus.

The ulcerative form may succeed either the miliary or fibrous form, or the inflammation may be of this type from the outset. Tuberculous lesions are always found in the other organs, especially in the lungs, where they are usually advanced.

Clinically the ulcerative cases are characterized by well-marked constitutional symptoms, which are due partly to the peritonitis and partly to the general tuberculosis. Fever is regularly present, the temperature usually ranging from 99° to 102° F. Sometimes it assumes a distinctly hectic type. There is progressive emaciation, anæmia, prostration, and sweating. Diarrhœa is frequent and the intestinal discharges may at times be bloody. The abdomen is large, but not so much distended as in some of the other forms; the superficial veins are often prominent. It is rare that ascites can be made out by percussion, although fluid can often be found by puncture. Areas of dulness and tympanitic resonance are irregularly distributed. Nodular masses from one to two inches in diameter may be felt anywhere in the abdomen. The epigastric and umbilical regions may be occupied by a smooth, hard tumour—the thickened omentum—which may resemble the liver. There may be the signs of phlegmonous inflammation of the abdominal wall in the neighbourhood of the umbilicus, and even an abscess, which, after opening, may leave a fistulous communication with the peritonæum. There are usually some signs of disease in the lungs, and the pulmonary symptoms may mask those of the abdomen. The course of the disease is steadily progressive, the usual duration being two to six months. Death results from the pulmonary disease, from tuberculous meningitis, from exhaustion, and occasionally it is due to accidents associated with perforation.

5. **Peritonitis associated with Tuberculosis of the Mesenteric Lymph Nodes.**—These nodes may be tuberculous in any of the preceding varieties. In certain cases this is the principal lesion, and it is accompanied by localized peritonitis, which results in the formation of a large, irregular, nodular mass lying close against the spine. It is usually associated with tuberculous ulcers of the intestine. There may be no symptoms except those depending upon the pressure of the glandular masses upon the great vessels. This may lead to œdema of the lower extremities or to thrombosis of the vena cava, and may give rise to an abdominal tumour. There may be diarrhœa due to the intestinal lesions.

Diagnosis of Tuberculous Peritonitis.—In children, chronic ascites with fever usually means tuberculous peritonitis. If the abdominal effusion is sacculated instead of diffuse, the probabilities of peritonitis are much increased. If there are added the physical signs and symptoms of disease of the lungs, the diagnosis is almost certain. Cirrhosis of the liver is much more chronic in its course, and is very rare previous to the ninth year, being almost unknown in infancy and early childhood. In it there is often a history of syphilis, and jaundice may be present. If ascites is absent, tuberculosis of the peritonæum may be suspected if there are irregular nodules or tumours in various parts of the abdomen, with tenderness, emaciation, moderate pain, and persistent fever. Chronic abscess in the neighbourhood of the umbilicus is always suspicious. The ulcerative form is generally accompanied by evidences of tuberculous disease in the lungs and other organs, and is easily recognised. The fibroid form may be suspected if, with tuberculosis of other organs, there are irregular colicky pains and abdominal tenderness. From the abdominal symptoms alone it can not be recognised unless there is ascites. In all doubtful cases an exploratory incision should be made.

Between tuberculous and non-tuberculous chronic peritonitis a diagnosis is at times impossible. If there is a good family history; if there are no signs of tuberculosis in the lungs or elsewhere; if abdominal tenderness is slight or absent; if there are no nodular tumours; if fever and marked emaciation are wanting; and if the amount of fluid is excessive, the probabilities are in favour of a simple inflammation. There are, however, some cases in which the diagnosis can be made only by an exploratory incision, and sometimes not even then without an examination of the fibrous nodules by the microscope or by inoculation experiments. In doubtful cases the chances are always much in favour of tuberculous inflammation on account of its greater frequency.

Prognosis.—Cases of the ulcerative type are hopeless. In the ascitic and fibrous forms the prognosis is better; a certain number recover under medical treatment, others are cured by operation. Exactly in what proportion the cure is permanent, it is at present impossible to say, for most of the reported cases were not under observation long enough to make it certain that relapses did not occur.

Treatment.—The general treatment of tuberculous peritonitis is the same as that of tuberculosis in other parts of the body. The essentials are, rest in the recumbent position, a climate mild enough to permit the patient to remain out of doors the greater part of the time, and very careful attention to feeding with the purpose of improving the general nutrition. Under this treatment a very considerable number of patients recover. Such a termination is more likely if the diagnosis has been made early and if the disease is limited to the peritonæum. Specific drugs play but a small part in the treatment of these cases.

In cases not progressing favourably under general medical treatment, the question of operation must be considered. By means of laparotomy very many cases have been cured completely. The most favourable cases for operation are those of the ascitic variety. Aldibert, in his monograph, gives the indications and contra-indications for operation as follows: Laparotomy is indicated in all forms accompanied by ascites, although in acute cases it may be only palliative; in suppurative forms which are diffuse, or with a unilocular cyst; in all cases of intestinal obstruction in the course of tuberculous peritonitis; and in all cases of doubtful diagnosis. Operation is contra-indicated in the fibrous form not attended by pain, this usually tending to spontaneous recovery; in the dry ulcerative form, except at the outset; in the suppurative form with multilocular cysts. The existence of other foci of tuberculosis does not contra-indicate operation except when these are chiefly intestinal, or when there is general tuberculosis with extensive and rapidly progressing lesions.

Aldibert has collected statistics of fifty-two operations, with seven deaths and forty-five recoveries. Nine patients were reported well one year after operation. It is possible that among these cases some of simple inflammation were included; of eighteen cases, however, in which the diagnosis of tuberculosis was established by the microscope or inoculation experiments, all recovered, and six were well one year after operation. Why opening the abdomen and draining or washing out the peritoneal cavity should have such an influence in arresting the disease, has not yet been satisfactorily explained. For the surgical aspect of the treatment the reader should consult works upon surgery.

ASCITES.

Ascites consists in an accumulation of fluid, usually clear serum, in the general peritoneal cavity. It is a symptom of the various forms of peritonitis, especially the chronic varieties described in the preceding pages. It may be due also to portal obstruction from cirrhosis of the liver, or pressure upon the portal vein by peritoneal adhesions or large lymphatic glands. It is occasionally seen in all forms of abdominal tumours. Ascites may occur in general dropsy from cardiac disease, chronic pleurisy, or interstitial pneumonia, or from any condition causing pressure upon the vena cava. It is also seen in the general dropsy of renal disease. A moderate amount of ascites is often met with in extreme anæmia or leukæmia.

Small accumulations of fluid in the peritoneal cavity are difficult of detection. Large amounts are generally easily made out. There is a uniform smooth distention of the abdomen and dilatation of the superficial veins, especially about the umbilicus. On palpation, the wave of fluctuation can be obtained by placing one hand against the abdomen upon one side and giving the opposite side a sharp tap. A similar wave may be felt when there is tympanitic distention. The two are, however, readily distinguished by having an assistant make pressure with the edge of the hand along the linea alba while the test is being made; this obstructs the wave transmitted through the abdominal wall, but does not affect that through the fluid. On percussion in the sitting posture, there are dulness below and resonance above. When the patient is recumbent, there are resonance in the median line and dulness or flatness in the lateral portion of the abdomen.

The prognosis and treatment of ascites will depend upon its cause.

Chylous Ascites.—This term is applied to certain cases in which the abdominal fluid contains fat. The colour may be milky-white or light brown, and the fluid, after standing, may have at its surface a thick, creamy layer. The amount of fat present has been as high as five per cent. This condition is rare in childhood. In 1884, Letulle* could find but seven cases on record. The exact pathology is as yet not well understood. In the cases which have thus far come to autopsy there has usually been found chronic peritonitis, sometimes simple, sometimes tuberculous. The lymph vessels in some of the cases have been empty, and often no obstruction of the lymph circulation could be discovered. The fat is believed by some to be derived from fatty degeneration of the products of chronic inflammation, but this seems hardly sufficient to explain the large amount of fat sometimes found. In some of the cases it has been due to a wound of the thoracic duct. The amount of fluid is frequently very large. The prognosis is usually bad, although Pounds has reported (British Medical Journal, 1892) a case in a girl of ten years, where recovery followed laparotomy. Tuberculous peritonitis was present.

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* Revue de Médecine, 1884, No. 9.

SUBPHRENIC ABSCESS.

In the group of cases of localized peritonitis or peritoneal abscess must be included subphrenic abscess. This is a rare condition in childhood, and consists in an accumulation of pus just beneath the diaphragm and above the liver. Its cause may be either in the thorax or in the abdomen. It may complicate acute pneumonia, usually of the right lower lobe, by a direct extension of infection through the lymph channels. Sometimes it has been associated with phthisical cavities. In the abdomen it may be associated with disease of the liver. The accumulation of pus is sometimes very great, so that the diaphragm is crowded high into the thorax.

The symptoms and physical signs closely resemble those of empyema, and most of the cases have been operated upon with the belief that the surgeon was dealing with empyema. Meltzer has reported a case in a child of two years which followed pneumonia of the right base. At the operation only a few drops of pus were found in the pleural cavity; but there was discovered a pinhole opening in the diaphragm, from which the pus had escaped from a large subphrenic abscess. This was evacuated, and the patient recovered perfectly. Subphrenic abscesses may contain air; they are then likely to be mistaken for pneumothorax. These abscesses require incision and drainage like other forms of peritoneal abscess.

SECTION IV.

DISEASES OF THE RESPIRATORY SYSTEM.

CHAPTER I.

NASAL CAVITIES.

ACUTE NASAL CATARRH—CORYZA.

ALTHOUGH the symptoms of acute nasal catarrh are chiefly nasal, the principal seat of the pathological process is the rhino-pharynx.

Etiology.—Certain children are predisposed to attacks of acute nasal catarrh. This predisposition, as it sometimes extends to entire families, may be inherited; but more frequently it is acquired, and usually by the following mode of life: It is seen in children who get very little fresh air, because they are kept indoors unless the weather is perfect; who live in houses always overheated; whose sleeping rooms are kept carefully closed at night for fear they may take cold; who are for the same reason so overloaded with clothing that they can not engage in any active play without being thrown into a profuse perspiration. These conditions after a time result in a great sensitiveness of all the mucous membranes, but especially those of the nose and pharynx, which is much increased by residence in a damp, changeable climate. A small adenoid growth is very often present. Young infants and those who are rachitic, are frequent sufferers from acute nasal catarrh. Attacks are often brought on by insufficient covering for the head, by wetting the feet, by cold and exposure, especially to the raw winds of spring, accompanied by the dampness which occurs with melting snow. In susceptible children the exciting cause is often a very trivial one. A draught of cold air for a few minutes may be sufficient to excite sneezing and a nasal discharge. Atmospheric conditions are probably not the only cause of acute nasal catarrh. Micro-organisms certainly play an important part, particularly in the purulent variety. Although pyrogenic germs are always present in the nose, they do not excite an attack of acute catarrh without the vascular changes which are produced by other causes. Acute catarrh may be sporadic or epidemic; it is probably contagious, being communicated by children using the same handkerchief or occupying the same bed.

Acute nasal catarrh may be a symptom of measles, nasal diphtheria, or influenza, and it may accompany erysipelas of the face.

Symptoms.—The changes in the mucous membrane of the nose are not great, and are usually secondary to those of the rhino-pharynx, being in a large measure due to the discharge. There are redness and slight swelling. The nasal passages may be for the time quite occluded by the discharge, which is usually profuse, at first sero-mucous, and finally, if the attack is severe, muco-purulent. The symptoms may be very transient, sometimes passing away in a few hours, in which cases there is only a vaso-motor disturbance; or they may continue and develop into a true inflammation. The discharge excoriates the nostrils and the upper lip. At the onset there is usually sneezing, and in infants often a slight fever. In older children there is no rise of temperature except in the most severe cases. The obstruction to nasal respiration causes mouth-breathing, and the dryness and discomfort which result from it produce disturbed sleep, snuffling and difficulty in nursing, this being in severe cases almost impossible. The inflammation may extend to the lachrymal duct, involving the eyes in a mild conjunctivitis. There may be closure of the Eustachian tubes, causing deafness and otalgia. There may also be secondary otitis. The process often extends to the larynx and bronchi, with hoarseness and cough.

In infants, severe cases may be followed by inflammation of the lymph glands of the neck or of the retro-pharyngeal region; in either it may terminate in abscess. Less frequently these catarrhal colds are accompanied by disturbances of the digestive tract, and there is vomiting, or diarrhœa with large mucous stools.

Attacks of acute nasal catarrh are stated by some writers to cause death in young infants by interfering with respiration. I have never seen dangerous symptoms, and believe them to be exceedingly rare, if, indeed, they ever occur as a result of a simple coryza. In the mild form the attack lasts from two to three days; in the severe form from one to two weeks. Repeated attacks are frequently followed by the development of the chronic form of the disease.

Diagnosis.—It is important to distinguish between a simple acute catarrh and one due to measles, influenza, nasal diphtheria, or hereditary syphilis. Measles and influenza cause more fever and general constitutional disturbance than does simple catarrh. Nasal diphtheria is usually characterized by the appearance of membrane in the anterior nares and by patches upon the tonsils. These may be wanting, however, and there may be only a very profuse discharge tinged with blood. When persisting for two or three weeks this is always to be regarded with suspicion, even though the constitutional symptoms may be very slight. The only positive means of excluding diphtheria is by cultures. A persistent acute nasal catarrh in a young infant should aways suggest syphilis, and the patient should be carefully watched for the development of other symptoms.

Treatment.—A child suffering from acute coryza should always be kept indoors in a room with an even temperature of about 70° F., the bowels freely opened, and the amount of food somewhat reduced. The only drug which seems to have much influence upon the secretion is belladonna. A good combination is that known as the "rhinitis" tablet (camphor gr. ¼, quinine gr. ¼, fluid extract of belladonna ♏ ⅛); one half a tablet may be given every hour to a child of five years.

Useful local applications are albolene oil, oleo-stearate of zinc, alkaline sprays, such as Seiler's solution, to clear away the secretions, to be followed by a spray containing adrenalin. If the nasal obstruction causes great interference with respiration or nursing, the following may be used with a medicine dropper or spray:

℞	Adrenalin (1-1,000 sol.)	ʒ iss.;
	Acidi carbolici	gr. v;
	Acidi borici	gr. xx;
	Glycerini	♏ x;
	Aquæ destillat.	q. s. ad. ℥ ij.

M.

In all cases the upper lip and nostrils should be protected by vaseline or some simple ointment. Under no circumstances should irritating or astringent injections be given. In older children inhalations of spirits of camphor may be used with advantage.

Prophylaxis consists in solving the perplexing question, so often put to the physician, of how to prevent children from "taking cold." This is a matter of the utmost importance, and follows what has been previously said under the head of Etiology. No amount of cod-liver oil and iron will remove this tendency to catarrh so long as bad hygienic conditions continue. Sleeping rooms should be large and well ventilated, and a window should be kept open at night, except in very severe weather or during acute attacks. The temperature of the house during the day should be from 68° to 70° F., but never above this. Children should be accustomed to go out of doors unless the weather is especially bad. So firmly rooted in the minds of the laity is the idea that acute catarrhs come from cold, that the habit of coddling delicate children is always likely to be carried to an extreme.

With every delicate and "catarrhal" child one should begin in the summer by having him live in the open air as much as possible, sleeping in a room with free ventilation, with moderate covering, and continuing the same practice into the fall and early winter. If begun gradually in this way there is little difficulty in continuing throughout the winter.

The next point to be insisted on is cold sponging immediately upon rising in the morning, especially about the chest, throat, and spine (page 57). The use of chest protectors, cotton pads, and extremely thick clothing should be prohibited. Flannel underclothing should be worn upon the chest throughout the year, and upon the legs also in winter; the very lightest in summer, and only a medium weight in winter.

Frequently repeated attacks point to the presence of adenoid vegetations in the pharynx, and no measures are of much avail until these are removed.

CHRONIC NASAL CATARRH.

This term is rather loosely used to designate a chronic nasal discharge. Such a discharge is frequent both in infancy and childhood. It is a condition much neglected by the general practitioner. Patients are too often subjected to routine constitutional treatment by cod-liver oil and preparations of iodine, with the idea that such cases are "scrofulous," while local treatment is either neglected altogether, or consists only of the use of the nasal douche or syringing with a saline solution. Sometimes, when suggested by parents, local treatment is opposed by the physician in the case of young children, and a great amount of harm follows. Permanent damage to the organs of hearing, smell, speech, and respiration may result from neglecting or ignoring chronic nasal catarrh in childhood.

Chronic nasal catarrh is not to be regarded as a disease, but only as a symptom which may be due to any one of a variety of pathological conditions, each of which requires very different treatment—viz., adenoid growths of the pharynx, foreign bodies in the nose, polypi, deviation of the septum or any other congenital deformity of the nasal passages, the various forms of chronic rhinitis, and syphilis, which causes a form of rhinitis peculiar to itself.

Adenoid Growths of the Pharynx.—These are more fully discussed elsewhere. They are by far the most frequent cause of chronic nasal discharge in infants and young children, and should be the first one suspected. Every general practitioner can easily familiarize himself with the method of digital exploration of the rhino-pharynx, by which means these growths can in most cases be easily recognized. The nasal discharge accompanying adenoid growths is due to a chronic rhino-pharyngitis. Treatment is without avail unless the growths are removed. After this is done the nasal discharge usually disappears quite promptly.

Foreign Bodies in the Nose.—This condition should be suspected whenever there is an abundant muco-purulent discharge limited to one nostril. Foreign bodies in the nose are quite frequent in young children. Peas, beans, beads, or shoe buttons are most frequently lodged there. The efforts at removal on the part of the child, or even of the mother, generally result in pushing the body farther into the nose. It first sets up a mechanical irritation, accompanied by pain, swelling, sneezing, and sometimes hæmorrhage. This is followed by a catarrhal inflammation, which in the course of a few days becomes purulent, and may last indefinitely. The discharge is generally quite abundant. The symptoms point to an obstruction of one nostril, and an examination with the probe readily detects the presence of the foreign body.

In recent cases the removal of the foreign body may sometimes be accomplished by compressing the empty nostril and having the child blow his nose strongly. Often the sneezing which the foreign body excites is sufficient to remove it. Before any attempt is made to seize the body with forceps cocaine should be used, not only for the purpose of preventing pain, but in order to shrink the mucous membrane so as to allow better manipulation. In many cases chloroform is necessary. In most circumstances ordinary foreign bodies can with proper forceps be extracted without difficulty. No subsequent treatment is required, except the use of some mild antiseptic to keep the nose clean for a few days, as the inflammation quickly subsides after the removal of the cause.

Nasal Polypi.—These are among the infrequent causes of chronic nasal discharge in childhood. They are especially rare before the seventh year, but both mucous and fibrous polypi are seen. The symptoms are those of a chronic nasal catarrh with partial or complete obstruction of one or both sides. Polypi increase in size with the occurrence of every acute coryza, and are always especially troublesome in damp weather. They may be accompanied by reflex symptoms, such as cough, sneezing, and even by attacks of asthma. There may be headache, and sometimes disturbances of smell, taste, and hearing. The symptoms are of much longer duration than in the case of obstruction from a foreign body, the discharge is not so abundant, and is not purulent. The diagnosis is made only by examining the nose with the mirror and nasal speculum.

Polypi may be removed with the forceps, but this is best accomplished by the use of the wire snare. When they have been present for a long time the accompanying chronic rhinitis may require subsequent treatment.

Deviation of the nasal septum, and other congenital deformities which cause narrowing of the nasal respiratory tract, are conditions which belong to the specialist.

CHRONIC RHINITIS.

Three forms of chronic rhinitis are recognised—simple, hypertrophic, and atrophic.

Simple Chronic Rhinitis.—Simple chronic rhinitis existing alone is of rare occurrence in young children. In the cases so classed the symptoms are usually due to rhino-pharyngitis, which almost invariably depends upon an adenoid growth. The growth may be a small one, so that the symptoms of obstruction are slight or absent. A frequent complication is chronic enlargement of the cervical lymph glands.

The only constant symptom is an excessive nasal discharge, which is usually mucous, but which may be muco-purulent. It is easily removed by blowing the nose, if the child is old enough to be taught to do this. Children too young to clear the nose in this way, suffer from almost constant discomfort. The amount of discharge depends upon the severity of the case. It frequently causes irritation of the upper lip, which may be the seat of eczema or impetigo, especially in infants. The lip may be swollen and prominent. The condition of the external parts is aggravated by the constant disposition to pick the nose, which may be overcome by the application of a short anterior splint to each elbow.

Epistaxis sometimes occurs. The duration of the disease is indefinite; it may last for months or even for years, the symptoms in summer being insignificant, but returning every cold season. It may terminate in recovery, or, in children with flabby tissues and delicate constitution, it may be followed in later childhood by hypertrophic rhinitis.

Treatment.—Prophylaxis is very important. The main purpose should be to prevent attacks of acute nasal catarrh by the measures mentioned in the discussion of that disease. The general treatment should not be routine, but directed according to the indications of each case. There should be careful attention to diet and to the condition of the bowels. Iron and arsenic are needed when there is anæmia. A general tonic treatment is required in most cases. Cod-liver oil and the syrup of the iodide of iron are both useful, but are not specifics, and must be intelligently combined with other measures.

Local treatment consists first in cleanliness, and, secondly, in the use of astringents in the form of powder or solution. For cleansing, a solution which is both alkaline and antiseptic is desirable. This may be used in the form of a spray, after which the nose is cleared by blowing; or in infants, if the discharge is abundant, the only efficient method of getting rid of it is by nasal syringing. This is attended by some risk of forcing materials into the middle ear; but if carefully done, the danger seems to me to be less than that of allowing the discharge to remain. Syringing should always be done with the mouth open and the head inclined forward. All solutions are to be made with sterilized water and used warm. But little force should be employed, and it may be well to have a syringe the nozzle of which does not completely fill the nostril. Either Dobell's or Seiler's solution may be employed, diluted with an equal amount of water. As a spray the following may be used:

℞ Listerine*	℥ ss.
Sodii bicarb.,	
Sodii biborat	āā ʒ ss.
Aquæ	℥ iv.

If this is to be used with a syringe, twice as much water should be added. Ordinarily, the nose should be cleansed thoroughly twice a day, more frequently in very severe cases. Once a day, after the nose has been cleansed, an astringent solution or powder should be applied. One of the best solutions is sulpho-carbolate of zinc (gr. v to water ℥ j). This may be used as a spray, or, better, dropped into the nostril with a medicine dropper, the head being held far back. A good powder is a combination of salicylic acid gr. iij, tannic acid gr. xxx, and stearate of zinc ʒj, which may be used with an insufflator once daily.

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* Listerine is a combination containing the essential oils of thyme, eucalyptus, baptisia, gaultheria, and mentha arvensis.

Hypertrophic Rhinitis.—This is a chronic inflammation of the nasal mucous membrane, accompanied by a marked hypertrophy of all its normal structures, particularly its blood-vessels. The parts chiefly affected are those covering the inferior turbinated bones. The mucous membrane and submucous tissue are so thickened and relaxed that they may greatly encroach upon the nasal respiratory space, and when these venous sinuses are filled with blood, they may entirely occlude the passage. There is usually associated with this condition some degree of hypertrophy of the adenoid tissue of the pharyngeal vault.

In young children hypertrophic rhinitis is a very infrequent disease, if, indeed, it ever occurs. It is fairly common in moderate degree in older children, although its severe forms are rare. It usually follows repeated attacks of acute nasal catarrh in children of a lymphatic diathesis. A frequent local cause is a deflected nasal septum.

The *symptoms* are those of nasal catarrh with bilateral nasal stenosis. The discharge is usually abundant, thick, and tenacious, being increased by dust and dampness. All the symptoms of nasal obstruction are present in varying intensity—the "wooden" voice, mouth-breathing, disturbed sleep, etc. There may be reflex cough, catarrh of the larynx or bronchi, accompanied by muscular or vaso-motor spasm, giving rise to spasmodic croup or asthma. Rhinoscopic examination shows the large pendulous masses of mucous membrane, usually red and irregular, more or less completely blocking the nasal passage. It is only by this examination that the disease is differentiated from adenoids of the pharynx, with which, however, it is frequently associated. In infants and young children the adenoid growth is much the more frequent, and throughout childhood generally the more important factor in producing these symptoms.

The *treatment* of these cases falls largely to the specialist, although very much can be done by the general practitioner if he will learn to use intelligently a few remedial agents. Constitutional treatment is indicated as in simple rhinitis, but if employed alone it accomplishes little or nothing. The purpose of local treatment is the reduction of the hypertrophied tissue by cauterization under cocaine anæsthesia, by glacial-acetic or chromic acid, or by the galvano-cautery. Each has its advantages and its advocates. If the hypertrophied tissue forms pendulous tumours, it may be removed by the wire snare. Both nostrils should not be operated upon at the same time. In most cases cauterization must be repeated several times at intervals of a few weeks. In the meantime one of the cleansing solutions mentioned on page 56 may be employed.

The following formula of Lefferts is an excellent one for a spray to be used in this condition:

℞ Iodi	gr. iv
Potass. iodidi	gr. x
Zinci iodidi,	
Zinci sulpho-carbolat	āā gr. xx
Listerine	℥j
Aquæ	℥iv

To be used as a spray once daily.

Atrophic Rhinitis (*Fetid Catarrh*).—This is rare in young children, and only occasionally seen in those over twelve years old. It is characterized by the formation of crusts in the nose, which decompose and produce a horribly fetid odour. By some writers the term *ozæna* is applied to this disease, but usually this term is limited to rhinitis associated with disease of the bones. Atrophic rhinitis has been regarded by some as the late stage of the hypertrophic form. This view, however, is strongly combatted by Bosworth, who considers it the result of a purulent form of acute rhinitis. The changes consist in an atrophy of the mucous membrane and the destruction of many of the secreting glands. The nasal fossæ are large and roomy. The voice is not affected, but the sense of smell may be much impaired. There are no symptoms of obstruction. The discharge is scanty, and tends to accumulate between the bones, forming large crusts, which are expelled with difficulty by blowing the nose.

In the severe cases the *treatment* is only palliative, yet this is of the utmost importance for the comfort of the patient and those about him. The object of treatment is to prevent as much as possible the formation of crusts by the frequent use of an oil spray, such as liquid albolene, in order to coat the dry mucous membrane. For the removal of crusts they must first be macerated by a prolonged nasal douche as hot as can be borne. This should be thoroughly used morning and evening as a part of the patient's toilet. In employing the douche, a bag containing from one to two pints should be suspended a few inches above the patient's head. One of the alkaline and antiseptic fluids mentioned on page 56 may be added to the douche. The head should be slightly inclined forward and the mouth kept open during the douche. The mechanical removal of the crusts may be necessary if they are large, hard, and impacted. Benefit may be derived in some cases from the daily use of a stimulating spray containing ten grains of menthol to one ounce of liquid albolene. One of the very best deodorizers for general use is listerine, which, diluted with two or three parts of water, may be employed as a spray several times a day, in addition to the other measures mentioned.

Syphilitic Rhinitis.—Rhinitis is seen both in early and late hereditary syphilis. Coryza, or snuffles, is one of its earliest and most constant symptoms. It usually begins between the third and sixth weeks of life, rarely after the third month. The pathological condition is a subacute catarrhal rhinitis, sometimes with the formation of superficial ulcers or mucous patches. The disease is attended by a profuse nasal discharge of sero-mucus or muco-pus, occasionally tinged with blood. It may continue from a few weeks to two or three months. It usually requires only constitutional treatment, and protection of the nostrils and lips by the use of the ointment of the yellow oxide of mercury diluted with four parts of vaseline. This may be introduced with the finger or brush for some distance into the nostrils. When the discharge is very abundant, any one of the cleansing solutions previously mentioned may be used as a spray.

The rhinitis of late hereditary syphilis is a very different pathological condition. There are here gummatous deposits which break down, and form ulcers of the mucous membrane and deeper tissues. There is also periostitis, with extension of the disease to the cartilages and bones of the nasal fossæ, particularly of the septum. There may be perforation of the triangular cartilage, necrosis of the vomer or nasal bones, perforation of the hard or soft palate, and at times extensive ulceration of the alæ nasi and the face. This may be followed by cicatrization, causing stenosis of the nostril. These lesions in the nose are generally accompanied by deep ulceration of the pharynx and soft palate. They usually occur in children who have presented the early symptoms of hereditary syphilis, but are occasionally seen when no such history can be obtained. Such was the case in a patient recently under observation in the Babies' Hospital, who had perforation of the nasal septum and of the floor of the nasal fossæ, causing a free communication with the mouth. These are cases of true ozæna. The odour from the discharge is at times almost intolerable. When neglected, these cases go on from bad to worse, and may continue for years, producing unsightly deformities.

The *treatment* is, to bring the patient fully under the influence of mercury, first by means of the mercurial ointment or by small doses of calomel—i. e., one-tenth grain four or five times a day. Later the biniodide or the bichloride should be substituted, and iodide of potassium given in doses of ten to twenty grains three times a day. Tonics are needed in most cases, as the general health is frequently undermined and the patients are usually anæmic.

Locally there may be used a spray of one of the cleansing solutions already mentioned, or black wash, or a solution of bichloride of mercury, 1 to 10,000. For purposes of deodorization, listerine is one of the best remedies. Although improvement may take place quite promptly, the results of treatment are often unsatisfactory, as the disease has usually progressed so far before treatment is begun that some deformity of the nose results, usually a sinking in of the bridge and flattening of the alæ, giving rise to the so-called "saddle-back" deformity.

MEMBRANOUS RHINITIS.

The results of bacteriological examinations have shown that these cases, whose etiology was formerly the subject of considerable controversy, are nearly always due to the Klebs-Loeffler bacillus, and hence are to be regarded as true nasal diphtheria. It has been difficult, from clinical features alone, to establish this relationship, as the disease differs in several important particulars from diphtheria of the pharynx and rhino-pharynx—viz., its prolonged course, the absence of glandular enlargements, and the presence of very mild constitutional symptoms, which are sometimes altogether wanting. These peculiarities are due to the very slight absorption which takes place from the nose, which is in striking contrast with that from the rhino-pharynx. The importance of recognising such cases as true diphtheria can not be overestimated, as they have often been the means of spreading infection in schools and institutions before their true nature was determined. The possibility of membranous inflammation of the nose arising from other micro-organisms than the diphtheria bacillus is not to be denied, but such cases are extremely rare.

The most striking clinical feature of primary nasal diphtheria is a nasal discharge of serum or sero-mucus, frequently streaked with blood. It is sometimes very abundant, at other times slight. There are also the symptoms of moderate nasal obstruction. The false membrane can in most cases be seen in the anterior nares as a gray or whitish exudation. It may cover the whole inner surface of the nose. It often remains for two or three weeks, when it may loosen and come away *en masse*, sometimes forming an entire cast of the nose. After forcible removal it may reform. The disease in very many cases remains limited to the nose, but it may at any time extend to the rhino-pharynx or to the larynx. When such an extension takes place it is accompanied by an increase in the constitutional symptoms, glandular swellings, etc. A positive diagnosis can be made only by means of cultures.

In addition to the use of antitoxin, the nose in these cases should be syringed frequently with a warm saturated solution of boric acid, or bichloride of mercury, 1 to 10,000, with 5 per cent of glycerin. Such cases must be isolated, like ordinary cases of diphtheria.

EPISTAXIS.

The hæmorrhage may come from any part of the nasal fossa, but it is generally from the anterior nares, and most frequently from the vessels of the septum. Epistaxis is a rare symptom in the hæmorrhages of the newly born, and when present indicates syphilis. It is infrequent throughout infancy, but in childhood it is quite common, occurring in boys more frequently than in girls. In the latter it is especially common about the time of puberty. Children who are kept much indoors in overheated apartments, and who have susceptible mucous membranes and flabby tissues, are particularly prone to it. The exciting cause may be a local one, like a fall or blow; it may be due to picking the nose, or to any kind of mechanical irritation; it may be associated with nasal catarrh; and it is often caused by a small ulcer upon the septum. An attack may be brought on by mental or physical excitement. It occurs as an occasional, often an early symptom, in typhoid or malarial fever, in measles, or during severe paroxysms of pertussis. It is seen in the hæmorrhagic form of all the eruptive fevers, in certain cases of diphtheria, most commonly late in the disease, in hæmophilia and scorbutus, in grave anæmia, leukæmia, and in diseases of the heart and blood-vessels.

Symptoms.—Epistaxis is frequently preceded by a sense of fulness or pain in the head, which is relieved by the bleeding. The blood is usually from one nostril, and comes slowly by drops. The amount lost is generally small, but it may be large enough, when repeated, to produce a serious grade of anæmia even in strong children, and the hæmorrhage may prove fatal. Epistaxis may be overlooked if the blood finds its way into the pharynx and is swallowed. In most of the cases the hæmorrhage ceases spontaneously in from ten to twenty minutes, recurring at longer or shorter intervals, according to the nature of the cause. Hæmorrhage from adenoid growths of the pharynx may closely resemble that from the nose, but otherwise there can rarely be any difficulty in recognising epistaxis. In doubtful cases an inspection of the pharynx reveals the presence of blood-clots.

Prognosis.—This depends upon the cause. In the great majority of the so-called idiopathic cases epistaxis is not serious. Occurring early in the course of the infectious diseases, it does not ordinarily affect the prognosis unless it is very severe. When it occurs late, however, it is always a bad sign, and particularly so in diphtheria. It may be serious in any of the hæmorrhagic diseases or in diseases of the blood, where it is not infrequently a cause of death.

Treatment.—To remove the predisposition, a child should receive general tonic treatment, especially plenty of outdoor exercise, and every means should be taken, by the use of cold baths, friction, and proper food, to tone up the vascular system.

An efficient means of arresting the hæmorrhage is compression of the nose between the thumb and finger. This may be combined with the application of ice over the nose, and sometimes small pieces of ice may be introduced into the nostrils. The application of cold to the back of the neck or its use in the mouth may be of service by exciting reflex contraction of the capillary vessels. All tight clothing or bands about the neck should be loosened, and the patient kept quiet in the sitting posture. After the hæmorrhage has ceased the child should not blow his nose for some time. The supra-renal extract in solution is one of the most efficient local means of checking the bleeding. Another valuable remedy is the peroxide of hydrogen, used full strength. If bleeding continues in spite of all the above measures, the anterior nares should be plugged with styptic cotton, and if this does not control it, the posterior nares should be plugged. Usually very little effect is seen from drugs given internally, although in frequently recurring hæmorrhages where no local cause can be discovered ergot should be given a trial in full doses.

In severe cases of nasal hæmorrhage recurring at short intervals without any apparent cause, ulcer of the septum should be suspected, and, if present, should be touched with chromic acid.

CHAPTER II.

DISEASES OF THE LARYNX.

THE characteristic feature of laryngeal disease in infants and young children is the association of muscular spasm with all forms of inflammation. Often it is the laryngeal spasm, rather than the inflammation, which gives rise to the principal symptoms. This spasm is only one expression of the great reflex irritability of young children.

CATARRHAL SPASM OF THE LARYNX.

Synonyms: Spasmodic laryngitis, spasmodic croup, catarrhal croup (sometimes improperly called laryngismus stridulus).

The term *catarrhal spasm,* first suggested, I think, by Goodhart, is fairly descriptive of this disease, which is characterized by a very mild degree of catarrhal inflammation associated with marked laryngeal spasm.

Etiology.—It is not often seen during the first six months, but is frequent from this time up to the third year. After five years it is rare. It occurs in children who are well nourished, as well as in those who are cachectic. Certain children have a predisposition to such attacks; those who have had one attack are likely to have others. Heredity seems to have some influence in producing this susceptibility. Catarrhal spasm of the larynx is most frequently associated with enlarged tonsils and adenoid growths of the pharynx, sometimes with elongated uvula. The exciting cause may be exposure to cold or an attack of indigestion.

Lesions.—The catarrhal inflammation of the larynx affects chiefly the parts above the cords; there is congestion and dryness, and later increased secretion of mucus. To this there is added a spasm of the muscles of the larynx, especially the adductors. There is no submucous infiltration, and no tendency to œdema glottidis.

Symptoms.—The attack may be preceded for several hours by slight hoarseness, or by a nasal discharge. During the day the child may appear perfectly well. Usually there is heard during the evening a hollow, barking cough, at first infrequent and not severe. About midnight this is apt to increase in severity, and there is now difficulty in breathing. As soon as this becomes marked the child wakes, and presents the characteristic symptoms of an attack. In the mildest cases the dyspnœa is not sufficient to waken the child. In severe cases there is marked dyspnœa, especially on inspiration, and a loud stridor as the air is drawn through the narrowed opening of the glottis. This may often be heard in an adjoining room. There is seen on inspiration deep recession of the suprasternal fossa, the supraclavicular spaces, and the epigastrium; also depression of the intercostal spaces, and even of the walls of the chest. The terror of the child or any excitement increases the spasm and aggravates the dyspnœa. The distress is very great; the breathing usually slow and laboured; the voice hoarse, but rarely lost; the cough stridulous, hoarse, and metallic; the pulse rapid; the temperature normal or slightly elevated, rarely over 101° F. The child sits up and struggles for breath, its forehead covered with perspiration. There may be slight lividity of the finger-tips and of the lips, and sometimes considerable prostration. In the course of three or four hours the attack slowly wears away and the child falls asleep. During the following day, aside from slight hoarseness and occasional cough, the child is apparently well. Most of the cases are not so severe as this; there are the croupy cough, hoarseness, and general discomfort, but not marked dyspnœa. On the second night there is a repetition of the experience of the first, usually quite as severe unless affected by treatment; and on the third day a remission similar to that of the day previous. On the third night the attack, if it occurs at all, is generally a mild one. Slight hoarseness persists for several days, but otherwise the child is apparently well. Many children have such attacks every few weeks in the course of the cold season, the slightest exposure or an indiscretion in diet being sufficient to induce one.

Prognosis.—This is good, the disease never, I think, proving fatal, although nothing is more alarming, at least to parents, than to witness for the first time one of these severe attacks of catarrhal croup.

Diagnosis.—Catarrhal spasm may be confounded with laryngismus stridulus and with membranous croup. Laryngismus stridulus is a rare disease, and occurs only in infancy. In it we have not simply stridulous breathing, but periods of complete cessation of respiration. These may be repeated many times during the day, and may continue for weeks, being often complicated by carpo-pedal spasm, sometimes by general convulsions.

From membranous laryngitis, catarrhal spasm is distinguished by its sudden onset, the mildness of the symptoms of inflammation, the spasmodic character of the dyspnœa, and the daily remissions. The history of previous attacks will often aid in diagnosis. In case of doubt, a positive diagnosis can often be made by allowing the child to inhale a little chloroform. This at once relieves dyspnœa due to spasm, while it has scarcely any effect upon that due to membrane.

Treatment.—The purpose of treatment during the attack is to produce relaxation of the laryngeal spasm. This is accomplished by the use of emetics, steam, and hot fomentations over the larynx. A favourite emetic is a tablet triturate of antimony and ipecac, gr. $\frac{1}{100}$ each. To a child of two years, one tablet may be given every ten or fifteen minutes, until free vomiting occurs; or a teaspoonful of the syrup of ipecac and fifteen drops of the wine of antimony at the same interval. When children do not vomit after two or three doses the antimony should not be repeated, as it may produce serious depression.

Emetics have a double value if the attack is due to indigestion. If there is constipation, an enema should be given. Following the free vomiting there is generally some improvement in the symptoms, but there may be a recurrence of the spasm unless other means are employed. To prevent this, antipyrine is one of the most useful drugs. Two grains may be given to a child two years old. This may be repeated in four or five hours if necessary. Quite as much relief as that obtained from the drugs mentioned is seen from the use of steam inhalations. For this purpose the child should be placed in a closed tent, and steam introduced from a croup kettle (page 60). This may be used in conjunction with other measures, and continued as long as necessary. Poultices or hot fomentations over the larynx are often useful. In one case in which severe spasm had recurred for eight successive nights in spite of everything that was tried, the child being in great distress from the dyspnœa, I performed intubation, which gave instant relief. Tracheotomy, however, would scarcely be advisable.

During the day following the first night attack, it is well to continue the antimony and ipecac in doses too small to produce vomiting—e. g., gr. $\frac{1}{100}$ each, every four hours. After 6 P. M. the doses should be doubled, and at bedtime two grains of antipyrine given. If so treated, the symptoms may not recur upon the second night, or there may be only the cough without the severe dyspnœa. The child should be confined to the house for two or three days after one of these attacks, the drugs being gradually reduced; but the antipyrine should be given at bedtime for three or four successive nights.

To prevent a repetition of the attacks and remove the tendency to them, it is most important that the child should have plenty of fresh air and cold bathing, especially cold sponging about the neck and chest.

Everything which experience has shown to bring on the attack should be carefully avoided. Local causes, such as adenoid growths, hypertrophied tonsils, elongated uvula, etc., should receive appropriate treatment. Generally it is not necessary to exclude fresh air from the sleeping room. Although an open window on a cold, damp night may sometimes excite the attack, plenty of fresh air tends rather to diminish the susceptibility. If the child's condition is poor, general tonic treatment is to be employed.

ACUTE CATARRHAL LARYNGITIS.

Acute laryngitis is not nearly so frequent as the disease just described, although it is much more severe, and may even be fatal. It occurs especially in children from one to five years of age, usually in the cold season. Predisposition to attacks is induced by the same conditions as in the case of acute rhinitis. Catarrhal laryngitis may be primary, when it is usually excited by cold or exposure,* or it may be secondary to measles, influenza, scarlet fever, or other infectious diseases. It may also be of traumatic origin, from the inhalation of steam or irritating gases.

Lesions.—There is a moderately intense congestion of the laryngeal mucous membrane, sometimes general and sometimes localized. This may be seen with the laryngoscope, but is not always visible after death. With the congestion there are swelling and dryness, followed by increased secretion. In the milder cases the process is limited to the mucosa. In the more severe cases it involves the submucosa also, which is congested, œdematous, and may be infiltrated with cells. The changes are especially marked in the lymphoid tissue of the subglottic region. The swelling may be sufficient to produce a very marked degree of laryngeal stenosis. In many mild and in all the severe cases there is associated catarrhal inflammation of the trachea, and often of the larger bronchi. In young children there is very little tendency to œdema glottidis, so frequent a complication in adults.

Symptoms.—In the mild form, such as that which is usually seen in older children, there is hoarseness, or even loss of voice, and a laryngeal cough which is sometimes hard and teasing, always worse at night. There may be pain and soreness over the larynx. Constitutional symptoms are mild or absent, the patient not usually being sick enough to go to bed, and often rebelling even at being kept indoors. The duration of the disease is from four to ten days, with a strong tendency to relapses from slight causes.

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* The following case is a good illustration of a severe attack excited by cold: A rather delicate infant, eight months old, an inmate of the New York Infant Asylum, was taken out on a raw December day with very slight covering. In a few hours hoarseness and stridor were noticed, and the temperature was 101° F.; three hours later it was 103°, and in spite of the usual remedies which were employed the dyspnœa had reached such a degree as to require intubation. The tube was worn only three days and the case made a prompt recovery.

The severe form of catarrhal laryngitis is sometimes preceded by acute coryza, or there may be mild laryngeal symptoms for a few days before the development of the more severe ones. In other cases the disease develops rapidly and severe symptoms are present within a few hours from the onset.

When the case is fully developed the voice is metallic and hoarse, and occasionally but not usually lost. There is a hoarse, dry, barking cough, which is very distressing, and sometimes almost constant. The cough, like the voice, is stridulous, and more or less stridor is present on inspiration. There is a slight amount of constant dyspnœa, but this is scarcely noticeable unless the chest is bared. Severe dyspnœa occurs in paroxysms, usually at night. Then, we may get the signs of obstructive dyspnœa similar to those mentioned in severe attacks of catarrhal spasm. This dyspnœa is chiefly inspiratory, but in some cases it increases steadily from the beginning of the attack, and may be indistinguishable from that due to membrane. Constitutional symptoms are usually present and may be severe. The temperature ranges in most cases from 101° to 103° F., but may go to 104° or 105°. The pulse is rapid and full and respiration is accelerated. Children sometimes complain of pain in the larynx and trachea, increased by coughing. The symptoms are severe for two or even three days, the fever continuing with moderate prostration and paroxysms of dyspnœa, sometimes even attacks of suffocation and cyanosis. Usually after two or three days there is a gradual subsidence of the dyspnœa and inflammatory symptoms, and the case goes on to recovery. At other times the inflammation extends downward to the large and then to the small bronchi, and finally results in broncho-pneumonia. The attack may prove fatal from laryngeal obstruction due to swelling and spasm.

Diagnosis.—This disease is chiefly to be distinguished from membranous laryngitis. The onset of the two diseases may be very similar, and for the first twelve hours we have no absolute means of distinguishing between them, except possibly by the use of the laryngoscope, which is often conclusive in older children but not usually so in infants. All cases, therefore, should be looked upon with a degree of apprehension. The temperature in the catarrhal is usually higher than in the membranous form. The dyspnœa is mainly paroxysmal, with daily remissions and nightly exacerbations, and is chiefly inspiratory, while that of membranous laryngitis is constant, steadily and often rapidly increasing, and is present both on inspiration and expiration. In catarrhal laryngitis the voice is not usually lost, but in the membranous form this is the rule. There can be little room for doubt when there are enlarged glands, membranous patches on the tonsils, nasal discharge, and albumin in the urine. Very often, however, all these evidences of diphtheria are wanting, the really difficult cases being those in which the process begins in the larynx. The prevalence of diphtheria and a known exposure count for something in favour of membranous laryngitis. If cultures from the pharynx show the presence of Klebs-Loeffler bacilli, diphtheria of the larynx is certain; but no conclusions can be drawn when cultures give negative results. In catarrhal as well as in membranous laryngitis there may be extreme dyspnœa, cyanosis, pallor, prostration, and even death.

Prognosis.—This depends somewhat upon the cause of the disease and also upon the age of the patient. It is much worse when it is secondary to measles or scarlet fever. It is better in children over three years of age than in infants, also when the general condition of the child is good. The prognosis in severe catarrhal laryngitis should always be guarded, not only on its own account, but also because it is impossible to be certain that the case may not be one of membranous laryngitis.

Treatment.—In all cases children affected are to be kept in bed; and the temperature of the room should be between 70° and 72° F. The diet should be light and fluid, and the bowels should be freely opened by calomel or a saline. A hot mustard foot bath should be given at the outset; also, benefit may sometimes be derived from aconite, given in one-quarter-minim doses every fifteen minutes for the first five or six hours. Antipyrine (two grains every four hours to a child two years old) is useful if there is much spasmodic dyspnœa. For this symptom emetics are beneficial, given as in catarrhal spasm. The use of ipecac and squills in smaller doses than is required for emesis (five drops each of the syrups of ipecac and squills every two hours) may give relief, especially in the early stage, when the cough is dry, hard, and severe.

All the remedies mentioned are to be regarded as accessories to the essential treatment, which consists in the use of inhalations. The child should be placed in a tent (page 60) into which steam is introduced from a croup kettle or vapourizer. Simple steam may be used, or turpentine, lime-water, or creosote may be added. In moderately severe cases inhalations should be used for fifteen minutes every two hours; in very severe ones they should be continued the greater part of the time. Poultices or hot fomentations may be applied over the larynx. Relief is sometimes obtained by using counter-irritation by a mustard paste, but blistering should never be allowed. In my experience the local use of cold is very unsatisfactory, on account of the difficulty of applying it properly, and the objection to it on the part of young children. Stimulants may be required late in the disease, the amount of prostration being the guide to their use.

In cases of extreme dyspnœa operative interference may be needed. It is required more often in infants and young children than in those who are older, and especially in the subglottic form of the disease. Opinions will of course differ as to when the dyspnœa has reached the danger point.

One should not wait for general cyanosis. If pallor, marked prostration, and steadily increasing dyspnœa are present the case should not be allowed to go on without interference. Intubation has, to my mind, every advantage over tracheotomy, and is always to be preferred in these cases. One should not hesitate to operate, even though he may be perfectly sure that the case is one of catarrhal inflammation only. The severity of the dyspnœa is the only guide, and more than once I have seen cases shown at autopsy to be catarrhal, which were regarded during life as undoubtedly membranous. If intubation is done, the tube can generally be dispensed with in two or three days. Convalescence is usually rapid, but there is danger of recurring attacks during the remainder of the cold season.

MEMBRANOUS LARYNGITIS.

Synonyms: Membranous croup, true croup, laryngeal diphtheria.

Bacteriology has settled many questions long debated with reference to this disease. For nearly half a century the identity of membranous croup and laryngeal diphtheria has been contended for by some observers, and denied by others equally good. The extensive bacteriological researches made since 1890, both in this country and in Europe, have yielded results sufficiently uniform to warrant the following statements:

1. Membranous inflammation beginning in the larynx is almost invariably true diphtheria—i. e., it is due to the Klebs-Loeffler bacillus.

2. Membranous laryngitis following a primary membranous inflammation of the tonsils, pharynx, or nose, is, in the great majority of cases, due to the Klebs-Loeffler bacillus.

3. Membranous laryngitis following membranous inflammation of the tonsils, nose, or pharynx, occurring as a complication of measles, scarlet fever, or influenza, is sometimes due to another kind of infection (usually the streptococcus), but more often to the Klebs-Loeffler bacillus.

The etiology, lesions, pathological relations, and bacteriological diagnosis of membranous laryngitis are considered in the chapter devoted to Diphtheria. In the present chapter there will be considered only the clinical aspect of the cases, especially of those in which the disease begins in the larynx; for even though in most cases the cause is diphtheria, the clinical picture is that of laryngitis.

In cases of primary laryngeal diphtheria there are wanting most of the characteristic clinical features which distinguish diphtheria of the pharynx. There are two reasons for this: one is the relatively rapid course of the disease, often producing death from local causes before the constitutional symptoms resulting from the absorption of the toxin have developed; the second reason is, that absorption of the poison by the laryngeal mucous membrane is very feeble as compared with that which takes place from the pharynx. Hence it follows that glandular enlargements, albuminuria, and asthenic symptoms are generally wanting; also, that in the cases which come to autopsy early, the parenchymatous degenerations of the heart, kidney, and other organs are seldom found, but instead only such lesions as are connected with the laryngeal disease. The feeble contagion is due to the fact that the course is much shorter, and that the discharge from the nose and mouth is slight, or absent altogether.

Symptoms.—In its onset, membranous inflammation of the larynx is indistinguishable from the catarrhal form. It is perhaps a trifle less abrupt, and apparently not quite so severe for the first twelve hours or even for a longer time. We have the same hoarse cough and voice, with a slight stridor, gradually increasing. The constitutional symptoms are usually not quite so marked, the temperature ranging from 99° to 101° F. The pulse is accelerated, but not weak or intermittent. It is the progress of the disease which indicates its character, usually during the first twenty-four hours. A child beginning in the morning with such symptoms as have been described, may by evening show a decided change for the worse, or the symptoms may increase with great rapidity during the night. At first the voice is hoarse; later it is entirely lost. Dyspnœa in the beginning is scarcely noticeable, but steadily increases hour by hour. At times of excitement it may be very great, but as the spasm subsides it diminishes. During the second twenty-four hours all the symptoms are usually well developed. The respiration is often somewhat accelerated, but it may be slower than normal. The face is pale and anxious. The alæ nasi dilate with each inspiration. The loud, "sawing," stridulous breathing is present. As the dyspnœa increases, all the accessory muscles of respiration are brought into action. There is now with every inspiration deep recession of the suprasternal fossa, the supraclavicular regions, and the epigastrium. The child tosses uneasily from side to side in its crib, at times struggling violently to get more air into the lungs. The pulse grows rapid and weaker. There is slight blueness of the finger nails and the lips; the face is usually pale; but later this too may be cyanotic. The skin is covered with clammy perspiration. On auscultating the chest, very rude respiratory sounds are heard, but no vesicular murmur. As the symptoms increase in severity the temperature usually rises gradually, in some very severe cases at the rate of a degree an hour, until shortly before death it reaches 104° or even 106° F. Late in the disease the intellect becomes dull, the violent struggles for air cease, and the child passes into a condition of semi-stupor which gradually deepens until death occurs, which may be preceded by convulsions.

Such is the usual course of the disease when unrelieved by treatment. Its progress is most rapid in infants, in whom death usually takes place in from thirty-six to forty-eight hours from the first symptoms. In older children the course is rather slower, and the attack may last from two days to a week, death occurring more frequently from bronchial croup or pneumonia. These are indicated by continued high temperature, rapid respiration, cyanosis, and increased prostration.

The course of the disease is not always so regular. Occasionally for a week or more the symptoms are precisely like those of catarrhal laryngitis of moderate severity—hoarseness, laryngeal cough, little or no fever, and slight or occasional dyspnœa. Then there may be the sudden development of very severe symptoms, and death in a few hours. Great improvement may follow the dislodgment of the membrane by vomiting or coughing, although in most cases it forms again.

Prognosis.—The issue of every case of membranous laryngitis is doubtful. The prognosis is worse in infants and very young children than in those over three years of age, and worse when secondary to measles or scarlet fever than when primary. Before the days of antitoxin the mortality of cases not operated upon was from 80 to 90 per cent. Later statistics are given in the chapter on Diphtheria.

Diagnosis.—The first point to be decided in any case is whether the dyspnœa is due to laryngeal inflammation; the second whether this inflammation is catarrhal or membranous. The dyspnœa of retro-pharyngeal abscess, of foreign bodies in the larynx or trachea, or of broncho-pneumonia, may be mistaken for that due to laryngitis. But in none of these conditions should there be any doubt if a careful examination is made and a history obtained. Retro-pharyngeal abscess may be recognised by digital examination of the pharynx; broncho-pneumonia by the signs in the lungs, the difference in the character of the dyspnœa, and especially by the absence of the noisy stridor; in the case of foreign bodies, whether they enter through the mouth or consist of ulcerating caseous glands which have ruptured into the trachea, the dyspnœa comes suddenly, and is not accompanied by fever. The main points by which catarrhal laryngitis is distinguished from the membranous form have been considered under the former disease. In brief, membranous inflammation may be assumed if there is severe, constant, and increasing dyspnœa with aphonia. Membranous laryngitis should always be regarded as diphtheria until the opposite has been proved by repeated cultures.

Treatment.—All cases of membranous laryngitis should be isolated like those of diphtheria of the pharynx, and should receive a full dose of antitoxin upon a clinical diagnosis without watiting for this to be confirmed by a bacteriological examination. Nowhere else are the beneficial effects from antitoxin so evident and so striking as in these cases. For dosage and other details regarding the use of antitoxin the reader is referred to the article on Diphtheria.

Emetics, inhalations of steam, and solvents for the membrane, although they all sometimes give relief, are never to be relied upon alone. In fact, leaving out antitoxin and surgical operation, the only therapeutic measure that can be said to be of much avail is calomel fumigation.

This is in no sense a substitute for antitoxin, but may be employed where the use of antitoxin is impossible, and in the few cases of membranous laryngitis due to streptococci. From ten to fifteen grains of calomel are vapourized upon any hot metal plate under a closed tent, in which the child is placed. This may be repeated every one to four hours, according to circumstances. One should watch both the child and the attendants for symptoms of mercurial poisoning. This treatment was introduced by Corbin, of Brooklyn, and was much in vogue from 1890 until the introduction of antitoxin.

Operative measures.—Opinions will always differ as to the time when operative interference is called for. One should never wait for general cyanosis, for often this does not occur until just before death. It is better to operate too early than too late. If, in spite of other measures, the dyspnœa increases steadily, and especially if the temperature begins to rise, operation should not be deferred longer. When this has been decided upon, the physician has the choice between intubation and tracheotomy. In America intubation has almost universally superseded tracheotomy as a primary operation for the relief of membranous laryngitis. In Europe also its advantages are coming to be appreciated, and its use has extended greatly since the introduction of antitoxin. Tracheotomy is still needed at times for the cases, very few in number, in which intubation fails to give relief on account of the position of the membrane or some other complication.

The general treatment of the child is important, and should not be overlooked. It includes careful feeding, and the use of alcoholic stimulants according to the amount of prostration present. All patients with membranous laryngitis should be closely watched, for marked changes may take place in the course of a few hours.

INTUBATION.

Intubation is the introduction of a tube through the mouth into the larynx for the relief of laryngeal dyspnœa. For the operation, as now performed, the world is indebted to the late Dr. Joseph O'Dwyer, of New York.

A set of O'Dwyer's instruments (Fig. 85) consists of six tubes, an introductor, an extractor, a mouth-gag, and a gauge. In the later tubes the lower extremity is made somewhat bulbous and not straight, as appears in the illustration. His latest tubes are made of hard rubber and lined with gold-plated metal, these proving much less irritating than the solid metal tubes formerly used. So carefully did O'Dwyer perfect his instruments that nothing of importance has been added by others. It is interesting to note that nearly all the modifications which have been suggested since his first publication had already been tried by him and discarded. No one thing is more essential to success with intubation than properly constructed instruments. The operation is not difficult, if one has had practice on the cadaver. Without this it should not be attempted. The tube is selected according to the age of the patient, this being indicated on the gauge. A very large child will often require a tube of larger size than its age would call for.

Introduction of the Tube.—Either one of two positions may be employed, the choice depending upon the preference of the operator. Formerly the usual method was to have the child seated upon the lap of a nurse while his head was steadied by a second assistant standing behind. In the other position the child lies upon his back upon a table, his head being steadied by an assistant. In both positions the arms should be pinioned to the sides by a sheet. In the recumbent position the child can be held more firmly; it has also the advantage of dispensing with one assistant, and in an emergency with both of them. The tube is attached to the introductor, and the gag is inserted into the left angle of the mouth and opened as widely as possible. The slipping of the gag and laceration of the mouth may be prevented by using a piece of rubber tubing to cover each arm of the gag where it comes in contact with the gum. The attempts at introduction must be made quickly, for during them respiration is practically arrested. Several short attempts are always better than a single prolonged one. Very

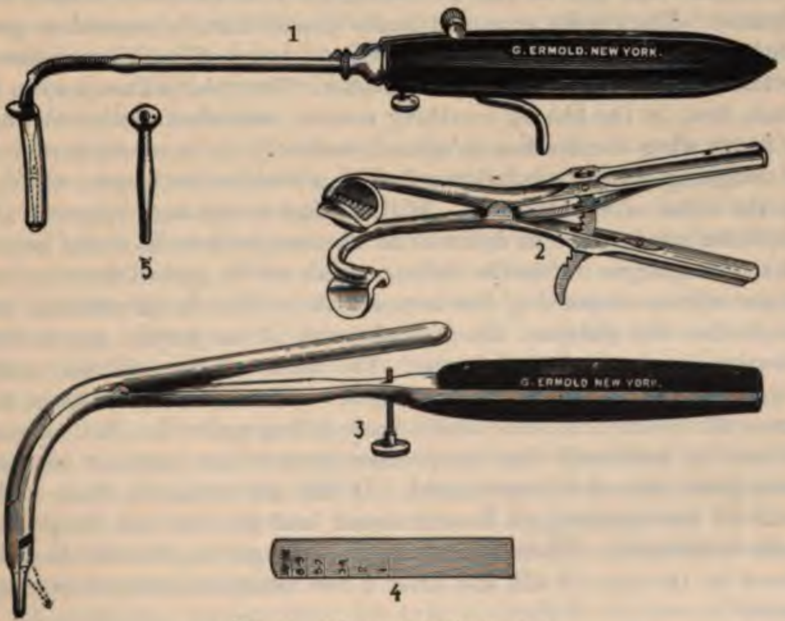

Fig. 85.—O'Dwyer's intubation set.
1, introductor; 2, gag; 3, extractor; 4, gauge; 5, tube.

little force is ordinarily required in introducing the tube, that used in passing a catheter being a good general guide. In cases of subglottic stenosis, however, quite a little force may be necessary.

The index finger of the left hand is used as a guide in introduction. This is passed well back into the pharynx, then brought forward until a hard nodule—the upper border of the cricoid cartilage—is encountered. This is the best of all landmarks, since the soft parts are often distorted by swelling. Directly in front of the cricoid cartilage may be felt the epiglottis and the opening of the larynx, which are readily recognised after the touch has become somewhat educated. The tube is passed along the palmar surface of the left index finger, by which it is guided into the larynx; it is then pushed off the introductor by a thumb-piece attached to its handle. When it is certain that the tube is in position, and the patient breathes properly, the loop of silk attached to the head of the tube is cut off and pulled through, the removal of the tube being prevented by placing the left forefinger upon its head. The silk is not usually left attached unless there is evidence of loose membrane below the tube. It may be desirable to leave the silk attached in case no one can be within reach who is able to remove the tube should it become obstructed. The child's arms and hands should then be secured to prevent him from seizing it himself. When not removed the silk is fastened to the cheek by a piece of adhesive plaster. The tube is known to be in place, first, by the hissing breathing sounds, somewhat similar to what is heard when the trachea is opened; secondly, by a severe paroxysm of coughing, which is usually excited by a tube in the larynx; thirdly, by the relief of the dyspnœa. If this relief is not very apparent the physician may still be in doubt as to whether the tube is in the larynx or the œsophagus. If in the former, it can not be pushed down by the finger without depressing the larynx with it; and by introducing the finger into the pharynx, the posterior wall of the larynx can be felt between the finger and the tube. The most common mistake made is to pass the tube into the œsophagus. This sometimes happens because the position of the child's head is improper—too far forward or too far backward—but more often because the operator has not been quite sure of his landmarks. If this has occurred, there is no relief to the dyspnœa, no hissing sound, and the tube can be pushed down indefinitely. When this condition is recognised, the tube is withdrawn by the loop of silk and after a few moments a second attempt made.

False passages in the larynx are most frequently made by employing too much force or because the operator has worked at the angle of the mouth instead of keeping in the median line. The tube usually goes into one of the ventricles, and may be pushed quite through the larynx into the cellular tissue. This is not likely to happen unless undue force has been used. The production of a false passage is recognised by the fact that, although the tip of the tube can be felt to enter the larynx, it does not descend, but projects above the epiglottis.

False membrane which has become loosened is sometimes crowded down by the tube and obstructs the larynx just below it. This is one of the most serious accidents that may occur, but fortunately it is not a frequent one. It is more likely to happen where the disease has existed for several days than in recent cases. The tube may be in place in the larynx as shown by all the signs above mentioned, except relief of the asphyxia. In such a case the immediate withdrawal of the tube is necessary, it being often followed by the discharge of masses of loose membrane. This is aided by the administration of a teaspoonful of pure whisky or brandy to excite a strong cough. Artificial respiration may be required, and if there is no relief by any of these means tracheotomy is indicated. Asphyxia is sometimes produced by prolonged and injudicious attempts at introduction.

After-treatment.—So far as the tube itself is concerned no treatment is required. The original disease is to be treated as before. The operation has removed only one danger from the patient, viz., that of asphyxia from mechanical obstruction of the larynx. A good expulsive cough should occur after the tube is in place. This is necessary to clear the tube of mucus, as the pharynx and larynx are generally filled with it as a result of the manipulation.

The child should not be allowed to lie upon its face, nor should it be held over the nurse's shoulder face downward, for in either position a slight cough is enough to expel the tube. Nursing infants may continue at the breast after the operation; ordinarily they have but little difficulty in swallowing. Older children often experience considerable trouble in taking liquids. This may be overcome by the device suggested by Casselberry (Chicago), of having the patient's head lower than his body while he drinks. If there is still trouble in taking fluids, semi-solid articles, such as condensed milk, wine jelly, corn starch, or scrambled eggs, may be tried. Feeding is always easier after the first day or two, and patients who wear a tube for chronic disease soon experience no trouble whatever, showing that the difficulty depends more upon the inability to co-ordinate the movements of the muscles of deglutition when the tube is in place than upon mechanical causes, for the head of the tube is effectually covered by the epiglottis.

It sometimes happens that the tube is coughed out soon after its introduction, because too small a size has been used. In some cases this occurs repeatedly. It happened in a case of my own twenty-eight times during four days. Such cases are probably due to paralysis of the laryngeal muscles. The dyspnœa does not usually return for two or three hours after the tube has been coughed out, so there is ample time to notify the physician. It may happen that the tube is coughed up and not seen by the nurse, or it may be coughed up and swallowed by the child. When called because of dyspnœa after operation, the physician should make a digital examination of the pharynx to be sure that the tube is still in place. Swallowing the tube generally causes no harm to the child, for tubes have repeatedly passed through the intestines.

The entrance of food into the bronchi through the tube is a danger that does not exist, as has been shown by the extensive post-mortem observations of Northrup in the New York Foundling Asylum. My own experience in the New York Infant Asylum coincides in every particular with his statement, that the broncho-pneumonia following intubation does not depend upon the entrance of food into the bronchi.

Ulceration at the head of the tube very rarely occurs, provided properly made tubes are employed.* The tube rests not upon the vocal cords, but upon the inferior ventricular bands. When ulceration occurs, it is usually of the anterior wall of the trachea, at the lower end of the tube, and appears to be produced by the movements of the tube during deglutition. With O'Dwyer's latest tubes there is much less liability of this occurring. The ulcers are usually small and superficial. Deep ulcers extending to the tracheal rings may be seen in ill-conditioned children, usually in connection with other complications severe enough to cause death.

Spontaneous descent of the tube into the larynx is impossible, and it can not be crowded down without using considerable force and severely lacerating the larynx.

Sudden blocking of the lower end of the tube by membrane loosened from the trachea or bronchi is an infrequent accident. The usual result of this is the immediate expulsion of the tube by coughing, the discharge of the loose membrane following. This condition is one of the safety valves of the operation. One of the strong points in favour of intubation is that the forcible cough which the patient is able to make on account of the narrow opening of the tube, often enables him to expel large accumulations of mucus, and even membrane, more readily than through a much larger tracheal opening.

The period for which the tube is required varies much in different cases. It is the experience of practically all operators that it has been materially shortened by the use of antitoxin. According to the statistics of Rosenthal (Philadelphia), the average reduction amounts to two and a half days, the average time of wearing the tube is five days, and in many it can be dispensed with in two or three days. Should the tube be coughed out at any time, its introduction should be delayed until dyspnœa returns.

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* This and many other bad results obtained after intubation are due to improperly constructed instruments. Those made by George Ermold, 201 East Twenty-third Street, New York, are the most reliable.

Removal of the Tube—Extubation.—This is rather more difficult than its introduction. The general arrangement of the patient and assistants is the same as for introduction. The left index finger is placed upon the head of the tube, which is steadied externally by the thumb of the same hand. The beak of the extractor is introduced within the opening of the tube, its jaws are then separated by pressure upon the lever at the handle, and the instrument withdrawn, very slight force being required.

The tube is first removed tentatively, the physician waiting to see if dyspnœa returns. It is well to give a full dose of morphine an hour before the removal of the tube, since the contact with the air almost invariably excites a marked degree of laryngeal spasm which lasts for ten or fifteen minutes. To avoid the production of vomiting and the entrance of food into the larynx, food should not be given for two hours previously. If dyspnœa does not return in the course of three or four hours, the probabilities are that the tube will no longer be required. It is very exceptional that the patient has great difficulty in dispensing with the tube, as so often happens after tracheotomy.

The Advantages over Tracheotomy.—The advantages claimed by O'Dwyer for this operation over tracheotomy are conceded by most of those who have had any considerable experience in the operation, viz.: (1) It is quicker, simpler, and adds no danger to the original disease; (2) there is no shock or hæmorrhage; (3) no anæsthetic is required; (4) no fresh wound is made which may prove an avenue of infection; (5) it gives an opportunity for a better expulsive cough, which is of great value in dislodging false membrane and mucus; (6) there are usually no objections on the part of the parents to be overcome—a point of great importance; (7) the air is warmed and moistened as it is normally, by passing over the nasal and buccal mucous membranes; (8) no skilled after-treatment is required: as the largest proportion of the cases of diphtheria are among the very poor, living under conditions in which the careful after-treatment required in tracheotomy is difficult or impossible to obtain, this is an important point; (9) in infancy, all who have had experience with both operations admit the great superiority of intubation; (10) the intubation tube can be dispensed with earlier than the tracheal canula, and also with much less difficulty; (11) if tracheotomy is subsequently required, the operation may be done upon the tube as a guide.

The only objection of much force urged against intubation is that asphyxia may be produced by crowding down loose membrane into the larynx. This is a very infrequent accident; should it happen, and the asphyxia not be relieved by coughing up the membrane, tracheotomy may be performed.

There is always some degree of hoarseness following intubation, but in the majority of cases it disappears within a week, occasionally it continues as long as three or four weeks, but it is very rarely if ever permanent. The duration of the aphonia seems to have no relation to the length of time the tube is worn.

Experience has clearly proved that intubation relieves the dypsnœa due to laryngeal stenosis promptly, efficiently, and certainly; it does this without many of the dangers and objectionable features of tracheotomy, while at the same time it does not deprive the patient of any essential advantage which tracheotomy affords.

Retained Intubation Tubes—Prolonged Intubation.—Difficulty is experienced in dispensing with the intubation tube much less frequently than with the canula after tracheotomy; yet when this condition occurs it is the cause of much concern and even danger. Trouble of this sort is seen, according to Rogers, in about one per cent of the cases of intubation. In the majority of these the patient is able to do without the tube in a few weeks, and such cases require very close attention, but no special treatment other than the substitution at times of a special O'Dwyer tube with an extra large "retaining swell." But occasionally there are met with cases in which every effort to dispense with the tube seems fruitless. Although the children breathe well with the tube in place, still if it is removed or expelled by coughing, in a short time, varying from a few minutes to an hour or two, the dyspnœa returns with such severity that the tube must be replaced immediately to prevent asphyxia. Inasmuch as these patients sometimes expel the tube several times a day, surgeons have often resorted to tracheotomy to avert the danger of suffocation, which might easily occur if no one were at hand who could replace the tube. This operation, however, gives only temporary relief. Many of these children, after wearing tubes of one sort or another for years, ultimately die from some accident connected with the tube or from pneumonia.

The causes and the exact pathological condition underlying this difficulty are subjects regarding which there has been much difference of opinion. O'Dwyer's opinion was that the cause of the returning dyspnœa was subglottic swelling and œdema which occurred in tissues which were the seat of chronic inflammation as soon as the pressure of the tube was removed. The primary cause of the condition he believed to be the injury inflicted by improperly made or badly fitting tubes, or by unskilful efforts at introduction. In a few cases a cicatricial condition, the result of previous ulceration, has been found; but it is doubtful if granulations, so frequent a cause of retained canula after tracheotomy, play any part whatever. Rogers's view is that the chronic inflammation of the mucous and submucous tissues of the subglottic region of the larynx which produces the symptoms, is due neither to a faulty tube nor to a clumsy operation, but to the nature of the pathological process.

For the relief of this condition, O'Dwyer advised in recent cases the application of astringents by means of an intubation tube coated with gelatine with which some astringent was combined. For those patients who cough out the tube frequently, tracheotomy is at times a necessity to prevent sudden death. But this does not affect the original condition, for the same difficulty exists in doing without the tracheal canula. The operations of laryngotomy, curetting, etc., have been such signal failures as to discourage one from repeating them.

The most successful method of treatment thus far proposed is that of Rogers,* which consists in increasing intra-laryngeal pressure by the insertion of larger and larger intubation tubes. This is not to be adopted until long after all acute symptoms have subsided. The first tube used is as large a one as can be introduced without force; after a few weeks, the next larger size, and after a longer interval, possibly a still larger one. When the very large tube had been worn for several weeks he was finally able to dispense with all tubes. In this way he succeeded in curing completely and permanently several cases of two or three years' standing.

True cicatricial stenosis may best be relieved by opening the trachea and dilating from below, and afterward inserting an intubation tube. When there is complete destruction of the cricoid cartilage, as sometimes occurs, tracheotomy is the only remedy, but this is only palliative, as the tube must be worn permanently.

SUBMUCOUS LARYNGITIS—ŒDEMA OF THE GLOTTIS.

These two conditions are not quite identical, although they are closely associated and may be conveniently considered together. They are both rare in early life. In true œdema of the glottis there is simply a dropsical effusion into the submucous cellular tissue of the aryteno-epiglottic folds, causing them to project as large rounded swellings on either side of the superior isthmus of the larynx. They may be of sufficient size to cause serious or even fatal obstruction to respiration. With the laryngoscope they appear as pale red tumours, lying usually in contact near the base of the tongue. By the finger their presence can be quite as readily distinguished. Œdema of the glottis occurs principally in the late stages of nephritis.

In the inflammatory form of œdema, or true submucous laryngitis, there is the same sort of swelling of these structures, but in this case it is due to some active inflammation in the neighbourhood. The swelling is partly from the œdema and partly from cell infiltration. Usually all the parts surrounding the upper opening of the larynx are in a state of acute inflammation. The epiglottis may be swollen to the thickness of a finger, and easily seen by depressing the tongue.

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* Post-Diphtheritic Stenosis of the Larynx, John Rogers, M. D., Annals of Surgery, May, 1900. See also monograph by von Bókay, Ueber das Intubations-trauma, Leipzig, 1901.

The *exciting causes* may be the mechanical irritation of foreign bodies, the inhalation of steam or irritating gases, erysipelas of the neck, primary catarrhal laryngitis, or retro-pharyngeal abscess.

The *symptoms* in both cases consist of great inspiratory dyspnœa with attacks of suffocation, while expiration may be quite easy. In true œdema there are in addition the symptoms of the primary disease. In the inflammatory form there are the evidences of local inflammation—hoarseness, cough, pain, and difficulty in swallowing. A positive diagnosis may be made by a digital examination. The symptoms develop with great rapidity in either variety, and frequently prove fatal in a few hours.

The *treatment* of true œdema consists in scarification or multiple puncture, the application of ice externally, and even the swallowing of ice; in the inflammatory form, in addition, local blood-letting by leeches and, as a last resort, tracheotomy. Intubation is useless in either form.

CHRONIC LARYNGITIS.

The following varieties are seen: (1) a simple form usually associated with adenoid vegetations of the pharynx; (2) tuberculous; (3) syphilitic; (4) that associated with new growths.

1. **With Adenoid Vegetations of the Pharynx.**—This is not very uncommon. The larynx is kept in a state of chronic congestion by the adenoid growth, and there finally develops a sight superficial catarrhal inflammation. The symptoms may continue for many months. These cases are often treated for a long time unsuccessfully by the use of sprays, inhalations, etc., but the symptoms disappear rapidly after the removal of the adenoid growth. Similar symptoms may be associated with hypertrophic rhinitis. In this also the treatment should be directed to the primary condition.

2. **Tuberculous Laryngitis.**—This belongs to later childhood, and is rare even then. In infancy it is almost unknown. Rheindorf * has reported a case in a child of thirteen months, which was regarded during life as syphilitic, but was shown by autopsy to be tuberculous. Of sixteen cases in children, reported by Rilliet and Barthez, none occurred during the first three years, and only four before the seventh year. The larynx alone may be affected, or the larynx and trachea, or the larynx, trachea, and lungs. Pulmonary tuberculosis is usually found to be present at autopsy,

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* Jahrbuch für Kinderh., Bd. xxxiii, p. 71.

even though there may have been no pulmonary symptoms. Demme has reported a case of tuberculous laryngitis in a boy of four years, whose lungs were healthy, death resulting from tuberculous meningitis.

The *symptoms* are hoarseness, aphonia, laryngeal cough, and mucopurulent, sometimes bloody, expectoration. The sputum may contain tubercle bacilli. With the laryngoscope tuberculous deposits may be seen, but more frequently tuberculous ulceration of the mucous membrane. In children this is usually superficial, the deep destructive ulceration seen in adults being very rare.

It is to be differentiated from syphilis chiefly by the general symptoms, as the laryngoscopic appearances may be very similar. The *treatment* consists in keeping the ulcers as clean as possible by the use of sprays and the local application of astringent powders, like nitrate of silver and sulphate of zinc or iodoform.

3. **Syphilitic Laryngitis.**—In the early stage of syphilis the larynx is often the seat of a catarrhal inflammation, which presents nothing especially characteristic except its protracted course. The laryngitis of late hereditary syphilis is quite rare, and is liable to be overlooked because of the difficulties in the way of a thorough examination, and because the disease is usually painless.

Strauss* has collected fourteen cases between the ages of three and fifteen years, and added three of his own. He states that deep-seated processes are much more rare than among adults. The parts most frequently affected are, first, the epiglottis; secondly, the aryteno-epiglottic folds; thirdly, the posterior laryngeal wall. The epiglottis was involved in twelve of fourteen cases. Usually there was only perichondritis; in the more severe cases there was partial or complete destruction of the cartilage. In four cases papillomatous masses were seen. In five cases the process extended from the epiglottis to the epiglottic folds of one or both sides. In several instances the superior vocal cords were thickened from hyperplasia, and occasionally small tumours were formed. In only one case was there ulceration of these folds. Changes in the vocal cords and the arytenoid cartilages were rare, occurring only with extensive inflammation. The symptoms are those of chronic laryngitis; hoarseness, sometimes aphonia, and in a few cases chronic laryngeal stenosis. The diagnosis can be made only by means of the laryngoscope. In most of the cases there are present ulcerations of the palate or uvula, or scars from previous ulcers; sometimes the disease extends into the nose. Serious symptoms often result when to old syphilitic lesions there is added acute laryngitis or œdema.

In addition to the usual constitutional remedies for tertiary syphilis, and to the means ordinarily employed for the relief of chronic laryngitis, intubation may be required in these cases for the relief of laryngeal stenosis. Nowhere are its advantages over tracheotomy more striking than here. The tube must usually be worn for many months.

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* Archiv für Kinderh., Bd. xiii.

NEW GROWTHS.

New growths of the larynx are not very rare in children. Excluding the granulations which follow the use of the tracheal canula, the only one that is likely to be met with is papilloma. This may occur even in infancy. According to Rauchfuss, the majority of the cases begin during the first year. Boys are more frequently affected than girls.

The *symptoms* depend upon the size and location of the tumour. The earlier manifestations are usually ascribed to chronic laryngitis. There is hoarseness, sometimes loss of voice, and a paroxysmal cough; later, dyspnœa develops. The symptoms are slowly progressive, and it may be several months before they are sufficiently severe to attract special attention. A positive diagnosis is made only by the laryngoscope. There is seen a whitish granular tumour, sometimes pedunculated, sometimes with a broad base, attached to any part of the larynx.

The *treatment* of these cases belongs to the specialist. Small pedunculated growths may be removed through the mouth by means of the forceps or snare. Larger ones require thyrotomy. The prognosis is generally unfavourable, on account of the danger of recurrence after operation. Operative measures are very frequently followed by bronchitis or broncho-pneumonia.

FOREIGN BODIES IN THE LARYNX.

The aspiration of foreign substances into the larynx is not a very rare accident in children. It usually happens from an attempt to cough, laugh, or cry while the child has something in its mouth. If the body is sharp and irregular, like a pin, the shell of a nut, or a fragment of bone, it is liable to become impacted in the larynx. If smooth, like a pea or a bead, it is usually drawn into one of the bronchi, generally the right.

When the body enters the larynx there is immediately excited a violent paroxysmal cough, with dyspnœa amounting almost to suffocation. Often the body is dislodged by this initial attack of coughing. If it becomes impacted in the larynx, it may cause sudden death by occluding the glottis; elsewhere it may excite acute laryngitis, usually of considerable severity.

The impaction of a foreign body in one of the primary bronchi, or one of the lobar divisions, is indicated by cough and a severe localized pain in the chest. There may be expectoration of blood. On auscultating the chest, there is found an absence of respiratory murmur over one lung or one lobe, according to the situation of the foreign body. Percussion gives increased resonance, which may even be tympanitic, owing to emphysema which rapidly develops. If the foreign body remains impacted in one of the bronchi, it usually excites a localized inflammation, which extends to the surrounding lung and terminates in the formation of an abscess. This may result fatally, or there may follow a prolonged illness, with hectic symptoms resembling pulmonary tuberculosis; and finally, after weeks or months, the foreign body may be expelled by an attack of coughing, and the patient recover completely.

The *diagnosis* of a foreign body in the larynx is made by the suddenness of the attack and the violence of the early symptoms. In older children the body may be seen with the laryngoscope, but in young children this is very difficult. The prognosis is always doubtful, and depends upon the nature of the foreign body and the point at which it has been arrested.

Treatment.—The first thing to be tried is inversion of the patient. By this means, assisted by the cough, the foreign body is not infrequently expelled, even though it has passed below the larynx. The symptoms of laryngeal obstruction may call for immediate tracheotomy or laryngotomy, intubation not being applicable to these cases. If, after tracheotomy, the foreign body can be located in the larynx, but can not be extracted through the tracheal wound, the thyroid cartilage should be divided in the median line. The removal of a foreign body from the bronchi or the tracheal bifurcation should be attempted only by a skilled surgeon.

CHAPTER III.

DISEASES OF THE LUNGS.

THE PECULIARITIES OF THE LUNGS IN INFANCY AND EARLY CHILDHOOD.

Thorax.—The general shape of the thorax is somewhat cylindrical, the conical or dome-shape of the adult thorax not being attained until puberty. The antero-posterior and the transverse diameters are nearly equal in the newly born, but after the third year the transverse diameter is always greater, the difference increasing steadily up to adult life. On account of the shape of the chest, the lungs are situated rather more posteriorly in the infant than in the adult.

The thoracic walls are very elastic and yielding, owing to the cartilaginous condition of a large part of the framework. They are relatively thinner than in the adult, chiefly from the imperfect development of the thoracic muscles. The greater part of the thickness of the thoracic walls is due to the deposit of fat, generally abundant in well-nourished infants; but where the fat is scanty the walls are extremely thin. The capacity of the thorax is considerably encroached upon by the high position of the diaphragm, the large size of the thymus gland, and the frequent distention of the stomach and intestines.

Respiration.—According to Uffelmann, the rapidity of respiration during sleep at the different ages is as follows:

At birth	35 per minute.
At the end of the first year	27 " "
At two years	25 " "
At six years	22 " "
At twelve years	20 " "

During waking hours this rate is very materially increased, and from comparatively slight disturbance it may be nearly twice as rapid.

The type of respiration in infants is diaphragmatic, and it continues to be chiefly so until after the seventh year, when the costal element gradually becomes more and more prominent. The rhythm of respiration is easily disturbed. In very young infants the regular rhythm is seen only in sleep. The lungs do not always expand equally; at certain times and in certain positions respiration may be carried on for a few moments almost entirely with one lung. For some moments it may be very superficial, and then quite deep. The length of the interval between inspiration and expiration varies much at different times. Regular rhythmical respiration is not fully established before the end of the second year. After this time disturbances of rhythm are chiefly due to pulmonary or cerebral disease; but in infancy quite marked irregularity may have little or no significance. It is very common in all asthenic conditions.

Structure.—As compared with the adult, the trachea of the young child is larger; the bronchi are larger, more numerous, and occupy a greater space; the air cells are much smaller and occupy less space; and the interstitial tissue is much more abundant.

Physical Examination.—This requires tact and time, but yields results which are quite as satisfactory as in adults. It should be undertaken only in a room having a temperature of about 72° F., or before an open fire.

Inspection.—This should be made with the chest bare. There should be noted, the shape of the chest, the presence of deformities from rickets, the want of symmetry in the two sides, bulging of the intercostal spaces, whether the two lungs expand equally or not, also variations in rhythm, and the presence and extent of any recession of the soft parts or bony walls as an indication of obstructive dyspnœa.

Palpation.—This also should be made upon the bare skin, always with the hand well warmed. Although we can not get the fremitus of the voice, we can get that of the cry. This is usually more intense than in adults, on account of the thinness of the chest walls. We frequently get a bronchial fremitus—a vibration produced by mucus in the tubes. This may enable one to recognise bronchitis quite as positively as by the ear.

The position of the apex beat of the heart should be determined, it being remembered that in infancy this is normally in the mammary line, or just outside of it, and usually in the fourth intercostal space.

Percussion.—For the examination of the back, the child may be laid face downward upon the nurse's lap, or be seated upon her arm. For the front and the lateral regions of the chest, the child is most conveniently placed upon its side across a hard pillow. The percussion blow must be light, either with a single finger or a small percussion hammer, using a finger of the opposite hand as a pleximeter. Percussion should be made both during inspiration and expiration. The normal percussion note is somewhat tympanitic, this being due to the relatively large bronchi and the thin chest walls. This note is exaggerated in the interscapular region and beneath the clavicle, especially upon the right side. Here cracked-pot resonance may be obtained even in health. In early infancy the thymus gives dulness over the sternum as low as the third rib, sometimes even below this point, this gradually diminishing as age advances.

Auscultation.—This may be practised with the naked ear or with the stethoscope. A stethoscope is absolutely necessary for a thorough examination of the apices of the lungs in front and in the axillary regions. Most children are less frightened by the instrument than by the head of the physician during anterior auscultation. For the posterior part of the lungs, the stethoscope may be dispensed with. One with a small bell from one-half to three-fourths of an inch in diameter is of great advantage. In auscultating with the ear it is not necessary to bare the skin. The physician should always auscultate the posterior part of the chest first, because he is most likely to find signs of disease there, and also because this is not so apt to frighten the infant. Every part of the chest should, however, be thoroughly auscultated, not omitting the high axillary regions. A convenient position for posterior auscultation is to have the child held over the nurse's shoulder.

The normal respiratory murmur of the infant is generally described as puerile. In quality this has been likened to the bronchial breathing of the adult, but the resemblance is not a very close one. It is rude, rather loud, and seems very near the ear. Its peculiar character is due to the fact that the tracheal and bronchial sounds are more distinct, because not transmitted through so thick a layer of lung and chest wall. It is especially loud in the regions where the bronchi are superficial, as between the shoulder-blades and beneath the clavicles, particularly of the right side. A careful comparison of the two sides of the chest will generally enable an observer to avoid errors. The irregularity of rhythm which occurs from slight causes should be remembered, and the infant's position changed several times during auscultation, to avoid the mistake of attaching too much importance to a feeble respiratory murmur of one side.

On account of the thinness of the chest walls, there is always great difficulty in distinguishing between râles produced in the bronchi and pleuritic friction sounds. Before drawing any inference from the auscultatory signs, both lungs must be examined for several minutes, changing the child's position, and often inducing a cry or compelling a deep inspiration by other means, in order to bring out signs which otherwise may be overlooked. As auscultation is extremely difficult or impossible in a crying infant, this part of the physical examination should first be made if the child is quiet, since upon it we must chiefly depend for diagnosis. Inspection and percussion can be deferred until later.

Peculiarities in Disease.—There are several peculiarities connected with the respiratory organs in infancy and early childhood which must be constantly borne in mind in studying their diseases. The muscular development of the thoracic wall is feeble. The soft, yielding character of the thoracic framework causes the chest to sink in readily from atmospheric pressure whenever there is obstructive dyspnœa. On account of the small size of the air vesicles, acute congestion may interfere with their function almost as completely as does consolidation. Because of the delicate walls of the air vesicles, emphysema is readily produced in obstructive dyspnœa, but it is rarely permanent. There is a tendency to collapse, either on the part of lobules or groups of lobules, but very rarely of an entire lobe. This is a much less important factor in the production of symptoms in acute pulmonary disease than many writers would lead us to suppose. The tendency of inflammation to spread from the large to the small bronchi is very much greater than in adults. In all forms of pulmonary disease the rapidity of respiration is much greater than in adults, on account of the rapid metabolism of the child. Areas of consolidation often exist without appreciable changes in the percussion note, because they are superficial and are surrounded by healthy or emphysematous lung. Flatness should always suggest the presence of fluid. Disease is often overlooked, from a failure to examine the whole chest.

Probably the most common mistakes are to confound bronchial râles with friction sounds, exaggerated puerile breathing with bronchial breathing, and to overlook the existence of fluid because of the presence of bronchial breathing.

ACUTE CATARRHAL BRONCHITIS.

Acute catarrhal bronchitis is one of the most frequent conditions for which the physician is called upon to prescribe in children. It occurs at all ages, from early infancy up to puberty. Its frequency, however, diminishes steadily after the second year. The predisposition to acute bronchitis exists with the same constitutional conditions, and is acquired in the same manner as the predisposition to the acute catarrhal inflammations of the upper respiratory tract. (See Acute Rhinitis). Bronchitis is very common in children who are suffering from rickets and malnutrition. It is much more frequent in the cold months, especially in the late winter and early spring, when there are sudden atmospheric changes and high winds.

Bronchitis may be a primary or a secondary disease. The primary form is excited by cold, exposure with insufficient clothing in severe weather, wetting of the feet, or chilling of the surface in any manner. Under these conditions it may occur alone, or be associated with or preceded by acute catarrh of the nose, pharynx, or larynx. In rare cases it is caused by the inhalation of irritants. Bronchitis is an almost invariable accompaniment of measles and influenza. It is very common in pertussis, in scarlet and typhoid fevers and diphtheria, and may occur in any acute infectious disease; it also complicates pneumonia and pleurisy. The relation of micro-organisms to the other etiological factors is the same as in the other acute catarrhs. (See Rhinitis).

Lesions.—Acute catarrhal bronchitis is an inflammation of the mucous membrane of the bronchi. As a rule it is bilateral, both sides being involved to the same degree. Localized bronchitis is secondary to some other pathological process in the lungs, usually tuberculosis or pneumonia. In acute bronchitis only the larger tubes may be affected, this usually being complicated with inflammation of the trachea (ordinary tracheo-bronchitis); or, in addition, the process may extend to the medium-sized tubes (severe bronchitis); or, in infants especially, it may extend to the smallest tubes (capillary bronchitis). In the last-mentioned form there are invariably changes in the zones of air vesicles surrounding the bronchi, and these cases are therefore more properly classed as broncho-pneumonia. In the first form the inflammation is superficial, and affects only the mucous membrane of the bronchi. In the second form it may involve the entire thickness of the bronchial wall, and in the third form it does so regularly.

The pathological changes consist in congestion and swelling of the mucous membrane, desquamation of the epithelium, and an exudation of mucus and pus-cells. At autopsy the injection of the mucous membrane is usually distinct; pus and mucus line the walls of the larger bronchi, and by pressure ooze from the cut extremities of the smaller tubes. The chief lesion of the walls of the bronchi consists in an infiltration with leucocytes. In infants dying from bronchitis, the lungs are much more frequently emphysematous than collapsed. There is swelling of the lymph glands at the root of the lungs, which in most of the acute cases is slight, but in protracted cases, and after recurring attacks, may be quite marked.

Symptoms.—It is convenient to consider separately the symptoms in infants and in older children.

The bronchitis of infants.—1. The mild form (bronchitis of the larger tubes).—The onset is generally gradual, and the symptoms of bronchitis may be preceded by those of catarrh of the nose, pharynx, or larynx. The change in the character of the cough, the slightly accelerated breathing, and a further rise in temperature, indicate an extension to the bronchi. The cough may be constant and severe, or very slight. There is no expectoration. The secretions are usually coughed up into the mouth or pharynx, and swallowed. This sometimes excites vomiting. At other times the mucus is coughed only into the trachea or larynx, and aspirated again into the lungs. The respirations are from 40 to 50 a minute, and often accompanied by a rattling sound, due to mucus in the large bronchi or trachea. The general symptoms are not severe, and unless the infant is very young or very delicate no apprehension need be felt as to the outcome. The temperature is generally from 100° to 102° F. for two or three days, then below 100° F. There are a moderate amount of restlessness dependent upon the severity of the cough, usually anorexia, and sometimes vomiting and diarrhœa.

The physical signs in the first stage are dry, sonorous râles over the whole chest. A little later these give place to coarse mucous râles heard everywhere, but especially distinct between the scapulæ and in the infraclavicular regions. On palpation there is usually a marked bronchial fremitus. Often there is not enough dyspnœa to cause recession of the soft parts of the chest. Unless the disease extends to the smaller bronchi and the air vesicles, the illness usually lasts about a week. Coarse râles in the chest may remain for some time after the symptoms have subsided. Relapses are exceedingly common. In a delicate or susceptible child, or in one whose surroundings are bad, one attack is likely to be followed by a succession of others, so that the child may not be really well until warm weather comes. The general health may suffer from the prolonged confinement to the house, although the patient may never have been seriously ill.

2. The severe form (bronchitis of the smaller tubes).—This differs from the preceding variety mainly in the greater severity of all its symptoms. The onset may be like that just described, the severe symptoms not appearing until the patient has been sick two or three days, or they may be severe from the outset. If the latter, it is indistinguishable from broncho-pneumonia. There is cough, dyspnœa, accelerated breathing, fever, and moderate, sometimes severe, prostration. The cough is tighter, and more frequently of a short, teasing character than severe and paroxysmal. There is difficulty in nursing. Dyspnœa may be quite marked and is shown by the active dilatation of the alæ nasi and the recession of all the soft parts of the chest on inspiration. The respirations as a rule are from 50 to 80 a minute. The temperature for the first day or two is usually 101° or 102°, but it may be 103° or 104° F. So high a temperature does not continue unless pneumonia develops. The prostration is in most cases more closely related to the dyspnœa and the rapidity of respiration than to the temperature. Often there is slight cyanosis.

In the beginning the chest is filled with sibilant and sonorous râles, many of them of a musical character. In twelve or twenty-four hours these are replaced by moist râles—coarse or fine, according as they are produced in the large or medium-sized tubes. There are often loud, wheezing râles on expiration. The respiratory murmur is feeble; the resonance on percussion is normal or slightly exaggerated. As the case progresses toward recovery, the finer râles are the first to disappear. The râles are always best heard behind, but they are present all over the chest.

At the onset of such a case it is impossible to say whether the disease will be limited to the medium-sized bronchi or will extend to the smallest bronchi and air vesicles. In young or very delicate infants, and during measles, it is very common for the disease to spread rapidly to the air vesicles. In other cases, usually in infants under six months old, there may develop attacks of respiratory failure or suffocation. These may occur in a severe case at any time, and, because of the infant's inability to empty the tubes of secretion, the dyspnœa steadily increases until the respiratory muscles are exhausted, the inspiratory force being too feeble to overcome the obstruction in the tubes. The symptoms which follow are usually ascribed to pulmonary collapse. I am, however, by no means certain that this is the correct explanation, for in autopsies made in such cases I have usually found the lungs to be the seat of acute emphysema. The clinical picture is a clear one. There is no disposition to cough or cry; the pulse is feeble; the respiration very rapid, superficial, often irregular; the skin cyanotic, and often clammy. Finally, there may be added to the others signs of carbonic-acid poisoning—dulness, apathy, and stupor. Such attacks may come on quite suddenly even in robust infants, and unless the treatment is energetic, even heroic, death often follows in a few hours, being frequently preceded by convulsions.

The usual course of the disease in infants previously in good health is that the severe symptoms continue for two or three days only, after which the temperature falls to 100° or 100·5° F., and gradually becomes normal. The constitutional symptoms usually decline with the temperature, and, except during the first thirty-six hours, they rarely give cause for anxiety. Recovery almost invariably occurs unless the disease extends to the finer bronchi.

Bronchitis is principally to be distinguished from broncho-pneumonia. The differential diagnosis is more fully considered under that disease. The most important points are that in pneumonia the temperature is higher and more prolonged, the prostration greater, the râles very often localized —being heard only behind, often over only one lung—the duration is more protracted, and all the symptoms are more severe.

The bronchitis of older children.—This is not nearly so serious as in infants, because the same danger does not exist of extension of the inflammation to the finer bronchi and air cells.

1. The mild form.—This is very common. The constitutional symptoms are slight, and often entirely absent after the first day. The patient is never sick enough to go to bed. The first symptoms are cough and soreness or a sense of oppression beneath the sternum. The cough is always worse at night. It is at first tight, hard, and racking; later it is loose, and in children over five years old there is usually expectoration—first of white, frothy mucus, but after a few days it becomes more abundant, and of a yellow or yellowish-green colour, from the presence of pus. The physical signs are only coarse râles, at first dry, and later moist, but heard over both sides of the chest, in front and behind. There may be some disturbance of digestion, anorexia, constipation, or diarrhœa. The usual duration of the attack is from one to two weeks. If the patient is not kept indoors the disease may pass into a subacute form, lasting for several weeks as a protracted "winter cough," but without any other important symptoms.

2. The severe form.—The onset is abrupt, with fever, chill, pains in the back, headache, cough, and sometimes pain in the chest. There is a feeling of tightness or constriction beneath the sternum. The onset resembles that of pneumonia, except that the symptoms are less severe. The temperature for the first two or three days ranges between 100° and 103° F. It is generally highest in the first twenty-four hours. The cough resembles that of the mild form, but it is usually more severe. The expectoration is more profuse, and occasionally, in the early stage, it may be streaked with blood.

The coarse râles of the mild form are present, and in addition there are finer râles—at first dry, and later moist—heard all over the chest. Frequently, wheezing râles are heard on expiration. The duration of the attack is ordinarily from two to three weeks, the patient being sick enough to be confined to bed for three or four days only. There is frequently a cough for some time after all physical signs have disappeared. Relapses are easily excited by any indiscretion before the patient has quite recovered.

The prognosis in the primary cases is good, such almost invariably terminating in recovery, and very exceptionally passing into broncho-pneumonia; but this not infrequently happens when the attack complicates measles or pertussis.

Treatment of Bronchitis. *Prophylaxis.*—To remove the predisposition to bronchitis the same means should be employed as those mentioned in acute rhinitis. General measures also should be adopted to build up the health of delicate infants. Those with tuberculous antecedents, and those who are especially prone to pulmonary disease, should if possible spend the winter in a warm climate. In all such patients the systematic administration of cod-liver oil should be continued throughout every cold season. The sleeping apartments of susceptible infants should not be too cold—never below 60° F.—but they should be well ventilated, best by an open fire. Such children should sleep in flannel night clothes, care being taken to see that the feet are always warm. While bronchitis of the large tubes is not *per se* a serious disease, it may become so by extension to the smaller tubes. It is consequently very important in infants and young children that these apparently mild attacks should not be neglected.

General management.—Every young child who has an acute catarrh of the nose, pharynx, larynx, or bronchi should be kept indoors. In every such catarrh accompanied by fever the child should be kept in bed while the fever lasts, even if the temperature does not go above 100·5° F., and is accompanied by no other constitutional symptoms. In infants and young children, many cases of bronchitis result from an extension of an acute rhinitis or laryngitis, hence this precaution is of more importance than everything else in preventing the extension downward of a catarrhal inflammation. A very large number of the cases will recover promptly when no other treatment is employed than to keep the child in bed. The temperature of the room should be about 70° or 72° F. It should be well ventilated and frequently aired, the child being removed to another room while this is done. Infants should not be allowed to lie for hours in the same position as there is a great advantage in changing from the crib to the nurse's arms. Careful attention should be given to feeding and to the condition of the bowels. A cathartic, preferably castor oil, should be administered at the outset. Distention of the stomach and bowels with gas adds greatly to the discomfort of the patient, and may cause serious symptoms.

Abortive measures are rarely successful, for, by the time the physician is summoned, the disease is generally so well established that they are futile. Mild cases may sometimes be cut short by a hot foot-bath, free catharsis, and diaphoresis, especially by the use of one or two doses of phenacetine and Dover's powder (phenacetine two grains, Dover's powder one grain, to a child of three years).

Local applications.—Poultices are objectionable on account of their weight and the difficulty in getting them properly applied. For infants the oiled-silk jacket (page 61) is decidedly preferable. This should be applied in the beginning, and may be worn throughout the attack. It accomplishes all that a poultice does, with much less disturbance to the patient. Counter-irritation is very valuable. In infants the best results are obtained by the frequent use of a mustard paste (page 54). It should be large enough to envelop the chest, and covered by a towel, so as not to soil the oiled-silk jacket or the clothing. The paste is removed as soon as the skin is thoroughly reddened, which will be in from five to ten minutes, according to the strength of the mustard and the condition of the child's skin. The skin should then be powdered and the oiled-silk jacket again pinned snugly about the chest. This may be repeated, according to indications, from two to eight times a day. If properly used, it may be continued for a week without causing any soreness of the skin.

Inhalations.—The value of these is not sufficiently appreciated. They may in the great majority of cases take the place of the administration of drugs by the mouth, a very great advantage in infants. They may be used by means of the croup kettle or vapourizer (pages 60 and 61), the child always being placed in a tent. In the early part of the disease relaxing inhalations, like simple watery vapour or lime-water, may be used. Later turpentine, creosote, terebene, or eucalyptol may be added. Of these, creosote has given me the most satisfaction. Inhalations are to be used for ten or fifteen minutes from four to twelve times a day.

Expectorants.—In infancy this class of drugs may usually be advantageously dispensed with. For older children the relaxing expectorants, especially antimony and ipecac in combination, may be used in the first stage. When the secretion is more abundant, either the alkaline or the stimulating expectorants may be given. Of the former, the best are liquor potassæ, citrate of potassium, and muriate of ammonia; of the latter, creosote, turpentine, terebene, and squills. Small, frequently repeated doses usually give the best results.

Opium.—This should be given very cautiously to young infants, as it is capable of doing great harm. The dry, harassing cough of the early stage sometimes yields to nothing so quickly as to small doses of Dover's powder (e. g., one tenth of a grain every two hours to a child of one year). In the case of infants, late in the disease, and especially in severe cases, opium should be withheld altogether. It disturbs the stomach, constipates the bowels, and, most of all, it greatly depresses the respiration.

Emetics may sometimes be used with advantage when the secretion is very abundant and the cough feeble, but they should be avoided with weak pulse, great prostration, and slight stupor. Syrup of ipecac is the best emetic under these conditions.

Cardiac stimulants.—These are required in most of the severe cases. The best is alcohol. It should be begun as soon as indicated by weak pulse and general prostration. For a child a year old, half an ounce of brandy, diluted with from six to eight parts of water, may be given in each twenty-four hours, in small doses at short intervals.

Respiratory stimulants.—The most valuable drugs are strychnine and atropine. To an infant of six months $\frac{1}{400}$ grain of strychnine and $\frac{1}{1200}$ grain of atropine may be given every two hours. For a short time twice these doses may be used. They are needed only in the most severe cases, and may be used in combination or alternately. An important respiratory stimulant is counter-irritation over the entire body by the mustard paste or hot mustard bath.

The management of mild cases in infants.—In the great majority of cases the disease is self-limited, tending to spontaneous recovery. Often no treatment is needed, except the hygienic measures mentioned. An oiled-silk jacket should be applied. If the cough is excessive, inhalations of creosote or turpentine three or four times a day may be used, or small doses of Dover's powder or phenacetine. The oppression which often comes on toward evening may be relieved by a mustard paste at bedtime. Stimulants are not required. All other drugs may be advantageously omitted, but during convalescence cod-liver oil should be given.

The management of severe cases in infants.—These must be treated very much like cases of broncho-pneumonia. The temperature is rarely high enough to require interference, but the chief danger is due to the inability of the child to get rid of the secretion by the cough. In my experience the two most valuable means of treatment have been the use of inhalations and counter-irritation. The former should be repeated for ten or fifteen minutes every two hours, and for a short period may often be given with advantage every hour. Early in the disease, vapour of plain water or limewater may be used; later, creosote is best. Counter-irritation by the mustard paste should be repeated every three hours, and the oiled-silk jacket worn continuously. Alcoholic stimulants are usually needed in delicate children, and in secondary bronchitis accompanying the infectious diseases. In most of the cases the medication should consist only of cardiac and respiratory stimulants. In strong children the occasional use of an emetic at bedtime is admissible.

Attacks of suffocation and respiratory failure.—The indications here are to get as much blood as possible to the surface and to the extremities, in order to relieve the overloaded right heart, and to compel the child to make full and deep inspiratory efforts. One plan of treatment is to induce frequent crying by flagellation or spanking, this being kept up for several hours. Another is to use alternately hot and cold douches to the chest until some reaction is obtained, and then to follow up this by the occasional use, for a few moments, of a very hot bath (110° F.). Both these means, but especially the first mentioned, are of great value, as I have had abundant opportunity to verify. Other useful measures are the hot mustard bath, the hot mustard pack applied to the entire body, and dry cups. In conjunction with the above means, both heart and respiratory stimulants should be given in full doses. If possible, oxygen should be administered. As these symptoms are liable to recur every few hours for a day or two, a repetition of the treatment will be needed, and if possible the physician should remain with the patient.

If a young infant can be tided over these critical attacks, recovery is probable. After this danger is past, the treatment previously indicated may be pursued. The use of expectorants, particularly the composite cough mixtures containing opium, can not be too strongly condemned in all severe cases of infantile bronchitis.

The management of cases in older children.—In the non-febrile cases confinement in bed is unnecessary, but children should be kept indoors. In the early stage, with hard, dry cough, one of the best remedies is brown mixture (the mistura glycyrrhizæ composita of the U. S. P.). It will be found advantageous in most cases to have the formula made up with one half the usual amount of opium. When the cough is especially hard and dry, a single inhalation may be used at bedtime. In the second stage, muriate of ammonia may be added to the mixture; or terebene, two or three drops upon sugar, may be given four or five times a day. Inhalations of creosote or turpentine should be used.

In the more severe cases the patients should be kept in bed and counter-irritation to the chest employed. In the beginning the liquor ammoniæ acetatis and spiritus ætheris nitrosi may be given for their effect upon the skin and kidneys. For the general discomfort, pain, headache, etc., nothing is better than phenacetine and Dover's powder (two grains of the former to one half grain of the latter to a child of five years), repeated every three to six hours. Heroin is a valuable remedy for the relief of troublesome cough, but should be used with caution; not more than gr. $\frac{1}{48}$ should be given every three hours to a child of five. All patients should be kept in bed as long as the temperature is above normal.

The protracted cough of convalescence.—It often happens, both in infants and in older children, that after all physical signs and constitutional symptoms have disappeared, a cough continues sometimes for weeks. Expectoration is scanty, or is wanting altogether; the cough is hard, dry, often paroxysmal, and in some cases occurs at night only. For this condition the best remedies are quinine, cod-liver oil, and creosote. The last named may easily be given to young infants as well as to older children, in combination with liquid beef peptonoids.* It may be also used in pill form or by inhalation. These measures may be tried alternately or in combination. Where they are not effective a change of climate should be advised.

FIBRINOUS BRONCHITIS (BRONCHIAL CROUP).

Fibrinous bronchitis is seen in diphtheria, usually as an extension from the larynx or trachea. There is, however, another form of bronchitis attended by a fibrinous exudate, which occurs as a primary disease. This is very rare in children. Weil has, however, collected twenty cases of the primary form. The etiology is obscure. It is seen at all ages, from infancy up to puberty, and it may be either acute or chronic. From the cases thus far reported it would appear that the acute form is relatively more common in children than in adults. The disease may be confined to certain branches of the bronchial tree, or it may affect all the bronchi, even to the minute subdivisions. The fibrinous membrane is found loose in the tubes or adherent. There are generally associated other pulmonary changes, such as emphysema, areas of atelectasis or of broncho-pneumonia.

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* A preparation put up by the Arlington Chemical Company, and a very palatable way of giving creosote.

The acute form somewhat resembles ordinary catarrhal bronchitis. The diagnostic features are the severity of the dyspnœa and the expectoration of tube casts from the larger bronchi, or elongated cylinders from the smaller ones, the former resembling macaroni, the latter vermicelli. The expectorated masses are often in balls or plugs, and their peculiar character is not recognised until they are placed in water. The casts are dissolved by alkalies, especially by lime-water. After the expulsion of a large cast, improvement in all the symptoms occurs. These, however, return as the exudate reappears. The ordinary duration of acute cases is from one to three weeks.

In the chronic form there are no constitutional symptoms, but only dyspnœa and cough, often recurring in paroxysms, with the expectoration of fibrinous casts. The patient may have these attacks at intervals of a few days or weeks, extending over a period of months, or even years. There are no characteristic physical signs. The diagnosis rests upon the peculiar character of the expectoration. The prognosis in acute cases is unfavourable, the mortality being 75 per cent (Weil). Chronic cases are not dangerous to life.

Treatment.—This is quite unsatisfactory. To loosen the membrane and facilitate its expulsion, the most efficient means are inhalations of the vapour of limewater and the internal administration of pilocarpine. Occasionally emetics are of value. Improvement in some of the chronic cases has resulted from the use of iodide of potassium.

CHRONIC BRONCHITIS.

Chronic bronchitis is not a common disease in children, particularly in young children, one reason being that chronic emphysema, so frequently an associated condition in adults, is rare in early life. Chronic bronchitis always accompanies chronic pulmonary tuberculosis and chronic interstitial pneumonia, with or without the occurrence of bronchiectasis. It is seen in chronic cardiac disease, especially with lesions of the mitral valve. It may occur as a late symptom of hereditary syphilis. Excluding the varieties mentioned, it usually follows attacks of acute bronchitis, the process becoming chronic because of the patient's constitutional condition or his unhygienic surroundings. The acute attack may be primary, but it often follows measles and whooping-cough. Rickets, general malnutrition, and lymphatism are the constitutional conditions in which acute bronchitis is most likely to pass into the chronic form. Deformities of the chest, the result either of rickets or of Pott's disease, are occasionally a cause.

Symptoms.—The only constant symptom is cough, which is persistent, obstinate, and nearly always worse at night or early in the morning. It often occurs in paroxysms strongly suggestive of pertussis. Expectoration is not generally abundant, but in older children it is usually present, and in a few cases it is profuse. A copious morning expectoration of fetid pus or muco-pus indicates bronchiectasis. There is no fever, little or no dyspnœa, and although the patients are thin they are not emaciated, and in many cases the general health is not much affected. There may be coarse mucous râles, or no physical signs whatever. The duration of the disease is indefinite, depending upon the cause. All these patients are better in summer and worse in winter, and suffer frequently from exacerbations of acute or subacute bronchitis.

The diagnosis is to be made mainly from pertussis and tuberculosis. From mild attacks of pertussis the diagnosis may be impossible except by the course of the disease. Tuberculosis may be suspected if the thermometer shows regularly a slight evening rise of temperature, if there is much anæmia, and steady loss of flesh. A positive diagnosis can be made only by the discovery of tubercle bacilli in the sputum.

Treatment.—The first indication is to treat the primary disease. In cardiac cases digitalis is the best remedy, and all sedatives are to be avoided. Attention should be directed to the general condition—rickets, malnutrition, and lymphatism each receiving its appropriate treatment. In most cases a general tonic plan of treatment is best, particularly the continuous use of cod-liver oil. In many cases a change of climate is the only thing which is really curative. For the relief of cough, opiates are to be avoided as much as possible. The main reliance should be upon potassium iodide, heroin, creosote and terebene, the last two being given both by mouth and by inhalation.

REFLEX COUGH—NERVOUS COUGH.

Strictly speaking, all cough is reflex and of nervous origin. The term "reflex cough" is, however, commonly used to denote that which occurs without any evidence of disease in the larynx, trachea, bronchi, lungs, or pleura. On account of the close connection through the vagus and its branches between the mouth, ear, throat, stomach, and thoracic organs, it is possible for cough to be produced by many forms of irritation in these organs or cavities. Clinically, the following varieties of nervous cough are observed:

1. That dependent upon rhino-pharyngeal irritation. This is the most frequent form, and is sometimes caused by an elongated uvula, but is usually due to adenoid growths of the pharynx, though enlargement of all the lymphoid tissues of the neighborhood no doubt have a part. The cough is generally excited by an accumulation of mucus in the posterior pharynx, and is dry, tickling, or hemming in character. It occurs chiefly at night and in some patients only then; it may begin soon after the child falls asleep and continue the greater part of the

night, often for months, especially in the cold season. Formerly, such coughs were often ascribed to disorders of digestion, to dentition, to inflammation of the ears, etc.

2. Cardiac cough. This is usually associated with mitral disease, and is due to pulmonary congestion. The cough is dry, hard, and often severe.

3. The variety which occurs usually about the time of puberty, and often associated with anæmia, chorea, or spinal irritation. It is a short, hacking, or teasing cough, sometimes very distressing, and it seems to be a manifestation of extreme nervous irritability.

4. The periodical night cough, which is generally ascribed to irritation of the vagus or its branches by enlarged, sometimes caseous, lymph nodes of the tracheo-bronchial group. This often occurs in severe paroxysms, the character of which is very much like pertussis. The attacks are apt to come on about the middle of the night and last for several hours. Vomiting is rare. The cough may recur regularly every night for months. On account of the loss of sleep the patient's general health may be considerably undermined.

5. A very similar cough may occur in connection with abscesses in the posterior mediastinum due to Pott's disease.

Symptoms and Diagnosis.—These cases are not common in infants, but are quite frequent in older children. In nearly all the varieties the cough is worse at night, and in many it may be confined to that time. The influence of habit is often seen, the attacks coming on regularly at certain periods. The general health may not be affected, except from the disturbance of sleep. The diagnosis between the different forms is often very difficult. The precise cause in a given case is discovered only by a careful examination of the ear, nose, pharynx, heart, stomach, lungs, and a consideration of the patient's general condition. The existence of enlarged or tuberculous bronchial glands may be suspected in patients of tuberculous antecedents, in those who have previously suffered from measles, pertussis, or repeated attacks of bronchitis, and when the cough is very severe and paroxysmal. A similar group of symptoms may exist with abscesses from Pott's disease. In either of these conditions there may be attacks of suffocation.

Treatment.—Opium and expectorants are not indicated, and inhalations are of little value. The only successful treatment is that which is directed to the cause of the disease. If no cause can be found, and the cough appears to be of purely nervous origin, the best results follow the use of the bromides or the administration of antipyrine at bedtime.

ASTHMA.

Asthma may be defined as a vaso-motor neurosis of the respiratory tract. It is characterized by attacks of severe spasmodic dyspnœa, which may be preceded, accompanied, or followed by bronchial catarrh of greater or less severity. In the asthmatic attacks of infancy the catarrhal element is very prominent, and these cases present quite a different clinical picture from the disease as seen in older children, which differs in no essential points from the asthma of adults.

Writers differ very much in their statements regarding the frequency of asthma in early life, mainly because of a want of agreement in regard to what shall be included under this term. The asthmatic attacks of infants are considered by some as a stage of bronchitis, by others as distinct from that disease. Typical attacks resembling those of adult life are rare in children, and extremely so before the seventh year. However, of 225 cases of asthma reported by Hyde Salter, the disease began before the tenth year in nearly one third the number.

Etiology.—The general or constitutional causes are the same in children as in adults. Asthma may be hereditary. It occurs especially in children whose antecedents have suffered from gout or from other neuroses. The local cause may be any form of irritation in the nose or pharynx—hypertrophic rhinitis, adenoid growths of the pharynx, hypertrophied tonsils, or elongated uvula—or in the bronchial mucous membrane, as a result of previous attacks of acute bronchitis. It is probable that it may also be caused by the irritation of enlarged bronchial glands. In susceptible persons a paroxysm may be excited by cold or damp air, indigestion, constipation, or the inhalation of various irritating substances, such as dust, the pollen of certain plants, etc. First attacks of asthma in children are apt to follow bronchitis.

Symptoms.—Four quite distinct clinical types of asthma are seen in children: (1.) Cases which in their onset simulate attacks of capillary bronchitis. (2.) Those in which asthmatic symptoms follow an attack of bronchitis, continuing for weeks or months, but not necessarily recurring. (3.) Hay fever, or the periodical form which occurs every summer. (4.) That which resembles the ordinary adult asthma, with the nervous element predominating. The prominence of the catarrhal symptoms is characteristic of all asthma in children, the first two varieties mentioned being peculiar to early life.

Attacks resembling capillary bronchitis.—These cases are rare, but may be seen even in infants. The onset is sudden, with moderate fever, incessant cough, severe dyspnœa, and sometimes symptoms of suffocation —cyanosis, prostration, and cold extremities. The chest is filled with sonorous, sibilant, and soon with subcrepitant râles. Instead of running the usual course of bronchitis of the finer tubes, the symptoms may pass away very rapidly, and in forty-eight, sometimes in twenty-four hours the patient may be quite well. It is only by the course of the disease and by recurring attacks that their true nature can be recognised. In infants this form of asthma may be fatal.

Cases following attacks of bronchitis—Catarrhal asthma.—This form is not uncommon, though it is frequently designated by some other term than asthma—sometimes as spasmodic bronchitis, or catarrhal spasm of the bronchi. The symptoms are, however, indistinguishable from asthma, and they evidently belong in the same category. This form is usually seen in infants, being rare after the third year. Many of the patients are rachitic; others have large tonsils, or adenoid growths of the pharynx; while in still others there is every reason to suspect the presence of large bronchial glands. Usually there is nothing peculiar about the antecedent bronchitis; in most cases it is not especially severe, and is limited to the larger tubes. The febrile symptoms subside in a few days, but the cough continues, as do also the dyspnœa and wheezing. When the symptoms are fairly established they are very uniform and characteristic. The respiration is accelerated, usually to 50 or 60, sometimes to 70 or 80, a minute. The temperature from time to time may be very slightly elevated, or it may remain normal. The respiration is noisy, laboured, and accompanied by distinct wheezing.

On auscultation, there is prolonged expiration accompanied by loud, wheezing râles, either sonorous, sibilant, or musical, and occasionally moist râles are heard. In cases which have lasted some time a moderate amount of emphysema can be inferred from prominence of the infraclavicular regions, and exaggerated resonance over the chest in front.

These symptoms and signs may continue for three or four weeks only, and gradually wear off, or they may last as many months—if they begin in the winter or spring, often continuing until the middle of the summer. While they are constantly present, they vary in intensity from time to time, being usually much worse at night. The symptoms are always increased by exposure to a cold, damp atmosphere, by any fresh accession of bronchitis, and often by trivial digestive disturbances. The usual duration of the cases I have seen has been two to six weeks. The cough is not usually severe, and expectoration in most cases is absent. The general health is often but little affected. With recovery from the asthmatic symptoms the emphysema usually disappears gradually, although I have seen one severe case in which it persisted.

What proportion of these children afterward develop ordinary asthma, from personal experience I am unable to say. Some undoubtedly do, but in others which I have been able to follow, recovery has seemed to be permanent. This would appear more likely in those cases closely associated with rickets, or with other causes which disappear spontaneously with time or as a result of treatment.

Hay fever.—This is very rare before the seventh, and but few well-marked cases are seen before the tenth year. In its clinical aspects it does not differ essentially from the disease as seen in adults, except possibly by the greater prominence of the bronchial catarrh.

Ordinary attacks of the adult type.—These usually occur at intervals of a few weeks or months, depending upon the nature of the exciting cause. The beginning is usually at night, with dyspnœa, a short, dry cough, and loud, wheezing respiration. Deep recession of the soft parts of the chest is seen, as in laryngeal stenosis. There is prolonged expiration, accompanied by loud, sonorous, sibilant and wheezing râles, and the vesicular murmur is very feeble. Later, moist râles may be heard. After many attacks emphysema is present. This occurs more rapidly than in adults, and may be extreme, giving rise in marked cases to serious thoracic deformity. On account of the loss of sleep and interference with nutrition, the general health may become seriously impaired.

Diagnosis.—Typical attacks of asthma are easily recognised. Some of the catarrhal forms seen in infancy, however, present great difficulty, and a positive diagnosis may be impossible except by the progress of the case.

Prognosis.—This is best in the cases of catarrhal asthma in infants, and in older patients when it depends upon some local cause which can be removed, as when the disease is due to reflex nasal or pharyngeal irritation. In the majority of other cases, asthma is likely to become chronic unless the child is removed to some climate in which the attacks do not occur. The younger the child, the shorter the duration of the disease, and the less marked the hereditary tendency, the better the prognosis.

Treatment.—The nose and the rhino-pharynx should be carefully examined in every case of asthma, and any pathological condition there present should receive attention as the first step in the treatment. Special importance, in children, should be attached to the removal of adenoid growths of the pharynx. During attacks, the best means of relieving the symptoms is the inhalation of fumes of nitre paper or stramonium leaves. Most of the proprietary remedies (Papier de Fruneau, Himrod's cure, and Kidder's pastilles) contain these ingredients. The two preparations last mentioned are by most children particularly well tolerated. The sleeping room may be filled with the fumes of these substances, or the child may be placed in a tent into which the fumes are introduced. Emetics should be employed when the attack is brought on by indigestion. Lobelia is the most satisfactory remedy for this purpose. To prevent the recurrence of night attacks, nothing in my experience has been so valuable as a full dose of antipyrine at bedtime—four grains at five years and six grains at ten years. Between the attacks the main reliance should be upon the syrup of hydriodic acid and potassium iodide, which are to be given for a long time in moderate doses. Tonics are to be used in nearly all cases. Those especially valuable in asthmatic patients are cinchonidia and arsenic.

In the cases of catarrhal asthma following bronchitis, expectorants and ordinary cough remedies are useless. Cod-liver oil and the iodide of potassium are valuable in some of the cases. Others are greatly relieved by the regular use of creosote inhalations several times a day, with a nightly dose of antipyrine. The fumes of nitre and stramonium often afford no relief, and sometimes the cases are made distinctly worse by them. The best of all measures is to send the child at once to a warm, dry climate.

For all children who have had repeated attacks, whether in the form of hay fever or the ordinary variety, the most important thing is removal to a place where they do not have the disease, and a residence there long enough to break up the tendency to recurrence. This will usually require at least three or four years. The region best suited to most asthmatics is one which is high, dry, and moderately warm. Patients often suffer less in cities than in the country. If taken early, asthma in children is frequently curable by these means; if neglected, the disease is almost sure to continue until adult life.

CHAPTER IV.

DISEASES OF THE LUNGS.—(*Continued.*)

PNEUMONIA.

IN early life the lungs are more frequently the seat of organic disease than any other organs in the body. Pneumonia is very common as a primary disease, and ranks first as a complication of the various forms of acute infectious disease of children. It is one of the most important factors in the mortality of infancy and childhood (page 41).

Cases of acute pneumonia are divided, from an anatomical point of view, into two principal groups: (1.) Broncho-pneumonia, also known as catarrhal and as lobular pneumonia. (2.) Lobar pneumonia, also known as croupous and as fibrinous pneumonia. These differ from each other as to the products of inflammation, the distribution of the disease in the lung, and somewhat as to the parts involved and the nature of the changes in them.

In broncho-pneumonia the large bronchi are the seat of a superficial inflammation, while in those of small size the entire bronchial wall is affected; the exudation into the air vesicles is mainly cellular, being made up of epithelial cells, leucocytes, and red blood-cells (Fig. 86), fibrin being either absent, or present only in small amount. In many cases there are marked changes both in the alveolar septa and in the interstitial tissue of the lung; resolution is often imperfect, and there is a strong tendency of the inflammation to pass into a chronic form, involving the connective-tissue framework of the lung. The lesion is widely and often irregularly distributed, usually being most marked in the vicinity of the small bronchi from which the inflammation spreads, and in the most superficial lobules of the lung.

In lobar pneumonia, bronchitis, when present, is usually superficial, the walls of the bronchi being very slightly or not at all affected; the same is true of the alveolar septa. The principal product of the inflammation is fibrin (Fig. 87), which fills the alveoli and the terminal bronchi, the cells being relatively few and chiefly leucocytes. The process is usually sharply circumscribed, involving an entire lobe or a part of a lobe. In most cases it clears up rapidly and completely, there being but little tendency to involve the framework of the lung in a chronic process.

While in typical cases the two forms of inflammation are quite distinct, there are seen many intermediate forms which partake of the characters of both, and one may be in doubt, even after a microscopical examination, into which group to place a case. It not infrequently happens that both varieties of pneumonia are present in different parts of the same lung or in both lungs at the same time. These mixed forms are especially frequent during the second and third years; but during the first year, and after the third, the types are usually well marked.

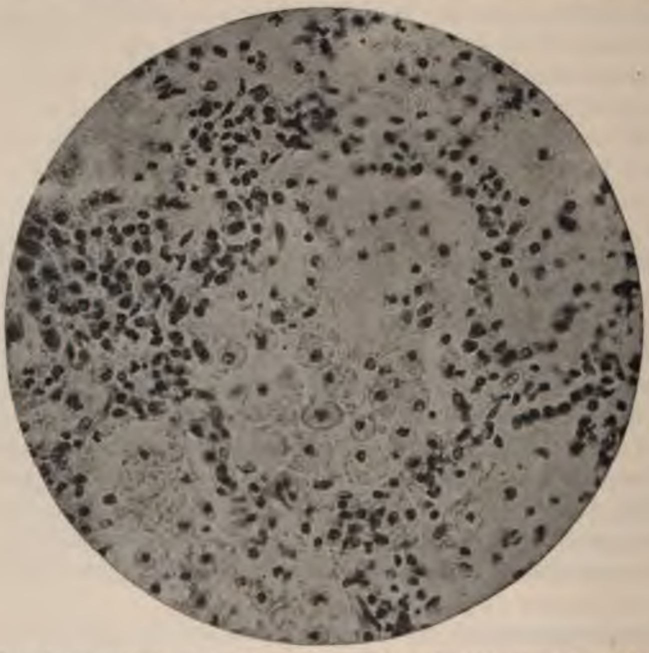

FIG. 86.—Broncho-pneumonia. The picture shows at its centre one entire air vesicle, and at its margin parts of four or five other vesicles; they are filled with large epithelial cells having small nuclei. There are also seen leucocytes with intensely black nuclei and narrow protoplasm. Between the cells is a finely granular material, which is the exudation fluid coagulated during the hardening process. The alveolar septa are somewhat infiltrated.—From Karg and Schmorl.

The following table shows the relative frequency of lobar and broncho-pneumonia in three hundred and seventy cases,* nearly all taken from one institution (New York Infant Asylum). There are included all the cases of acute primary pneumonia occurring during a period of seven years:

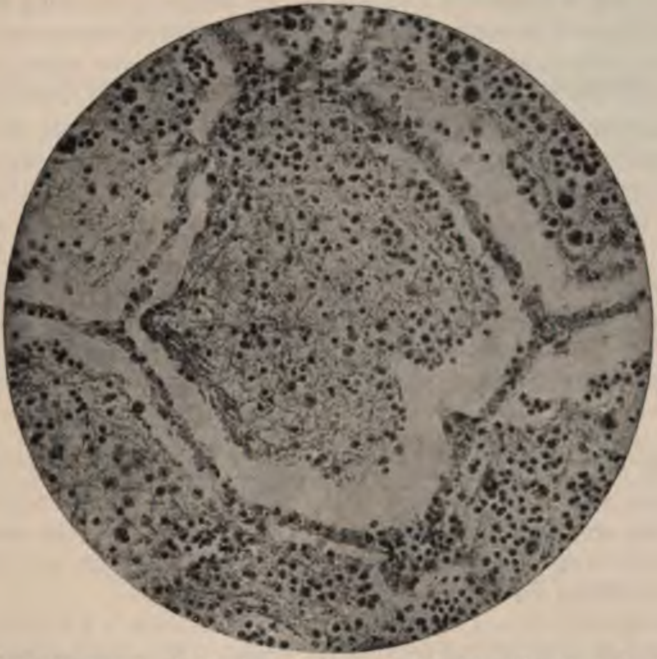

FIG. 87.—Lobar pneumonia. In the air vesicle shown in the picture there is a firm, close network of fibrin, in the meshes of which are leucocytes. At the lower part the exudation has contracted away from the wall in consequence of the process of hardening.—From Karg and Schmorl.

Under six months,	broncho-pneumonia,	73	cases;	lobar	pneumonia,	11	cases.
Six to twelve "	"	96	"	"	"	29	"
Second year,	"	73	"	"	"	40	"
Third "	"	19	"	"	"	23	"
Fourth "	"	0	"	"	"	6	"
Totals,	"	261	"	"	"	109	"

Thus it will be seen that, of the cases of acute pneumonia occurring during the first two years, 25 per cent were lobar and 75 per cent were broncho-pneumonia.

When we come to a consideration of the micro-organisms with which the different forms of pneumonia are associated, we find that they do not correspond to the anatomical varieties. Lobar pneumonia is regularly associated with the presence of the pneumococcus (micrococcus lanceolatus), which is frequently found pure. In broncho-pneumoñia no single form is regularly present. In the primary cases the pneumococcus is most frequently found, and in many cases it is alone. In the secondary cases there is almost always mixed infection. In measles and diphtheria the streptococcus is usually present, such cases being generally of a very severe type. In other secondary cases there is found the staphylococcus, and sometimes Friedländer's bacillus. Each of these varieties of bacteria may be found alone, but they are often associated, and with any of them may be found the pneumococcus, or other specific germs, most frequently the bacillus of influenza, diphtheria, or tuberculosis.

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* The division was here made according to the predominant clinical or pathological features. Most of the doubtful cases were classed as broncho-pneumonia.

Why the same cause—the pneumococcus—in one case produces broncho-pneumonia and in another lobar pneumonia, is in part owing to the difference in the structure of the lung at the different ages—that of infancy being more bronchial, and that of older children more vesicular. Another reason is to be found in the constitution of the patient: in the very young and in feeble and delicate children, the process tends to become diffuse and the products are chiefly cellular; in those who are older and more vigorous it is likely to be circumscribed, with fibrin as its chief product; in the intermediate ages and intermediate conditions the types are often mingled.

Etiologically as well as clinically, lobar pneumonia is a single disease, usually running a regular self-limited course. Broncho-pneumonia, on the other hand, includes a number of quite distinct diseases, which are not only etiologically but clinically different. Sometimes when it is due to the pneumococcus it has more features in common with lobar pneumonia than with cases of broncho-pneumonia due to another kind of infection, such as the streptococcus.

The immediate source of infection of the lungs is the mouth, the nose, or the pharynx. All the forms of bacteria found in pneumonia are found in these cavities, some of them constantly, others only at certain times, especially during an attack of any of the acute infectious diseases. What part direct contagion plays in the spread of pneumonia can not be settled without fuller data than at present exist. There seems to be no doubt, from clinical observations alone, that the secondary forms, especially those complicating measles and diphtheria, are sometimes communicated in this way. This is probably not often true of primary cases except in hospitals for infants where the rapid development of case after case in the same ward can not be well explained on any other hypothesis.

The different forms of pneumonia which will be considered are: (1) Acute broncho-pneumonia. (2) Acute fibrinous pneumonia. (3) Acute pleuro-pneumonia. (4) Hypostatic pneumonia. (5) Chronic broncho-pneumonia.

Tuberculous broncho-pneumonia will be discussed in the chapter devoted to Tuberculosis.

ACUTE BRONCHO-PNEUMONIA.

Synonyms: Catarrhal pneumonia, lobular pneumonia, capillary bronchitis.

This is essentially the pneumonia of infancy. Under two years, the great majority of the cases of primary pneumonia are of this variety, and throughout childhood nearly all the cases of secondary pneumonia. The term broncho-pneumonia describes a lesion rather than a disease, several quite distinct forms of infection being included under this head. Its mortality is high, because of the tender age of the patients in which the primary cases occur, and also because when secondary it complicates the most severe forms of the acute infectious diseases of children.

Etiology.—*Age.*—The 426 cases of broncho-pneumonia of which I have notes occurred as follows:

During the first year	224 cases,	or 53 per cent.
" " second year	142 "	" 33 " "
" " third "	46 "	" 11 " "
" " fourth "	10 "	" 2 " "
" " fifth "	4 "	" 1 " "
	426	100

After four years broncho-pneumonia is very infrequent as a primary disease, although it is seen throughout childhood as a complication of the infectious diseases.

Sex.—In the primary cases males are more frequently affected than females, the proportion being five to four. In the secondary cases the sexes are about equally affected.

Season.—Of the cases referred to, 38 per cent occurred during the winter months, 31 per cent during the spring, 13 per cent during the summer, and 18 per cent during the autumn. While, therefore, nearly 70 per cent of the cases occurred in the cold months, broncho-pneumonia is seen throughout the year.

Previous condition.—Broncho-pneumonia affects all classes, but is most frequent in children having poor hygienic surroundings, especially in inmates of institutions, and in those previously debilitated by constitutional or local disease. In 246 consecutive cases of primary pneumonia, 110 were in good condition prior to the attack, and 126 were delicate, rachitic, or syphilitic.

Previous disease.—The following table gives a good idea of the conditions with which acute broncho-pneumonia is most frequently seen; 443 cases were classed as follows:

Primary *	164
Secondary to bronchitis of the large tubes	41
Complicating measles	89
" pertussis	66
" diphtheria	47
" acute ileo-colitis	19
" scarlet fever	7
" influenza	6
" varicella	2
" erysipelas	2
	443

A large number of the patients had previously suffered from one or more attacks of bronchitis, and fifteen previously had broncho-pneumonia.

As an exciting cause, exposure to cold must still be classed among the potent factors of primary pneumonia.

Bacteriology.—Much light has already been thrown upon broncho-pneumonia by bacteriology, but many points still remain to be settled.

In 1892 Netter published a report upon 42 cases. He did not separate the primary and secondary cases. Of 25 cases in which but one form of bacteria was found, the pneumococcus was present in 10, the streptococcus in 8, the staphylococcus in 5, and Friedländer's bacillus in 2. In the 17 cases of mixed infection, the streptococcus was present in 15, the pneumococcus in 9, the staphylococcus in 8, and Friedländer's bacillus in 4.

In 1897 Pearce (Boston) published a report upon 82 cases of broncho-pneumonia complicating various infectious diseases: 62 were associated with diphtheria alone: 9 with diphtheria and scarlet fever: 2 with diphtheria and measles: 9 with scarlet fever alone. In the 73 diphtheria cases the Klebs-Loeffler bacillus was present in 63, and in 17 it occurred alone. The streptococcus was present in 38 cases, 27 of these being in diphtheria uncomplicated by scarlet fever or measles, and in 7 of these it was the only organism found. The staphylococcus aureus was present in 26 cases, but never alone. It is surprising that the pneumococcus was present in but 8 cases, 5 of these being scarlet fever.

Dr. Martha Wollstein has studied bacteriologically one hundred cases of broncho-pneumonia. Most of these were under my personal observation in the wards of the Babies' Hospital. Her results have been published in the Journal of Experimental Medicine, vol. vi, 1904. All of these children were under three years old: in 33 the pneumonia was primary and in 67 secondary. Of the latter, 25 complicated tuberculosis, 19 marasmus, 5 diphtheria, 3 measles, 3 malaria, 4 septicæmia, 2 pyæmia, 2 meningitis, 3 intestinal disease, 1 abscess of the brain.

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* It is probable that a number of cases complicating influenza were included among these primary cases.

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direction the lung is cut, there are seen surrounding the small bronchi, zones of pneumonia (Fig. 88). In other cases the process seems to begin almost at the same time in the small bronchi and the air vesicles, as both are found involved, even when death occurs within a few hours of the first symptoms.

There are, however, cases in which the parts of the lung affected bear no relation to the bronchi—where there are found simply smaller or larger areas of pneumonia irregularly scattered through the lung, usually near the surface (Plate XI). From the distribution of the lesions such cases might better be termed lobular than broncho-pneumonia.

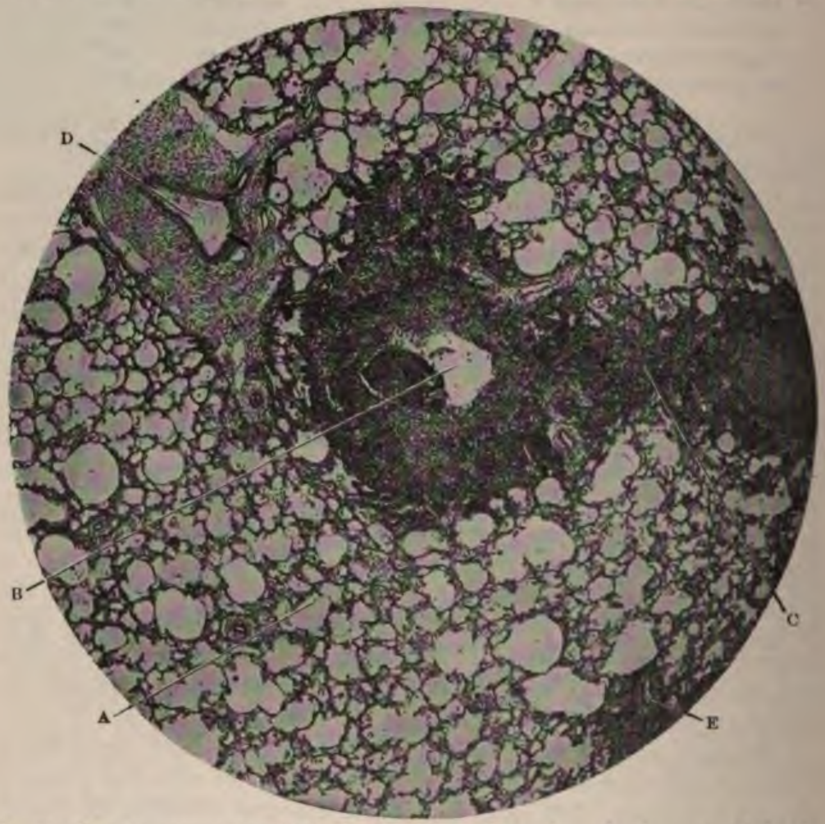
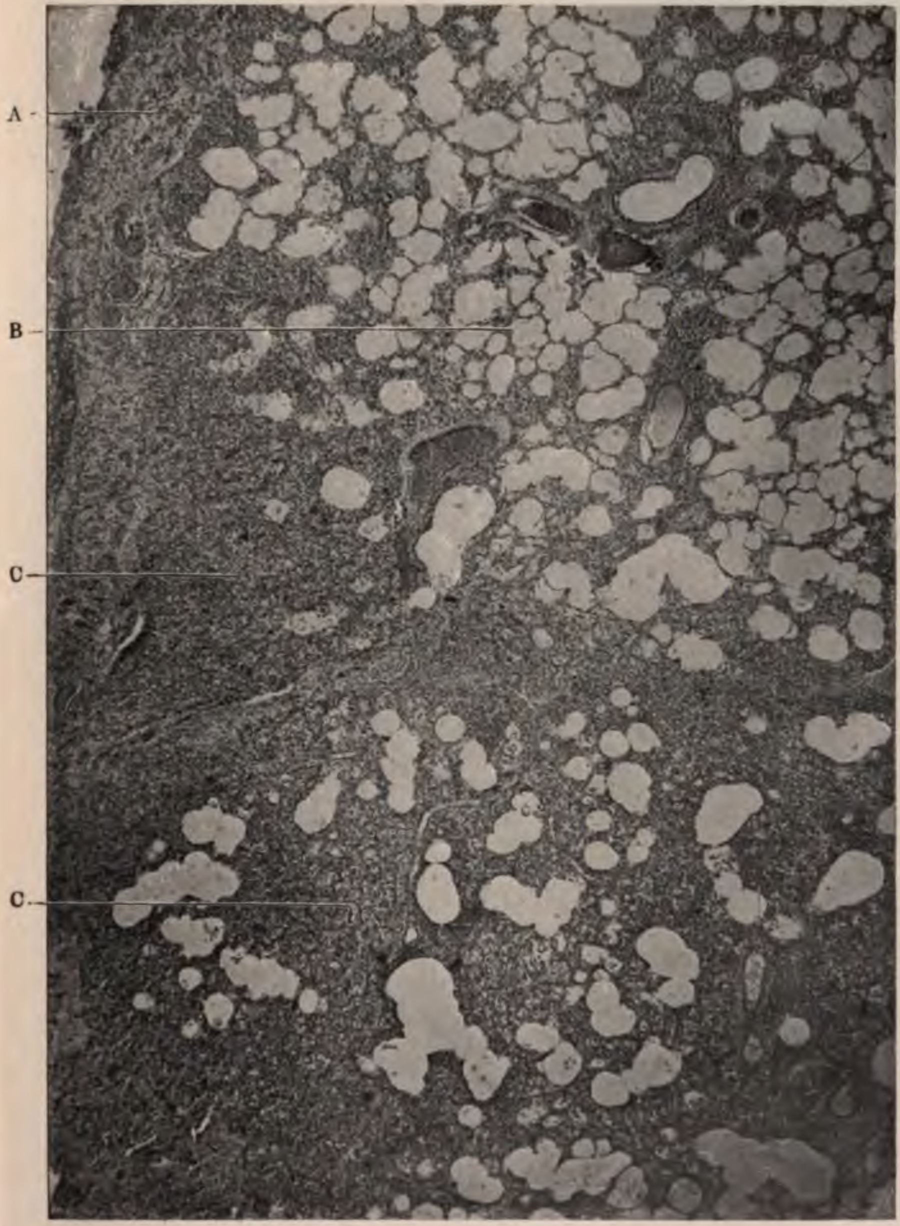


FIG. 88.—Broncho-pneumonia, with thickening of a small bronchus. In the centre of the picture is seen a small bronchus, B, which is cut somewhat obliquely, so that the degree to which its wall, C, is thickened is well shown. It is partially filled with pus, its mucous membrane is nearly destroyed, and its walls greatly thickened from infiltration with leucocytes. This infiltration extends to the lung tissue in the neighbourhood; it forms a peri-bronchitic zone of pneumonia. Elsewhere in the picture the lung tissue, A, is practically normal. D is a small blood-vessel. E is another smaller bronchus. Throughout the lung everywhere accompanying the small bronchi similar changes were seen, in addition to which there were present some large areas of consolidation. The disease was of four and a half weeks' duration; the child, five months old.

Much has been said in the past about pulmonary collapse from ob-

PLATE XI.



ACUTE BRONCHO-PNEUMONIA.

Primary pneumonia in a child two years old, showing the irregular distribution of the hepatization and its incomplete character. A is the pleura somewhat thickened; B, lung tissue which is practically normal; C C are hepatized areas, scattered through which are groups of air vesicles still containing air. (Slightly magnified.)

struction of the small bronchi, as a condition antecedent to this form of pulmonary inflammation. So far as my own observations go, there has been adduced but little evidence that this is the rule, or, indeed, that it often occurs. Even in autopsies made very early in the disease, but little collapse was found, most of the cases supporting the view of Delafield, that when the disease extends from the bronchi to the air cells it involves those surrounding the tube quite as regularly as those to which the tube leads.

The following observations are made from a study of 170 autopsies of which I have records, microscopical examinations having been made in about one third of the number.

Seat of the disease.—In 82 per cent of the autopsies extensive disease was found in both lungs. The parts most affected were the lower lobes posteriorly; next to this the posterior part of both the upper and lower lobes. The left lower lobe was more extensively diseased than the right in over two thirds of the cases. Only a single lobe was involved in but 9 per cent of the cases. It is not common for the disease to be situated in the anterior portion of the lung only, but when this occurs the right apex is the most frequent seat.

Just as the clinical symptoms of broncho-pneumonia follow no regular type, so the pathological process does not pass through a regular order of changes such as are seen in lobar pneumonia. There are a certain number of cases which appear to follow tolerably well-defined stages of congestion, red hepatization, gray hepatization, and resolution; but the disease may be arrested at any of the stages and the case recover, or death may occur at any stage and there may be found at autopsy different portions of the lung representing all the stages mentioned. In considering, therefore, the lesions of broncho-pneumonia, it seems best to describe the condition in which the lungs are found at the various periods when death is likely to occur, rather than to attempt to describe the different stages of the disease, as in lobar pneumonia.

1. *The acute congestive form* (*acute red pneumonia*).—This is the condition in which the lung is usually found if death occurs during the first two or three days of the disease. In the cases severe enough to cause death in the first twenty-four hours, very little can be seen by the naked eye except acute congestion. The vessels of the pleura are distended, and there may be small superficial hæmorrhages. Both lower lobes are usually heavy and dark-coloured. There is to the naked eye no consolidation. All, or nearly all, the lung can be inflated. On section, there is found intense congestion with some œdema. When the process has lasted a little longer the affected areas are more sharply defined. These, usually the posterior portions of both lungs, are of a brownish-red colour, and appear partially hepatized, although with a little force they may in most cases be inflated. After section, pus and mucus flow from the divided bronchi, and the whole lung may be more or less congested or œdematous.

The microscope alone reveals the fact that these are not cases of simple pulmonary congestion or bronchitis of the finer tubes. In one case in which death occurred twelve hours from the first symptoms, I found well-marked evidences of inflammation of the air vesicles. In these hyper-acute cases, the microscope shows great distention of all the small blood-vessels of the affected area, and small or large extravasations of blood just beneath the pleura, into the alveoli (Fig. 89) and interstitial tissue of the lung. In some cases these hæmorrhages form the most striking feature of the lesion. The air vesicles are partially, some almost completely, filled with red blood-cells, swollen and desquamated epithelial cells, and a few leucocytes (Fig. 86). The red blood-cells predominate. The inflammation may be diffuse, involving nearly a whole lobe, or in small areas in the

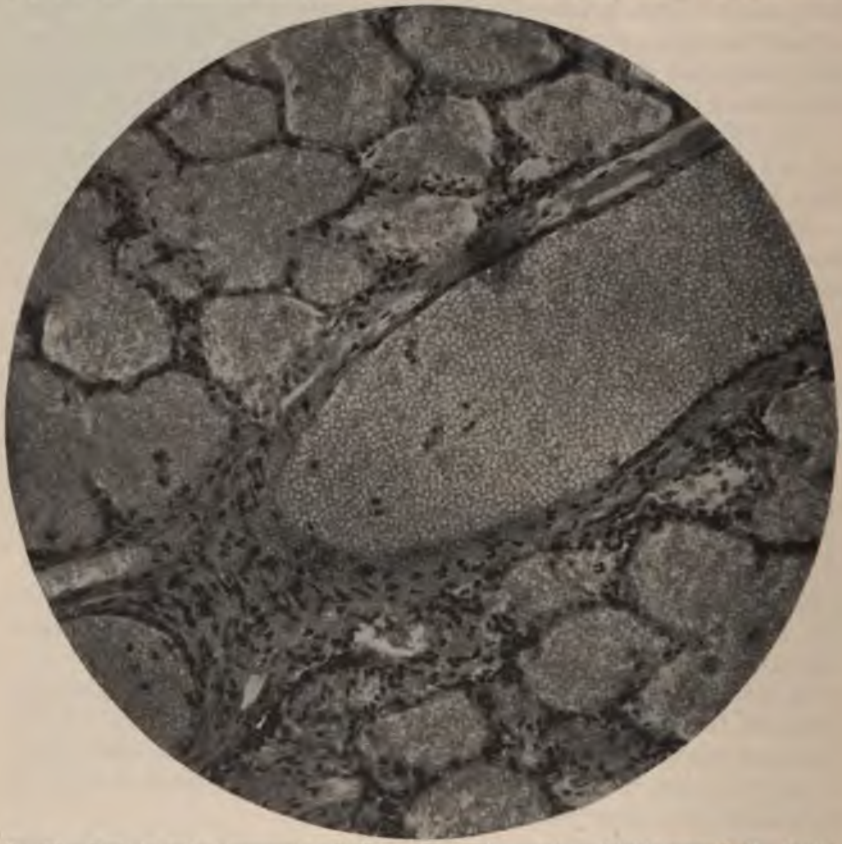


FIG. 89.—Acute broncho-pneumonia with intra-alveolar hæmorrhage (highly magnified). In the picture is shown a small vein, which, as well as the surrounding alveoli, is filled with blood-cells. In other respects the lung shown is normal. This is from the border of a consolidated area. Child fifteen months old: pneumonia of ten days' duration, with a severe exacerbation forty-eight hours before death, temperature 106° F. Extensive hæmorrhagic areas were scattered through the lung most affected.

neighbourhood of the small bronchi. The mucous membrane of the large and small bronchi is the seat of catarrhal inflammation, and the walls of the latter are infiltrated with round cells.

When the process has lasted from twenty-four to forty-eight hours all the changes described are more marked, but the red colour of the inflammatory products still persists. Such cases give during life only the signs of congestion and bronchitis.

2. *The mottled, red and gray pneumonia.*—This is the usual appearance when the disease has lasted somewhat longer, and is found in most of the cases dying between the fourth and fourteenth days. There are usually at this time quite large areas of consolidation, sometimes affecting nearly an entire lobe, so that at first sight the case may resemble lobar pneumonia. This is sometimes described as the "pseudo-lobar" form. The extent of these areas depends largely upon the duration of the disease. In most cases there is pleurisy over the consolidated portions. This may cause the lung to adhere to the chest wall, the firmness of the adhesions depending upon the duration of the process. The surface of the lung is usually of a mottled red and gray colour; it often has a gran-

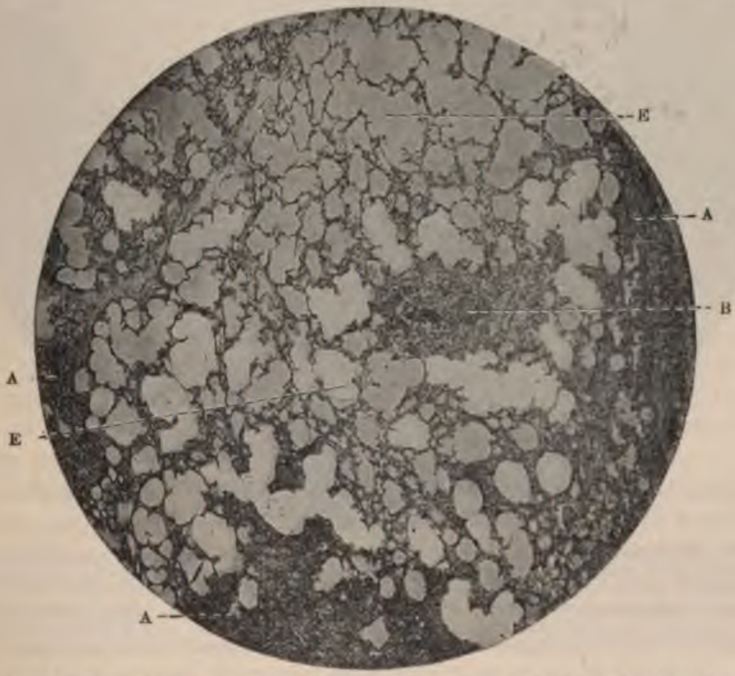


FIG. 90.—Acute broncho-pneumonia. In the centre is shown a small bronchus, B, with a zone of pneumonia about it. The greater part of the section is made up of emphysematous lung tissue, E E, showing dilatation of the alveolar spaces and rupture of some of the alveolar septa. At the border, A A A, are seen the margins of consolidated areas of lung.

ular feel, due to the consolidation of some of the superficial lobules of the lung. On section, it is rarely found that an entire lobe is consolidated, the superficial portion being most affected, while the central part is normal or only congested. The colour is mottled, like that of the surface. In some places the hepatization appears complete; in others the hepatized areas are separated by healthy, congested, or emphysematous lung tissue (Fig. 90). The gray areas surround the small bronchi and vary in size. The smallest ones look very much like miliary tubercles. The larger ones are seen where the process has existed for a longer time and has gradually invaded the contiguous air cells. If the lung is cut parallel with the bronchi, there may be seen small gray striæ of pneumonia along their course (Fig. 88, C). From the cut bronchi, pus flows

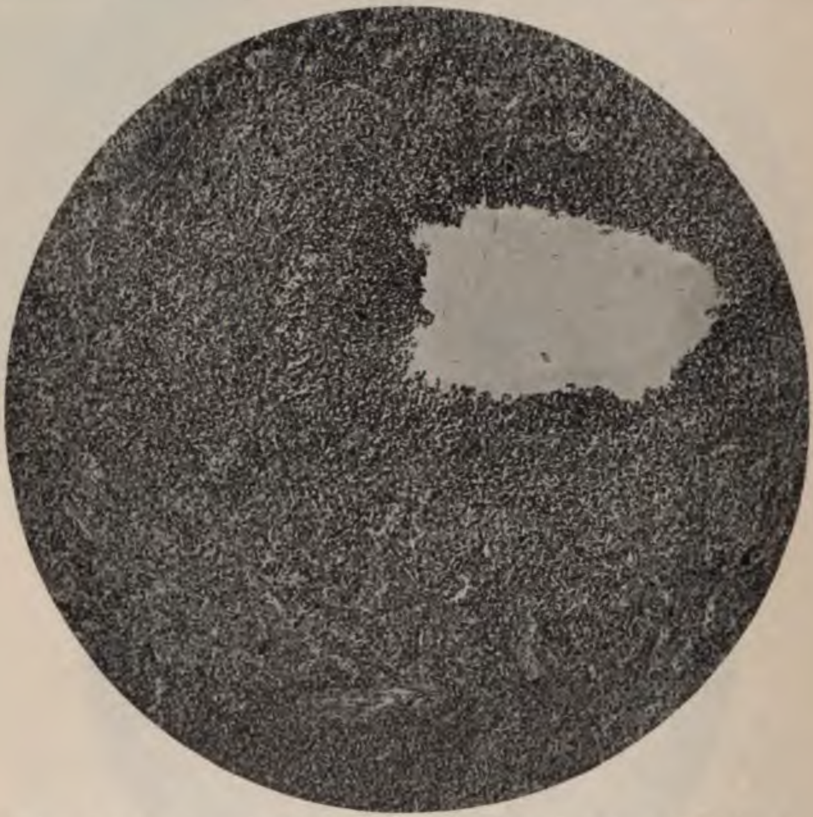


FIG. 91.—Broncho-pneumonia. Dense infiltration of pus cells in and about a small bronchus; under a low power. The cavity shown in the specimen is a cross-section of one of the small bronchi, which is partially filled with pus cells: the epithelium is destroyed. The bronchial wall and the pulmonary tissue in the neighbourhood are so densely infiltrated with leucocytes that almost every trace of normal structure is effaced. Child fifteen months old, disease of four weeks' duration. Extensive areas like this were found in both lungs.

quite freely on pressure. The bronchial walls can often be seen to be thickened even by the naked eye. The parts affected are usually the posterior portions of the lower lobes of one side, the remainder of the lobes being congested or œdematous, while in front the lung is emphysematous.

Under the microscope the smaller bronchi (Fig. 88) are seen to be much thickened and infiltrated with leucocytes. The gray areas surrounding the bronchi are made up of groups of air vesicles, which are packed with leucocytes (Fig. 91). Fibrin is sometimes seen in small amount, also red blood-cells and desquamated epithelial cells, but the leucocytes predominate. Surrounding the areas densely infiltrated are groups of air vesicles which are normal or congested, or which show only the earlier stages of the inflammatory process.

3. *Gray pneumonia* (*persistent broncho-pneumonia*).—This form is seen in protracted cases where there have been continuous symptoms usually for from three to six weeks. The pleuritic adhesions are more general and firmer. The amount of lung involved may be very great, often nearly the whole of both lungs posteriorly. The affected lung appears completely consolidated and slightly enlarged. On section, it is of a nearly uniform gray colour, sometimes of a yellowish-gray. On pressure, pus exudes from the smaller and larger bronchi. The bronchial walls are markedly thickened, and in some places there may be a slight dilatation of the smaller bronchi. The part of the lung not consolidated may be almost white, owing to vesicular emphysema. In some cases there is also interstitial emphysema. Small cavities containing pus may be found in the lung. The bronchial glands are frequently swollen to the size of a large bean, and are of a reddish-gray colour.

The microscope shows that the air vesicles of the consolidated portions are distended chiefly with leucocytes, but there are also epithelial and connective-tissue cells. The alveolar septa may be so much thickened as to encroach upon the alveolar spaces (Fig. 92). Complete resolution is then impossible.

Terminations.—Death may occur at any stage, or the pathological process may be arrested at any stage and the case go on to recovery. Resolution may take place before any consolidation recognisable by physical signs has occurred; in such cases it is usually rapid and complete. If there has been consolidation, resolution may take place after two or three weeks and be complete, or it may be delayed for five or six weeks and still be complete. In many cases, especially those in which it is delayed, resolution is only partial, and there are relapses or recurring attacks. After the first, or after several attacks, there may develop a chronic interstitial pneumonia; or simple pneumonia may be followed by tuberculosis. Such cases as these are to be carefully distinguished from the much more frequent ones in which the broncho-pneumonia is tuberculous from the outset.

Associated Lesions of the Lungs.—*Pleurisy* is almost invariably found over every large area of consolidation, and in cases of more than four days' duration; while in most of those fatal within the first two or three days the pleura is normal or only congested. It is seen in all grades of severity, from a slight gray film of fibrin that can hardly be stripped off, to a yellowish-green exudation one fourth of an inch thick. A small amount of serum—one or two ounces—in the pleural sac is not uncommon, but a large serous effusion is very rare. Cases in which there is an excessive inflammation of the pleura are considered elsewhere under the head of Pleuro-Pneumonia. Empyema occurs both during the stage of acute inflammation of the lung and while this is subsiding, but it is less frequent than in lobar pneumonia.

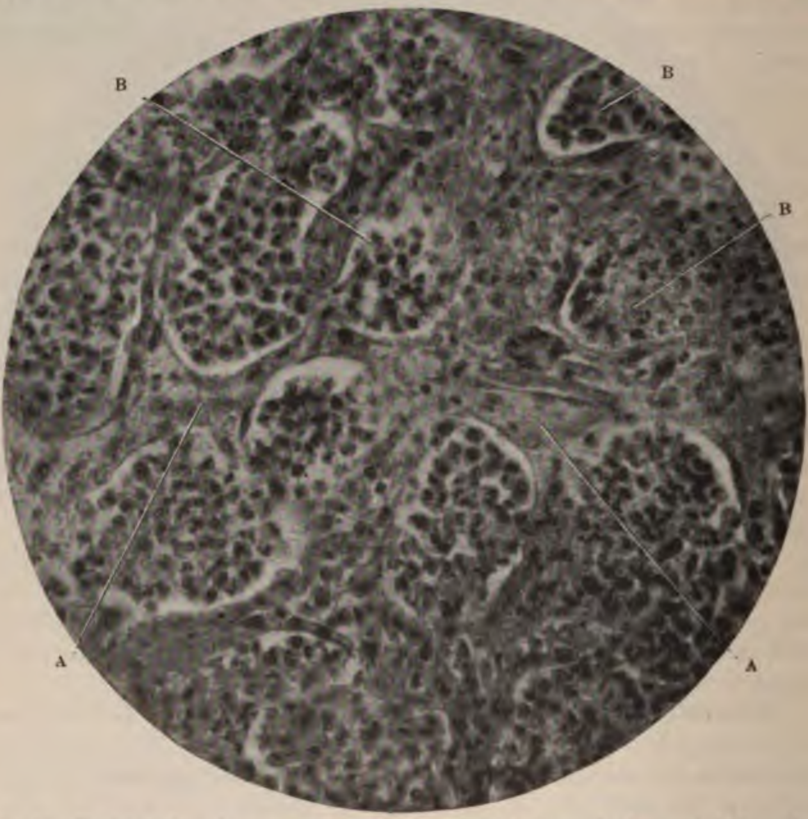


Fig. 92.—Persistent broncho-pneumonia; highly magnified. There is shown at A A marked thickening of the alveolar septa, encroaching upon the alveolar spaces. All the alveoli, B B, are densely packed with leucocytes. A similar condition also through nearly the whole of the affected lung. (For history and temperature, see Fig. 101.)

Bronchial glands.—In all the recent acute cases these are swollen and red; the usual size is that of a pea or a bean. They show microscopically the usual changes of acute hyperplasia. In protracted cases, and after repeated attacks, they may be two or three times the size mentioned, and of a gray colour. It is rare that they are large enough to give rise to symptoms unless they become the seat of tuberculous deposits.

Emphysema.—In almost all cases a certain amount of emphysema is present, it being more marked in the protracted cases. It is usually vesicular, involving the greater part of the upper lobes in front and the anterior margin of the lower lobes. Occasionally interstitial emphysema is seen, forming either large striæ upon the surface of the lung, or blebs of considerable size along the anterior margin. This may occur even in cases uncomplicated by pertussis or laryngeal stenosis.

Gangrene.—Gangrenous areas were found in six of my cases. In four of these the pneumonia was primary, in one it followed diphtheria, and in one ileo-colitis. It occurred in scattered areas of a grayish-green colour, varying from one fourth of an inch to two inches in diameter.

Abscesses of the lung are by no means uncommon. They were noted in seven per cent of my autopsies. They are usually minute and multiple, varying in size from one sixth to one half inch in diameter. Sometimes a portion of a lobe is fairly honeycombed with minute abscesses. In one case a large abscess was found occupying the greater part of a lobe, the symptoms resembling those of empyema. Abscesses are usually found in regions where the inflammatory process has been especially intense. They may be found in prolonged cases, in those of unusual severity, as shown by excessively high temperature and rapid extension of the disease, and in very delicate subjects. The microscope shows that these abscesses usually begin as an accumulation of pus in the small bronchi, whose walls become softened and break down on account of the intensity of the inflammation. They may be superficial, but are more commonly in the interior of the lung; they contain yellow pus and sometimes broken-down lung tissue. Small abscesses can not be recognised clinically; the large ones give the symptoms and signs of empyema. They are discussed more fully elsewhere. In several instances they have been successfully operated on, though wrongly diagnosticated.

The lesions in other organs will be considered under Complications.

Symptoms.—Broncho-pneumonia has no typical course. The cases differ from each other very markedly, but they may be divided into a few quite distinct groups.

1. *The acute congestive type.*—This may be seen at any age, but is more frequent in young infants. It may be either primary or secondary, being not uncommon in either form. Its symptoms are few and irregular, and the disease is often unrecognized. The entire duration may be only twenty-four hours. High temperature, extreme prostration, cyanosis, and rapid respiration may be the only symptoms. The temperature varies between 104° and 107° F., usually rising steadily until death occurs. The

prostration is extreme from the outset, the patient being overwhelmed by the suddenness and severity of the attack. Cyanosis is frequently present, and is almost always seen shortly before death. The respirations are from 60 to 80 a minute, but in most cases not strikingly laboured. Cough is frequently absent. Cerebral symptoms are often marked. There are dulness and apathy, sometimes quite profound stupor, and not infrequently convulsions just before death. The physical signs are few and inconclusive. There is often nothing abnormal except very rude breathing over both lungs behind; sometimes the breathing on one side is feeble, and on the other much exaggerated. There may be no râles whatever, and no change in the percussion note.

The suddenness and severity of these symptoms are something which it is hard for one who has not observed them to appreciate. I have known an infant to die in twelve hours from the time in which it was apparently in perfect health, and had an opportunity to confirm the diagnosis of pneumonia by a microscopical examination of the lung. The diagnosis can not be positively made during life, and in most of the cases the disease passes under some other name. It is often regarded as malignant scarlet fever or measles with suppressed eruption, or cerebro-spinal meningitis.

If the children are sufficiently strong to withstand the onset of violent symptoms, they may recover completely in four or five days, the lung clearing up very rapidly. In other cases these grave symptoms may abate in a day or two, to be followed by those of ordinary broncho-pneumonia, which runs its usual course.

The symptoms of some of these cases may be explained by the sudden intense engorgement of the lung, which, owing to the small size of the air vesicles, interferes with its function almost as much as does consolidation. In other cases the symptoms are due not so much to the pulmonary condition as to a general pneumococcus infection. A case lately came under my notice in which death occurred after a thirty hours' illness, where the pneumococcus was found by culture in both kidneys, the spleen, heart's blood, and both lungs.

2. *Acute disseminated broncho-pneumonia* (*capillary bronchitis*).—Although the symptoms in this class of cases are chiefly due to the bronchitis, I have never failed to find at autopsy evidences of pneumonia also. These are not very common cases. The process begins as an inflammation of the medium-sized and small bronchi, but not of the finest bronchi. The onset is acute, with fever, very rapid and laboured breathing, severe cough, moderate prostration, and in most cases cyanosis.

The temperature is not high, usually only from 100° to 102° F., and it often continues so for three or four days. The pulse is rapid, and at first is full and strong. The respirations are exceedingly rapid, often from 80 to 100 a minute. There is dyspnœa with marked recession of all the soft parts of the chest during inspiration. Cough is always present, usually severe, and sometimes almost incessant. The prostration is not so great as in the cases previously described, and the development of the symptoms is much less rapid.

There are at first sibilant and afterward subcrepitant râles over the entire chest, with which are usually mingled coarser moist râles. There are no evidences of consolidation. The respiratory murmur is everywhere feeble, but not otherwise altered. Percussion generally gives exaggerated resonance, owing to the emphysema which is present, the note being sometimes almost tympanitic.

The symptoms may gradually increase in severity until death takes place by the third or fourth day, from respiratory and cardiac failure. There is usually marked cyanosis, and toward the end rapidly increasing prostration. Just before death the temperature often rises rapidly to 106° or 107° F. At the autopsy there are found evidences of bronchitis of the tubes of all sizes, and minute zones of pneumonia about the smaller bronchi. The lungs are generally in a state of hyper-inflation, on account of which they do not collapse on opening the chest. There may be in addition extensive congestion or œdema, the development of which has been the immediate cause of death.

In cases which do not prove fatal there is usually by the third or fourth day great improvement in the general symptoms; the finer râles may disappear, and the coarse ones become more and more prominent. By the end of a week there may be complete recovery. Instead of this, there may be a continuance of the constitutional symptoms, and disappearance of the fine râles in front only, while behind there are gradually added to them the signs of consolidation in one of the lower lobes near the spine. From this time the case may progress as one of ordinary broncho-pneumonia.

The prognosis in this class of cases is very much better than in the congestive variety, recovery being probable unless the patients are very young or very delicate infants.

3. *Broncho-pneumonia of the common type.*—When primary, this usually begins suddenly with symptoms not unlike those of lobar pneumonia. This was the mode of onset in two thirds of my cases. In only ten per cent was the pneumonia preceded by bronchitis of the large tubes. In these the symptoms of bronchitis may slowly (Fig. 102, p. 552) or rapidly (Fig. 93) merge into those of pneumonia. When the onset is sudden it is marked by high fever, frequently by vomiting, rarely by convulsions. In addition there are rapid respiration, cough, prostration, and sometimes cyanosis. The symptoms are more distinctly pulmonary than is generally the case in lobar pneumonia.

The temperature, as a rule, is high; rarely is it continuously so, but it is of a remittent type. The daily fluctuations often amount to four or five degrees. The fever usually continues from one to three weeks, and gradually subsides. It is rare for it to terminate by crisis. Although, as a rule, we expect a high temperature with acute pneumonia, this is not invariable. Primary cases may run their course, and even terminate fatally, although the temperature has not been above 101° F. I have records of several such cases. A low temperature is more often seen in young and delicate infants than in those who are older and more robust.

The respirations are frequent and laboured; there is real dyspnœa. On inspiration, there are marked recessions of all the soft parts of the chest, and the alæ nasi dilate actively. The usual rapidity of the respirations is from 60 to 80 per minute; very often, however, it rises to 100, and on several occasions I have seen it even 120. Respiration generally seems more embarrassed than the action of the heart, and respiratory failure is a more frequent cause of death than cardiac failure. The pulse is always rapid—from 150 to 200 a minute—and when so it is often irregular. The pulse rate is of much less importance than its character. Early it is full and strong, but soon it becomes soft, compressible, and weak.

The prostration is usually moderate for the first day or two, but steadily increases as the lung becomes more and more involved. Toward the close of the disease there may be present all the symptoms of the typhoid condition.

Cough is much more constant than in lobar pneumonia, and more distressing; sometimes it is almost incessant. It disturbs rest and sleep, and may cause vomiting if the paroxysm occurs soon after eating. There is no expectoration. Mucus is sometimes coughed up into the trachea, or even the pharynx, to be swallowed again, or more frequently aspirated into the lung. If during a severe paroxysm the patient is turned upon his face or inverted, much of this mucus may be dislodged. A strong cough is a good symptom; suppression of the cough is always a bad symptom, indicating a loss of the reflex sensibility of the bronchial mucous membrane and feeble respiratory muscles.

Pain in the chest is not common, and is rarely an annoying symptom.

Cyanosis is present at some time in most of the severe cases. It may occur at the onset, or at any time during the course of the disease. It is usually due to sudden congestion of a portion of the lung not previously involved. Even when slight, it is always a danger-signal of respiratory failure, and when present only in the finger tips or lips indicates that the patient must be carefully watched and energetically treated. In the severe cases the whole body may be of a dull leaden hue.

Nervous symptoms at the onset are not so frequent as in lobar pneumonia, convulsions being rare; but late convulsions, particularly in the pneumonia which complicates pertussis, are exceedingly frequent, and usually fatal. Delirium may be present at any time during the attack. In infants this shows itself by excitement and inability to recognise the nurse or mother. Occasionally patients present marked cerebral symptoms throughout the disease. In one of my cases nearly every symptom of tuberculous meningitis was present, the autopsy revealing only an extreme degree of cerebral anæmia. As elsewhere stated, the nervous symptoms depend not upon the location of the disease, but upon its extent, the intensity of the infection, and upon the susceptibility of the patient, such symptoms being especially common in rachitic children and in those suffering from pertussis.

Gastro-enteric symptoms are frequent in infancy, and are of much importance. Often there are from four to six stools a day, of a green colour, containing mucus and undigested food. These symptoms depend upon the feeble digestion which is associated with the febrile process, and are often from improper feeding. This may lead to vomiting, which is also due to over-medication or to severe paroxysms of coughing. Vomiting and diarrhœa add much to the danger of the attack, and not infrequently, when the issue is doubtful, turn the scale against the patient. In summer this complication is more frequent and is likely to be more severe. Distention of the stomach or intestines from gas may be the cause of severe symptoms, owing to the added embarrassment of respiration produced by this upward pressure. In infants it may lead to attacks of cyanosis, and even convulsions.

The urine in most cases is scanty, high-coloured, and loaded with urates. A trace of albumin is often present when the temperature is very high; but casts, renal epithelium, and a large amount of albumin are rare.

The following temperature chart (Fig. 93) is a good example of a very frequent course of primary pneumonia of moderate severity terminating in recovery. In cases of this type the constitutional symptoms are not grave, and follow very closely the temperature curve.

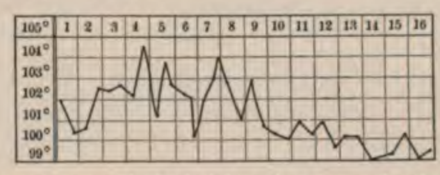


FIG. 93.—Temperature curve in typical broncho-pneumonia of the milder form.

History.—Male, sixteen months old; delicate child; previous bronchitis; onset gradual; signs of consolidation at left base on fifth day, but fine râles over both lower lobes behind; resolution slow, râles persisting for a long time in both lungs.

The next chart (Fig. 94) illustrates a more severe but not uncommon course of the disease in which the fever is prolonged. The usual duration of cases of this type is between three and four weeks. The irregular fluctuations of the temperature, rarely touching the normal line, are exceedingly characteristic of broncho-pneumonia.

The chart shown in Fig. 95 is that of relapsing pneumonia. The first attack was fairly typical, with about the usual duration. Resolution had begun, and was apparently progressing favourably, when there was a return of the fever, accompanied by new signs in the chest, the second attack being shorter and milder than the first. Very often the temperature falls to normal without any signs of resolution, and after an interval varying from two or three days to a week there is recurrence of the fever

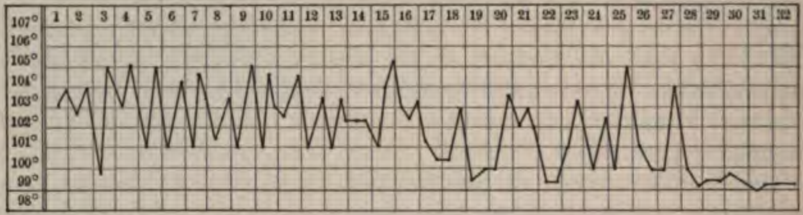


FIG. 94.—Temperature curve of broncho-pneumonia with a prolonged course; recovery.

History.—Female, eighteen months old; in fair condition; sudden onset. Early signs were localized, fine râles over left base; on fifth day signs of consolidation at left base, with râles on both sides behind. General symptoms of moderate severity. Signs of consolidation disappeared about a week after cessation of fever; râles persisted nearly two weeks longer.

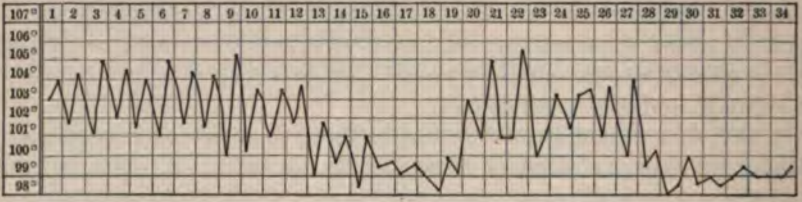


FIG. 95.—Temperature curve of relapsing broncho-pneumonia; recovery.

History.—Male, nineteen months old; delicate. Consolidation on sixth day in left lower lobe behind; two days later small area of consolidation in right lower lobe behind; many râles both sides; eighteenth day, signs of consolidation had disappeared, but many râles persisted. Accession of fever on nineteenth and twentieth days, accompanied by extension of disease as shown by new râles, but no evidences of consolidation during second attack. Slow resolution and convalescence.

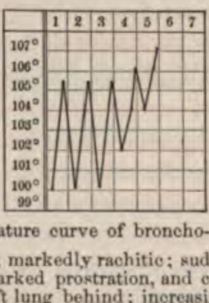


FIG. 96.—Temperature curve of broncho-pneumonia; fatal.

History.—Male, six months old; markedly rachitic; sudden onset. Signs first day were fine moist râles throughout the chest, marked prostration, and cyanosis; on third day, a small area of consolidation in upper lobe of left lung behind; increasing prostration, cyanosis, and death. *Autopsy.*—No pleurisy; consolidation at left apex behind, and posterior two thirds of left lower lobe; consolidation of right apex posteriorly, lower lobe intensely congested.

and other constitutional symptoms, the second attack frequently proving fatal.

A frequent course in fatal cases is shown in Fig. 96. The duration of the disease, instead of being five days as in this case, is often only three or four. The temperature at first fluctuates widely, then rises gradually until death.

Duration of the fever.—The following figures give the duration of the fever in 231 cases. The majority were primary; none were secondary to diphtheria, and only a few complicated measles. Of the 169 cases that were fatal—

There died during the first six days	25·0 per cent.
" " between the seventh and twenty-first days	55·5 " "
" " " " twenty-first and sixtieth days	19·5 " "
	100·0 " "

Of 78 cases which recovered, the duration of the fever was—

Less than seven days	11·5 per cent.
From seven to twenty-one days	66·6 " "
From twenty-one to ninety days	21·9 " "
	100·0 " "

Physical Signs.—In considering the signs of broncho-pneumonia, it is better to connect them with the different conditions in the lung than to group them in stages, as in lobar pneumonia.

(*a*) *Without consolidation.*—It can not too often be repeated that broncho-pneumonia may exist without signs of consolidation at any period during the course of the disease. When the attack is primary, the earliest signs are due to congestion of the lung, associated with bronchitis of the fine tubes, which is usually localized, but which may be general. If the disease has followed bronchitis of the large tubes, its signs are added. Congestion of the lung gives feeble breathing over the affected area, and occasionally slight dulness or diminished resonance. With this are found coarse sonorous, and finer sibilant râles, due to congestion and swelling of the mucous membrane of the larger and smaller bronchi respectively. These signs are soon replaced by very fine moist râles, which are usually localized in one of the lower lobes behind (Fig. 97). These localized fine râles are the first distinctive sign of broncho-pneumonia. Soon a change in the respiratory murmur is heard in the affected area, becoming feebler in intensity and higher in pitch. Elsewhere in the chest there may be coarse râles, due to bronchitis of the large tubes. In such cases the areas of pneumonia are so small and so scattered as to give in themselves no additional signs, and the case may go on to recovery without presenting anything more distinctive than the signs mentioned.

(*b*) *With areas of partial consolidation.*—In the lung at this time there are small areas of consolidation, generally superficial and separated

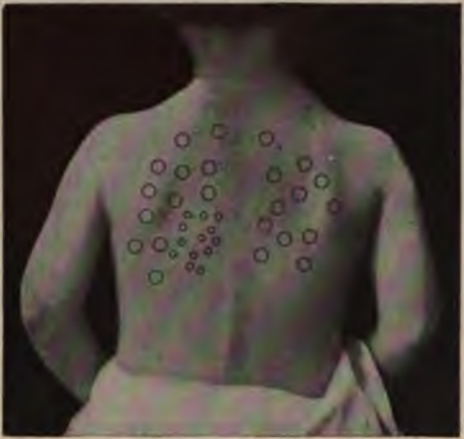


FIG. 97.—First stage. Coarse râles over both lungs; localized fine (subcrepitant) râles at the left base. No change in breathing sounds.

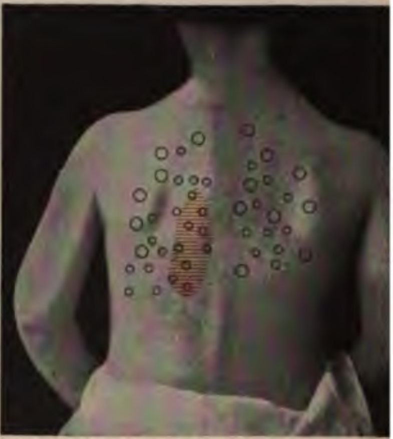


FIG. 98.—Second stage. Coarse and fine râle
both lungs behind; at left base an a
partial consolidation, with broncho-ves
breathing, exaggerated voice, and very
râles.

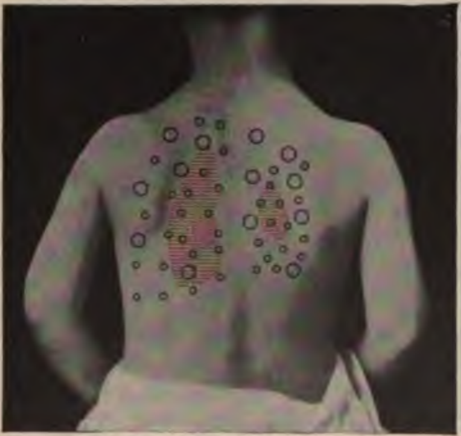


FIG. 99.—Third stage. A larger area of partial consolidation, and in the centre a small area of complete consolidation, with bronchial breathing and voice and slight dulness. Signs over the right lung similar to what were previously present over the left.

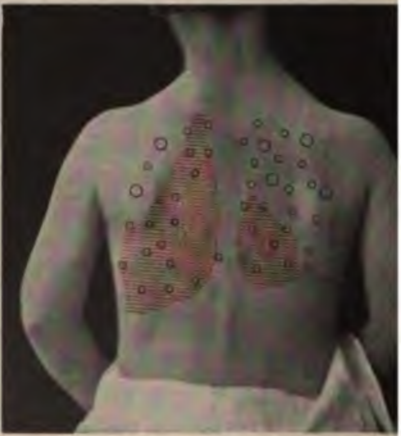


FIG. 100.—Fourth stage. Extensive disease o
sides; large area of complete consolidati
the left, with dulness, bronchial breathin
voice, and no râles; surrounding this, bro
vesicular breathing, with many râles.
in the right lung similar to those prev
present over the left.

NOTE.—The disease may stop at any one of these stages and resolution take place.

by healthy or congested lobules. Percussion in these cases usually gives negative results, but sometimes there is very slight dulness. The vocal fremitus is not usually altered. The fine moist râles may be heard over quite a large area, but at some point, usually near the spine, over one of the lower lobes, they are sharper, louder, higher pitched, and seem close under the ear (Fig. 98). Respiration is feebler here than elsewhere, and broncho-vesicular in quality, approaching bronchial breathing more and more as the consolidation increases. The resonance of the voice and cry is exaggerated.

(c) *With areas of consolidation more or less complete.*—On percussion there is dulness, but surprisingly little in comparison with the other signs of consolidation present. It is due to the fact that the consolidated portion, though extensive, is superficial, and does not involve the lung to any great depth, and also that there are in the consolidated area many alveoli which still contain air (Plate XI). On palpation there is usually a slight increase in the vocal fremitus. On auscultation, there are still present the evidences of bronchitis, usually only behind, but sometimes over the entire chest. Coarse and fine râles are intermingled. Over the consolidated parts are heard bronchial breathing and bronchial voice. At the centre of these areas the bronchial breathing is pure and râles are usually absent, but at the margin râles are present and the breathing approaches the broncho-vesicular type (Fig. 99). The signs of consolidation are rarely sharply circumscribed as they are in lobar pneumonia, but shade off gradually. The consolidated area is at first small, usually in one of the lower lobes near the spine, but may gradually extend until nearly the whole of one or even both lungs behind are more or less completely solidified (Fig. 100). The signs are found as far forward as the axillary line, but usually stop there. Friction sounds may be heard over the consolidated areas, but very rarely except where signs of complete consolidation are present. It is often impossible to obtain any idea of the condition of an infant's lung during quiet, superficial respiration. Sometimes over a part which is completely consolidated there is heard only very feeble breathing, or the lung may be almost silent. If, however, the child be made to cry or to take a deep inspiration, both the bronchial breathing and râles are distinctly brought out. The intensity of the consolidation increases as the case advances, and the signs become more and more like those of lobar pneumonia. During resolution there is first a disappearance of the signs of consolidation, which may be quite rapid, but friction sounds and râles of all kinds often persist for three or four weeks longer.

The following statistics are of some interest, as showing the frequency with which signs of consolidation were found, and the day when they were discovered. Their value is increased by the fact that the children were under observation in an institution at the time they were taken sick, and that in all the fatal cases—thirty-six in number—in which signs of consolidation were absent, the diagnosis of pneumonia was confirmed by autopsy:

Consolidation noted on or before the fourth day	47 cases.
" " from the fifth to the seventh day	36 "
" " " the eighth to the twelfth day	12 "
" " after the twelfth day	9 "
No signs of consolidation	62 "
	166 "

In general, it must be borne in mind that in many cases signs of consolidation are never present, as the areas of pneumonia are small and widely scattered; that where there is consolidation it is usually incomplete, because there are small areas of healthy lung tissue between the hepatized portions; that the signs of consolidation usually shade off gradually; and that both sides are almost invariably involved, although one side usually to a greater degree than the other.

(4) *The protracted form—Persistent broncho-pneumonia.*—This is seen in primary cases, especially among delicate children, and it is not uncommon in pneumonia complicating pertussis. The onset and course of the disease for the first two or three weeks do not differ from an ordinary attack of moderate severity, but at the end of this period there is seen no tendency in the process to subside. The fever continues, but it is not high, and by physical examination it is found that the areas of consolidation are gradually increasing day by day, until sometimes the greater part of both lungs behind are involved. The air vesicles become so distended with cells that the signs of consolidation are more complete than in ordinary broncho-pneumonia. There is marked dulness, sometimes almost flatness; bronchial breathing is exaggerated in intensity, until it resembles cavernous breathing, and it may be impossible to distinguish between them. However, the fact that it is heard over so large an area, that it shades off gradually, and that it is accompanied by friction sounds, usually make a distinction possible.

The temperature in these protracted cases for the first two or three weeks is from 100° to 105° F.; but after this time it is generally lower—from 100° to 102° or 103° F. The course is not at all regular, but marked by frequent exacerbations and remissions. The general symptoms are those of progressive asthenia. There is continued wasting, anæmia, and steadily increasing prostration. The appetite is lost, often there is an aversion to food, and vomiting is easily excited if food or stimulants are forced. The stools show that even what food is taken is very imperfectly digested and assimilated. The skin becomes dry and loses its elasticity; bed-sores may form; fine punctate hæmorrhages are seen over the abdomen, sometimes over the chest and extremities. The latter is always a very bad symptom, and I have never seen recovery where it was present.

The chart in Fig. 101 is typical of the course of one of these protracted cases terminating fatally. The temperature shows four distinct exacerbations.

Death takes place from slow asthenia, usually after five or six weeks, but the attack may be prolonged for eight or ten weeks. The general symptoms, the temperature, and the wasting strikingly resemble cases of tuberculosis, and such is the diagnosis often made.

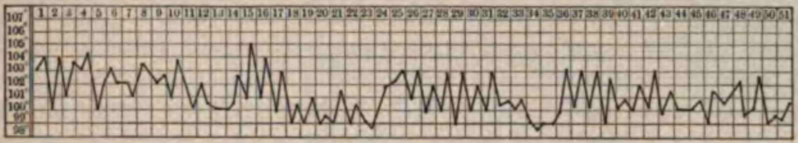


Fig. 101.—Temperature curve of persistent broncho-pneumonia, terminating fatally.

History.—Male, two and a half years old; healthy; sudden onset; for two weeks the only signs were very fine moist râles throughout both lungs, front and back. The râles in front in great part gradually cleared up; those behind persisted, but it was not until the thirty-fourth day that positive signs of consolidation were discovered in the left lower lobe behind; these signs gradually extended, and, before death, were present over nearly the whole left lung behind and over the right lower lobe. There were also friction sounds over both lungs. *Autopsy.*—Old and recent pleurisy with general adhesions; left lower lobe completely solid, patches of consolidation in left upper lobe. Right lower lobe about one half consolidated, with patches elsewhere. Bronchial glands large, but not cheesy. No evidence of tuberculosis upon either gross or microscopical examination (see Fig. 92).

Although the majority of the cases in which the fever lasts over four weeks run the fatal course just described, such apparently hopeless cases occasionally recover. The temperature gradually falls lower and lower, until it remains at the normal point. For some time after this, often two or three weeks, little change can be seen, either in the general symptoms or in the physical signs. Gradually the appetite returns, the child is brighter and begins to take an interest in its surroundings, the cough abates, and little by little the signs in the lungs clear up, and the case may go on to complete recovery. Convalescence, however, is always slow, and may be interrupted by relapses, it being many months before health is fully restored. Although the signs of consolidation disappear in a few weeks, râles are apt to persist for a much longer time. It is probable in such cases, even though all signs of disease disappear from the chest, that the lung does not become quite normal, and relapses and second attacks are always possible. The general health may be so undermined that the child never regains his former vigour; yet in a surprising number of these cases recovery seems to be complete.

5. *Secondary pneumonia.*—(*a*) Complicating pertussis.—It is not often that pneumonia develops during the first two weeks of this disease. The most frequent time is from the third to the fifth week, when the patient has become exhausted from the previous severity of the pertussis. In two thirds of my cases the development of the pneumonia was gradual, following bronchitis of the larger tubes. The temperature chart shown in Fig. 102 well illustrates this course.

When the onset is sudden, the symptoms do not differ essentially from those of primary pneumonia. The temperature of pertussis-pneumonia is usually low, in a very large number of cases not rising above 103·5° F., and ranging most of the time from 101° to 103° F. These cases are very apt to be prolonged, the fever often lasting for three or four and sometimes even for six weeks. The physical signs of consolidation may persist for a long time after the temperature has become normal, and yet the case may recover entirely. I have seen one case in which complete recovery occurred after the signs of consolidation had persisted for six months, and another in which they had persisted for over eight months. Very often the signs continue during the entire attack of pertussis. Cerebral symptoms are common, especially toward the close of the disease. Of fifty-four fatal cases twenty-five had convulsions, and in twenty-two this was the mode of death. Only one case which developed convulsions recovered.

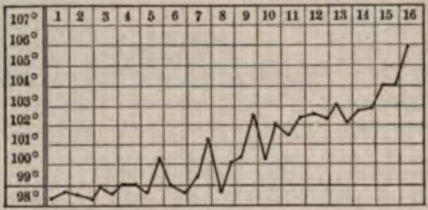


FIG. 102.—Temperature curve of fatal broncho-pneumonia, complicating pertussis.

History.—Male, six months old; delicate; pertussis for three weeks. Early signs of bronchitis of large tubes only; on the eleventh day signs of consolidation in right upper lobe. Increasing prostration, cyanosis, and death. *Autopsy.*—Large areas of consolidation in right middle and upper lobe, small scattered spots throughout left lung.

(*b*) Complicating measles.—In a small number of cases the pneumonia begins simultaneously with the invasion of measles, but generally not until the eruption appears. Instead of gradually falling to normal with the fading of the eruption, the temperature continues high. Any of the clinical types of primary pneumonia may occur in measles, the acute congestive variety which is fatal in two or three days, being especially common. In its course and duration the pneumonia of measles resembles the severe form of primary pneumonia. The broncho-pneumonia of scarlet fever differs in no way from that of measles.

(*c*) Complicating diphtheria.—In many cases this does not give a distinct clinical picture of its own, its symptoms being mingled with those of diphtheritic bronchitis, with which it is frequently associated. In others the forms resemble those seen in measles. The majority of cases occur as a complication of diphtheria of the larynx, although it is not infrequent in the septic cases in which only the upper air passages are involved. Pneumonia developing after laryngitis is usually seen within two days from the beginning of laryngeal symptoms, and runs a very rapid course. In rare cases it may develop as late as the middle or end of the second week. When it complicates diphtheritic bronchitis, pneumonia is recognised by the high temperature, rapid breathing, and increased prostration, much more certainly than by the physical signs, which are always obscured by the laryngeal sounds. Percussion may aid in the diagnosis of consolidation where the signs on auscultation are doubtful. In the early cases, death usually occurs before the disease has advanced far enough to give the physical signs of consolidation, but in the late pneumonia, which develops more slowly, these may be present.

(*d*) Complicating influenza.—Without doubt many cases regarded as primary are really secondary to influenza, particularly when that disease is prevailing, for very often the pneumonia of influenza differs in no essential points from the primary form. There are, however, two types which are quite characteristic. In the first, high temperature and prostration exist for several days before there are any physical signs of pulmonary disease, and often before there are any symptoms pointing definitely to the lungs. Pneumonia may then develop and run its usual course. The second variety are the cases of short duration often lasting but three or four days, and sometimes only two, but with excessively high temperature and very severe general symptoms.

(*e*) Complicating ileo-colitis.—This is usually a somewhat subacute form of pneumonia, which is scarcely recognisable except by the physical signs. It is seen in the protracted cases of ileo-colitis, usually of the ulcerative variety, and occurs late in its course. The temperature is not high. Cough, pain, and dyspnœa are slight or entirely wanting. Accelerated respiration is frequently the only symptom suggestive of pulmonary disease. By physical examination there are found the usual signs, generally involving both lungs posteriorly. Very often pneumonia is not suspected during life, the constitutional symptoms being sufficiently explained by the intestinal lesions, although the autopsy discloses the fact that death was due to pneumonia.

Complications.—Those relating to the lungs have been described with the lesions. Pleurisy will be separately considered. Emphysema can rarely, and abscess and gangrene never, be recognised by the physical signs.

Purulent meningitis may complicate acute broncho-pneumonia. It was met with twice in one hundred and seventy autopsies. It is in all respects similar to that occurring with lobar pneumonia. Meningeal hæmorrhage was seen only once, and was the cause of death in a patient eleven months old, who a few days before was seized with convulsions, followed by a gradually increasing stupor, which continued until death. The hæmorrhage covered the entire convexity of the brain. Endocarditis is extremely rare; it was not observed in any of my cases. Acute pericarditis was seen but twice, in both cases complicating pneumonia of the left side. Complications referable to the digestive tract are quite common. Herpetic stomatitis is frequent, and occasionally the ulcerative variety is seen. Thrush often occurs in the protracted cases among very young infants. Gastro-enteritis is not very common, considering the frequency of vomiting and diarrhœa, these depending usually upon functional derangement. In only three of my cases was there nephritis. In all it was of the acute exudative variety, and in only one case was it severe enough to affect the prognosis.

Old lesions of tuberculosis—cheesy nodules in the lungs and sometimes in the pleura—are not infrequently met with in patients dying of acute pneumonia of a non-tuberculous character.

Diagnosis.—An acute onset with continuous high fever, rapid respiration, and cough, should always lead one to suspect pneumonia. When to these symptoms are added prostration and cyanosis, the diagnosis of pneumonia is almost certain. Cases of the acute congestive type are the ones most frequently unrecognised, and in many of these cases a positive diagnosis is impossible during life. Many atypical cases of pneumonia are seen, particularly in young infants. An unusual temperature course is perhaps the symptom most likely to lead to a mistake. While this, as a rule, is high and remittent, it is sometimes not so, and may be but little above normal. Rapid respiration is almost always present, but cough may be very slight, especially in infants. In very young infants, the diagnosis often rests upon the prostration, cyanosis, and rapid respiration, the other acute inflammatory symptoms being absent. Only the physical signs of the disease can positively settle the question of diagnosis.

When pneumonia follows bronchitis of the large tubes, whether the bronchitis is primary or complicates one of the infectious diseases, the extension of the disease to the lungs is usually marked by three symptoms —a steadily rising temperature, more frequent respiration, and increasing prostration. It may be twelve or twenty-four hours before the change is indicated by the physical signs.

The diagnosis of broncho-pneumonia from congenital atelectasis has to be considered only during the first three or four months of life, it being rare for atelectasis to give symptoms after this time. In early infancy the danger of confusing the two is increased by the fact that atelectasis and broncho-pneumonia may be associated. If the infant has been strong and well for the first two months, congenital atelectasis can be excluded. It is likely to be found in delicate infants, where there is a history of difficulty in resuscitation at birth and feeble cry during the early days of life. The temperature is low, often subnormal, the cyanosis is out of proportion to the other symptoms, and the physical signs are doubtful or absent.

At the outset, pneumonia can not be positively diagnosticated from severe bronchitis. Such a bronchitis often begins with severe pulmonary symptoms and a temperature of 103° or 104° F.; but this high temperature is of short duration, usually falling after twenty-four or forty-eight hours to 100° or 101° F. The prostration is much less, and all the symptoms, possibly excepting the cough, less severe. The only physical signs are coarse râles, which are heard throughout the chest.

The same rules apply to bronchitis of the smaller tubes. The râles are heard both in front and behind, and usually over both sides. If with such râles the temperature continues to rise for three days in succession above 103° F., it may be assumed that pneumonia is present, provided there is no other disease which might explain the temperature. If, instead of being generalized, the signs of bronchitis are limited to a single lung, or to one lung posteriorly, the existence of broncho-pneumonia may be regarded as certain. Localized bronchitis, then, is always to be interpreted as broncho-pneumonia, provided tuberculosis can be excluded. In doubtful cases the chances largely favour broncho-pneumonia rather than bronchitis. Attention is again called to the fact already mentioned, that there are a large number of cases of pneumonia without signs of consolidation.

The differential diagnosis of broncho- from lobar pneumonia will be considered in connection with the latter disease. On account of the remittent temperature, broncho-pneumonia may be confounded with malarial fever; if with the latter there is some bronchitis, or if accompanying the onset of a severe malarial paroxysm there is pulmonary congestion—two not infrequent combinations—the difficulties are increased. A positive diagnosis is often impossible except by careful observations of the temperature for one or two days. The points of differentiation are, that the temperature of pneumonia, though often remittent, is very rarely intermittent, and that it is not affected by quinine. In addition, the characteristic features of malaria—enlargement of the spleen, the plasmodium in the blood, and a history of exposure—must, of course, be taken into account.

Both the acute and the persistent forms of simple broncho-pneumonia may be confounded with the tuberculous form; the points of distinction are considered in the chapter on Tuberculosis.

Prognosis.—Broncho-pneumonia is always a serious disease, and in an infant dangerous to life. The prognosis depends upon the age, surroundings, and previous condition of the patient, upon the nature of the infection, whether the disease is primary or secondary, and, if the latter, upon the character of the primary disease. In private practice the mortality from broncho-pneumonia is from 10 to 20 per cent, depending upon the conditions mentioned. One whose knowledge of broncho-pneumonia is derived from observations in private practice can, however, form but little idea of the frequency and severity of this disease in hospitals and asylums for infants and young children, particularly when it occurs with epidemics of measles, diphtheria, and pertussis. The statistics in the following table are taken from the records of two institutions with which I am connected, and fairly represent the results seen in such places in children under three years:

FORMS OF PNEUMONIA.	Cases.	Deaths.	Percentage mortality.
Primary broncho-pneumonia	194	96	49·4
Following bronchitis of the large tubes	29	19	65·5
Secondary to measles	89	56	62·9
" " pertussis	66	54	81·8
" " scarlet fever	7	7	100·0
" " diphtheria	47	47	100·0
" " ileo-colitis	19	18	94·7
" " epidemic influenza	6	1	16·6
" " varicella	2	2	100·0
" " erysipelas	2	2	100·0
Totals	461	302	65·5

The mortality varies directly with the age of the patient, being the highest during the first year, and diminishing steadily thereafter, as shown by the following table giving the result in three hundred and forty-five cases:

AGE.	Cases.	Percentage mortality.
During the first year	202	66
" " second year	102	55
" " third year	33	33
" " fourth year	6	16
" " fifth year	3	..

In this table are included no cases secondary to measles, scarlet fever, or diphtheria.

Probably the best of all guides to the nature and virulence of the infection is the temperature. An excessively high temperature indicates a virulent type of infection. Some idea of this may be gained from these figures, giving the highest temperature and the mortality in two hundred and thirty-one cases, not including cases with measles or diphtheria:

HIGHEST TEMPERATURE.	Cases.	Deaths.	Percentage mortality.
106° F. or over	55	47	85·5
105° or 105·5°	94	56	60·0
104° or 104·5°	53	26	49·0
102° to 103·5°	22	13	60·0
99·5° to 101·5°	7	5	71·0

The high mortality of the cases with unusually low temperature is due to the fact that they nearly always were seen in infants with very feeble vitality. Cases with a steadily high temperature—between 102·5° and 104° F.—usually do better than those with wide fluctuations, such as 100° to 105·5° F. The probable explanation of this is, that the former are due to the pneumococcus, while the latter are apt to be cases of mixed infection, or due to the streptococcus. As a rule, the danger from the disease increases steadily with every degree of temperature above 104·5° F.

An important factor in the prognosis is the previous condition of the patient. The association with rickets is unfavourable, both on account of the feeble muscular power of these children and their thoracic deformities. Any condition which diminishes the general vitality increases the danger from broncho-pneumonia. As a rule, second attacks are more serious than the primary ones, especially if the interval between them is short.

In making the prognosis in any given case, the symptoms to be considered are the height and course of the temperature, the presence or absence of nervous symptoms, the condition of the organs of digestion, the presence of cyanosis and the extent of the disease as shown by the physical signs.

Nervous symptoms early in the disease do not affect the prognosis. Three cases in which convulsions occurred at the onset recovered, but of thirty-seven cases in which convulsions occurred at a late period during the course of the disease, all but one proved fatal.

So long as the food is well taken and retained and the stools show that it is being assimilated, no case is hopeless, no matter how severe the other symptoms may be; but the existence of vomiting, diarrhœa, or severe indigestion makes the issue doubtful, even though the other symptoms are very favourable. These conditions are especially important in protracted cases, where death is usually due to slow asthenia.

Treatment.—The most important part of prophylaxis is to give careful and early attention to every attack of bronchitis in an infant, for every such attack should be regarded as a possible precursor of pneumonia. It is striking that one sees broncho-pneumonia so seldom in private practice among the better classes, even though bronchitis is very frequent; while among hospital and dispensary patients, where bronchitis is very often neglected, broncho-pneumonia is constantly seen. The question of isolating cases of pneumonia is one which is lately becoming more and more important. While it may not often be the case that primary pneumonia is due to contagion, there seems to be little doubt that this is at times true of the pneumonia secondary to measles and diphtheria. Twice in one institution have I seen regular epidemics of broncho-pneumonia occur with outbreaks of measles—in some of the wards nearly every case of measles developing pneumonia. In another institution, during one entire season (1888–'89), almost every case of diphtheria transferred to a certain isolation pavilion developed pneumonia, and died from that complication. Cases of measles and diphtheria which are complicated by pneumonia should, if possible, be carefully isolated from others, and wards in which they are treated should be thoroughly disinfected before they are used for simple cases.

The hygienic treatment of pneumonia is important, and usually it receives too little attention. The child should be kept in a large, well-ventilated room, preferably one with an open fire; if possible, he should be changed from one room to another two or three times a day, to allow thorough airing. Nothing is more important for an infant sick with acute pulmonary disease than plenty of pure air. Older children should be kept in bed. Infants for a considerable part of the time may be held in the nurse's arms. A frequent change of position in all cases is essential; no child should be allowed to lie for hours directly on the back. The general rules previously laid down for feeding all sick children should be followed here. As a rule, neither stimulants nor medicine should be administered in the food.

The same local treatment may be employed as in cases of bronchitis. Counter-irritation, best by means of the mustard paste, may be employed from three to six times daily. It is of the greatest value in the early stage of acute pulmonary congestion, and during attacks of cardiac or respiratory failure. The oiled-silk jacket may be applied with advantage in cases with low temperature, but should not be used when the temperature is high, as it seriously interferes with the means employed for its reduction. Poultices should not be used at all.

Emetics.—What was said of expectorant mixtures and emetics in the treatment of bronchitis applies here with even greater force.

Stimulants.—Alcoholic stimulants are needed in all secondary cases, and in a large proportion of those which are primary. No doubt they have been greatly abused, and, when pushed in the early stage, often do much harm; but in most of the severe cases they are indispensable. They are usually needed from the outset when the pneumonia is secondary to measles, diphtheria, scarlet fever, or other infectious diseases. They are called for when the pulse is weak, compressible, rapid, and irregular. Whisky or brandy is usually to be preferred, although the taste of the patient often has to be consulted, and when these are refused, some wines, like sherry or tokay, may be readily taken. (For methods of administration see page 51.) The dose is to be regulated by the condition of the patient. From one-half to one ounce daily may be given to an infant of one year. It is rarely advisable to go above this quantity except for a few hours at a time at critical periods. Stimulants are most needed when the temperature is low, or falls suddenly, as at the crisis of the disease. When the temperature is high, smaller amounts are generally required.

In many cases strychnine is even more valuable than alcohol. Usually they should be combined, as the indications are the same. When the dose is to be repeated every three hours, $\frac{1}{300}$ of a grain is as much as it is wise to give to an infant a year old. This may be kept up for days, and for a shorter time larger doses may be given, the effect always being carefully watched. For older children digitalis may be used, but I have rarely seen much benefit from it in infants. In attacks of heart failure associated with pulmonary congestion, nitroglycerin should be given, to a child of one year gr. $\frac{1}{500}$ every hour.

Respiratory stimulants are needed in most cases, even more than are cardiac stimulants, but we have none which can be wholly depended upon. For a short time, atropine gr. $\frac{1}{400}$, caffein gr. $\frac{1}{8}$, or strychnine gr. $\frac{1}{200}$, may sustain a child with sudden failure of respiration, but in the slow respiratory failure that results from exhaustion their effect is but temporary. The doses mentioned are for an infant of one year. The drugs may be used successively or together; for immediate effect they should be given hypodermically. Oxygen may be classed with the respiratory stimulants. It may be given continuously, but always mixed with atmospheric air. To the rubber tube coming from the cylinder a glass funnel may be attached and held one inch from the child's face. Gentle friction of the chest wall, without disturbing the patient, is sometimes useful in stimulating the respiratory muscles, especially in protracted cases.

Antipyretics.—It must be remembered that the normal range of temperature in broncho-pneumonia is from 101° to 104·5° F. This temperature is not in itself exhausting, and the chances of recovery are not, I think, improved by systematic efforts at reducing it so long as it remains within these limits. Too much can not be said in condemnation of the practice of giving such drugs as phenacetine and other coal-tar products in full doses for the reduction of temperature. In small doses they are often useful to allay nervous irritability, restlessness, and promote sleep. Quinine can not be considered an antipyretic in pneumonia except in cases complicated by malaria. Otherwise it does little if any good, and often great harm, by disturbing the stomach.

Antipyretic measures are indicated in cases of hyperpyrexia, which we may define as 105° F. or over, or when extreme nervous symptoms exist, even though the thermometer may not register the degree mentioned. Under these circumstances, the most certain, the most within our control, and hence the safest antipyretic, is cold. It may be used by the evaporation bath, the cold pack (pages 49, 50), sponging, cold compresses, or an ice-bag applied to the chest.

The most convenient and efficient methods of using cold are the bath and the cold pack—the bath for infants, and the pack for older children. The peripheral circulation should be closely watched, and maintained by friction of the body during the bath, and the application of heat to the extremities immediately after it. In most cases the bath should be preceded by stimulants. The effects are often very striking; when there have been a flushed face, hot dry skin, extreme restlessness, and muscular twitchings, all these symptoms may subside rapidly and a quiet sleep follow.

The bath should be repeated as soon as these symptoms return, whether the thermometer has risen to its former height or not. Not all children bear cold well, and in its use and frequency of repetition one must be guided by its effect upon the child's general condition as well as upon the temperature. When with hyperpyrexia we have general cyanosis, cold surface, feeble pulse, shallow respiration, and stupor, cold is contraindicated and a hot mustard bath should be used.

Inhalations.—These are of more value in relieving cough and in promoting bronchial secretion than any other means we possess. The same substances are to be used, and in the same way as mentioned in the article on Bronchitis.

The *nervous symptoms,* restlessness, loss of sleep, etc., are often best controlled by cold or tepid sponging; in other cases by small doses of phenacetine—i. e., one grain every three hours to a child of six months. Opium is to be avoided unless there is severe pain, which is very rare; or, when the incessant cough is not relieved by inhalations. Codeine may be given in doses of gr. $\frac{1}{20}$, or heroin gr. $\frac{1}{100}$, every three or four hours to a child of one year.

Sudden *attacks of general collapse* with cyanosis are frequent in severe cases of broncho-pneumonia. They may come on at any period in the disease. When occurring in the early stage, if promptly and energetically treated, recovery may take place, but when they come on in the late stages they are usually fatal. They may be due to acute congestion or œdema of the lung not previously involved. The most efficient treatment is to put the child into a hot mustard bath (page 56), to use strychnine and nitroglycerine hypodermically, and to give oxygen continuously. For a few hours alcohol should be given freely. A valuable remedy for immediate effect is adrenalin; from one to three minims of the 1–1,000 solution may be used hypodermically. It should be injected deep into the muscles.

Treatment of protracted cases.—When the fever continues for five or six weeks, with no disposition on the part of the disease to subside, about all that can be done is to continue the sustaining treatment adopted in the earlier part of the disease—careful feeding, judicious stimulation, and proper hygienic means. Many of these cases will recover if the patient's strength holds out; but, unfortunately, in the majority the continuance of the pneumonic process is in itself evidence of the weakened vitality of the patient, and, though he may live a long time, most such attacks ultimately prove fatal.

When the fever has disappeared, and there is only a persistence of the physical signs and the general cachexia, the cases are more hopeful. Here, a change of air is more important than all other means of treatment. If in the winter or spring the child can be removed to a warm, dry climate where he can be kept in the open air, or if, in the summer, he can be taken to the mountains, immediate improvement is often seen, followed by rapid recovery. This experience we see repeated every year with hospital patients when they are transferred from the city to the country in May or June. With the change of air a general tonic plan of treatment should be followed, cod-liver oil, arsenic, iron, and quinine being used, according to the indications in each particular case.

One should never declare one of these cases of protracted pneumonia to be hopeless, nor should he be too ready to assume that tuberculosis is present because the child is wasted and anæmic, and the physical signs have persisted. In private practice the cases of simple protracted pneumonia outnumber the tuberculous ones, three to one.

Summary.—In the treatment of broncho-pneumonia it should be borne in mind that, while very little can be done for the disease, very much can be done for the patient. The hygienic measures generally grouped under the term "careful nursing" are of great importance, and many of the mild cases need no other treatment. One should watch the digestive organs closely, keep the bowels freely open, and not allow the abdomen to become distended with gas, since this often seriously interferes with the action of the diaphragm. In severe cases, the patient may be in great danger in the early stage from two causes: first, from the intensity of the general infection, which is best combatted by the use of alcohol and strychnia; and, secondly, from the mechanical embarrassment of the heart and respiration, in consequence of the sudden interference with the function of the lungs, partly from inflammation, but chiefly from congestion; this is best relieved by counter-irritation to the chest and heat to the extremities. During the later stage the principal danger is from exhaustion; this forbids the use of all depressing measures, and necessitates the most careful attention to the nutrition of the patient throughout the disease. All unnecessary medication is to be avoided, particularly the use of expectorant mixtures, on account of the disturbance of the stomach. Opium is to be used very sparingly, and in most cases it should be withheld altogether. The cough is best relieved by inhalations of creosote, and the nervous symptoms by phenacetine or baths. For local use, poultices should be discarded and the oiled-silk jacket used only when the temperature is not high. Counter-irritation by mustard should be continued throughout the attack, when there is much bronchitis. Where antipyretics are required, cold is safer and more efficient than the use of drugs. Of the cardiac stimulants, alcohol and strychnia are most to be depended upon. Care should be taken in all cases to maintain a good peripheral circulation. In sudden general collapse, the most valuable measures are hot mustard baths, strychnia or adrenalin hypodermically, alcohol freely by the mouth, and the inhalation of oxygen. In protracted cases, and in those with delayed resolution, change of air is more important than all other means combined.

CHAPTER V.

DISEASES OF THE LUNGS.—(*Continued.*)

LOBAR PNEUMONIA.

Synonyms: Fibrinous pneumonia, croupous pneumonia, pneumonic fever.

WITH our present knowledge, lobar pneumonia may be best defined as an infectious disease, caused by the micrococcus lanceolatus (pneumococcus) and accompanied by a local lesion in the lungs. While in most cases the general symptoms correspond with the extent and severity of the local lesion, they may be out of all proportion to each other.

Etiology.—*Age.*—Lobar pneumonia may occur at any age. I have recently seen a case in an infant of three months which followed the typical course. It may be seen even in the newly born, but it is not until after the second year that it begins to be frequent. After the third year nearly all the cases of primary pneumonia are of this variety.*

Of 160 personal cases, and 340 collected from various sources, the ages were as follows:

AGE.	Cases.	Per cent.
During the first year	76	15
From the second to the sixth year	309	62
" " seventh to the eleventh year	104	21
" " twelfth to the fourteenth year	11	2
Totals	500	100

The greatest susceptibility appears to be from the second to the sixth year, and during this period it is most frequent from the third to the fifth year.

Sex.—Of my own cases, 60 per cent were males, and the same proportion was noted in 544 collected cases. This predominance of males has been everywhere observed, but is as yet unexplained.

Season.—In my series of cases, the seasons were divided as follows:

	Cases.	Per cent.
In the three winter months	48	35
" " spring "	62	46
" " summer "	6	4
" " autumn "	20	15
Totals	136	100

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* For the relative frequency of broncho- and lobar pneumonia during infancy, see the table in the introductory chapter on pneumonia.

Lobar pneumonia, in children therefore, as in adults, occurs most frequently during the spring months. April shows the largest number of any single month.

Previous condition.—In my hospital cases, 82 per cent of the children were previously in good condition, and only 18 per cent were delicate, rachitic, or syphilitic. This observation has been borne out by my experience in private practice—viz., that as a rule lobar pneumonia affects children who were previously healthy.

Previous disease.—Previous attacks of pneumonia are observed in but a small proportion of cases. It was noted only five times in 160 cases. In the vast majority of cases lobar pneumonia is a primary disease, although it occasionally occurs as a complication of pertussis, measles, typhoid or scarlet fever, and even diphtheria—chiefly, however, in children over three years old.

Epidemics of lobar pneumonia I have never witnessed, although on several occasions I have seen two children in a family attacked either simultaneously or in rapid succession. Exhaustion, fatigue, and exposure are to be ranked as associated exciting causes.

In addition to other causes, there is required for the production of the disease the presence and growth of the pneumococcus.

Lesions.—*The seat of the disease.*—In 950 cases in children under fourteen years, this was as follows:

SEAT OF DISEASE.	Personal cases.	Collected cases.	Totals.
Right lung, upper lobe only........	39	137	176
“ “ middle “ “	8	4	12
“ “ lower “ “	26	142	168
“ “ more than one lobe............	13	64	77
Totals, right lung......................	86	347	433
Left lung, upper lobe only..................	25	68	93
“ “ lower “ “	49	214	263
“ “ more than one lobe..............	9	29	38
Totals, left lung........................	83	311	394
Both lungs, upper lobes....................	..	13	13
“ “ lower “	3	38	41
“ “ elsewhere......................	9	60	69
Totals, both lungs......................	12	111	123

The right lung was thus affected in 45·5 per cent; the left lung in 41·5 per cent; both lungs in 13 per cent. In the order of frequency, the disease involves, first, the left base; second, the right apex; third, the right base; fourth, the left apex. The disease affects, as a rule, a single lobe, and often only a circumscribed portion of a lobe, stopping sharply at the interlobar fissure.

Lobar pneumonia among children is so rarely fatal that the opportunities for a study of the peculiarities of the lesion have been somewhat limited. I have myself made eleven autopsies, and have among my hospital records reports of nine others, making twenty cases in all. The anatomical changes resemble those seen in the adult lung. There is an exudation into the alveoli and smaller bronchi of fibrin, serum, leucocytes, and red blood-cells (Fig. 87). There is usually in addition an inflammation of the mucous membrane of the larger bronchi and of the pleura. The frequency and severity of the pleurisy is a peculiarity of the lesion in children.

In the first stage, that of *congestion*, the portion of lung involved is dark-coloured, heavy, and œdematous, and shows under the microscope a serous and cellular exudation into the air vesicles, with swelling of the epithelial cells lining the alveoli.

In the second stage, that of *red hepatization*, there is usually some exudation upon the pulmonary pleura, generally a thin layer of fibrin, giving it a dull, granular look. The lung itself is of a uniform dark-red colour. It is solid, and cuts like liver. It looks as if it had been inflated to its utmost extent and then injected with a material which had solidified. The consolidated area is sharply defined. Under the microscope the air vesicles are seen to be distended with an exudation which is chiefly fibrin, but with some leucocytes, red blood-cells, and desquamated epithelial cells. The cells are chiefly leucocytes, and are usually more abundant than in the pneumonia of adults.

In the third stage, that of *gray hepatization*, the lung is more moist, and the inflammatory products are partly decolourized. This change takes place irregularly throughout the lung, giving it a mottled appearance.

The fourth stage, that of *resolution*, follows gray hepatization, and consists in the degeneration and liquefaction of the products of inflammation, which are ultimately carried away by the lymphatics, or pushed out into the bronchi and removed by coughing.

The duration of the stage of congestion is from a few hours to several days; that of the stage of red hepatization from two days to two or three weeks. This is the condition in which the lung is most often seen at autopsy. The stage of gray hepatization is commonly shorter. Resolution usually begins when the temperature falls to normal, but occasionally it may be delayed for several days. It is generally complete in about a week.

Variations in the lesions.—(1.) Instead of clearing up at the usual time, the lung may remain consolidated for several weeks, and then resolve. (2.) The stage of gray hepatization may be followed by a great exudation of pus cells, which may everywhere infiltrate the affected lung; or these may be circumscribed so as to form a single large abscess or many small ones. (3.) There may be small areas of gangrene. All these conditions are very rare in children. Purulent infiltration and delayed resolution were noted in none of my cases, and gangrene but once. (4.) There may be excessive pleurisy, or pleuro-pneumonia. This was found in one-half of my autopsies. These cases will be separately considered elsewhere.

Lesions in other organs.—With pneumonia of the left side, if complicated by pleurisy, there may also be pericarditis. This is seen even in infants. The pericardial inflammation closely resembles that of the pleura. There is a very abundant exudation of fibrin and pus, coating both surfaces of the pericardium. Acute meningitis was met with twice in my cases. The form was an acute purulent meningitis, with a very abundant exudation of greenish-yellow lymph, chiefly at the convexity. In one of my cases peritonitis was also present. As the pneumococcus is found in all these inflammations, they may be regarded as examples of a more generalized infection than usually occurs. In most of these the other processes are secondary to that in the lungs, but sometimes they begin simultaneously with, or may even precede, the pulmonary lesion. In a very small proportion of cases the pneumococcus is found in the blood, spleen, the kidney, and liver—i. e., a general pneumococcus septicæmia.

The heart is generally found in diastole, with the cavities, especially those of the right side, distended with soft clots. There may be found ante-mortem thrombi, which may extend into the pulmonary artery or the aorta.

Symptoms.—(1.) *The typical course.*—A child three or four years of age, after a few hours of slight indisposition, is suddenly taken with vomiting, followed by a rapid rise in temperature. He is dull and heavy, complains of headache and general weakness, refuses food, and is easily persuaded to remain in bed. He has the appearance of being quite ill, even after a few hours. Occasionally sharp pain in the side is complained of. The skin is dry; there are marked thirst, restlessness, and the other symptoms which accompany fever. The temperature is found to be 104° F., or even higher; the respirations 40 to 50 a minute; the pulse full, strong, and 120 to 130. On the second day the patient is no better. The temperature remains high; the tongue is coated; the anorexia continues; the pain is more severe; cough is present and may be quite frequent.

After the second or third day the patient is usually more comfortable, and sleeps better, but may be disturbed by the cough. At times there is restlessness, and at night there may even be slight delirium. The respiration continues rapid and the temperature high. These general symptoms show very little change until the sixth or seventh day, when, after a long sleep, which has been more natural than before, the patient wakes, decidedly improved as to all his symptoms. There is less fever, and the temperature continues to fall rapidly until it touches the normal line, or it may even go below this. As the fever subsides the pulse drops to 90 or 100, and the respirations to 25 or 30 a minute. The appetite soon returns, and convalescence is usually rapid. In a week the patient is out of bed, and in a month from the beginning of the illness he is out of doors; but it may be another month before he can be considered to have entirely recovered. This is the course seen in fully two-thirds of all the cases of lobar pneumonia at this age.

(2.) *Pneumonia of short duration.*—Instead of running the usual course of from five to eight days, cases are seen in which the duration is only three or four days, although the physical signs indicate that the process in the lung passes through the usual stages. These differ from the ordinary type chiefly in their duration. They are always mild.

(3.) *Abortive pneumonia.*—This form of the disease is rarely seen in hospitals, but it is not infrequent in private practice where the physician is summoned at the earliest signs of illness. The onset is precisely like that of ordinary pneumonia, and may even be as severe as the average case. The physical examination of the chest gives all the signs of the first stage of the disease, but on the second or third day the physician is greatly surprised to find that the temperature has fallen to normal, and that all the physical signs have disappeared. The process in such cases does not seem to go beyond the first stage of congestion; there is no evidence of hepatization of the lung. The course is often such as to lead the physician to the opinion that he has made a mistake in his diagnosis. There seems, however, to be no doubt that these are cases of genuine pneumonia. D'Espine found the pneumococcus in the sputum of such a case. This type of pneumonia corresponds with abortive types of other infectious diseases so frequently met with in children. The temperature curve in such a case is shown in Fig. 106. The diagnosis of these cases is always attended with some uncertainty. There can be no doubt that very many of the unexplained high temperatures of brief duration which are seen in children are from this cause. Exactly why it is that the disease sometimes terminates in this way can not always be explained. It may be because the resistance of the patient is greater than usual, or the virulence of the pneumococcus is less.

(4.) *The prolonged course.*—Although usually lasting about a week, it is not rare for pneumonia to continue ten, twelve, or even fifteen days. This prolonged course is usually due to the fact that the disease spreads from one part of the lung to another, or even to the opposite lung, involving in succession two, three, or more lobes. This is sometimes known as "creeping" pneumonia; it is always severe and the outlook is generally unfavourable. A prolonged temperature with physical signs limited to a single lobe should always suggest complications, most frequently empyema, occasionally pericarditis.

(5.) *Cerebral pneumonia.*—This term was first applied by Rilliet and Barthez to cases of pneumonia in which the cerebral symptoms predominate. They will be considered later.

Onset.—Prodromal symptoms of more than a few hours' duration are quite rare. The onset of lobar pneumonia is almost invariably sudden, with well-marked symptoms—vomiting, diarrhœa, chill, or convulsions. Vomiting is altogether the most frequently seen. It was the mode of onset in about one half my cases. In summer particularly, there may be vomiting and diarrhœa. A distinct chill is rare in a child under five years of age, and is not very common even in older children. Convulsions are not very infrequent, being seen in about five per cent of the cases. Their occurrence depends upon the suddenness of the invasion and the susceptibility of the patient.

Cough.—This is present in most of the cases throughout the disease, but often is not marked for the first day or two. It is seldom a distressing symptom. A disposition to suppress the cough on account of pain is very frequently noticed.

Expectoration.—This is rarely seen in childhood, and practically never under five years of age. Children of ten or twelve may have the same expectoration as adults—white and viscid, or brownish-red early in the disease, yellow and abundant toward its close.

Pain.—Headache and general muscular pains in the back and extremities are frequent during the invasion. The characteristic pain, however, is pleuritic. It is not necessarily felt in the region of the affected lung, and often not in the chest at all. It is frequently referred to the loin, the epigastrium, or to any region to which the intercostal nerves are distributed. In a recent case, in a boy of seven years, for the first twelve hours there was intense localized pain in the right iliac fossa, associated with such extreme tenderness as to lead to the suspicion that the case was one of appendicitis. The pain may last throughout the disease, and occasionally it is a most distressing symptom; but usually it is only moderate, and rather more severe early than late in the disease.

Prostration.—This is one of the characteristic features of pneumonia. The patient is generally willing to go to bed on the first day of the attack, and shows little desire to leave it while the disease continues. "Walking cases" are not common in children.

Respiration.—This is always accelerated, and generally out of proportion to the pulse. The normal ratio of the respiration to the pulse is one to four; in pneumonia, frequently one to two. The respiration is not laboured and not quite panting, although this term is sometimes used to describe it. It is jerky. There is a short inspiration, then a momentary pause, followed by a quick expiration, which is accompanied by a short moan. This expiratory moan is very characteristic. The rapidity of respiration is usually in proportion to the amount of lung involved, but it is also modified by the temperature, as the respirations often drop from 60 to 30 in the course of a few hours at the crisis.

Pulse.—In the early part of the disease this is frequent, full, and strong, from 120 to 150 a minute. Later it may be weak, small, compressible, and sometimes irregular. It is much more rapid in the child than in the adult, 160 and 180 being often seen in cases not especially severe. The pulse rate is of less importance than its character.

Temperature.—The typical temperature curve of lobar pneumonia (Fig. 103) is characterized by an abrupt rise usually to 104° or 105° F., and by daily fluctuations generally within the limits of two or three degrees until the crisis, at which time the temperature falls to normal, usually in the course of twenty-four hours. After this time it does not go above the normal line. Such a curve is seen in the majority of cases over three years of age.

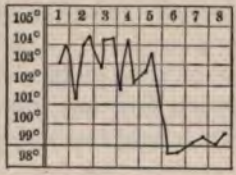


Fig. 103.—Typical temperature curve of lobar pneumonia.

History.—Male, three years old; in fair condition; sudden onset; signs of consolidation—bronchial respiration and voice, and dulness—over left lower lobe behind, not distinct until the morning of the fifth day. On the seventh day the lung was resolving.

In cases under three years of age it is not uncommon for the temperature to be of a more or less remittent type (Fig. 104).

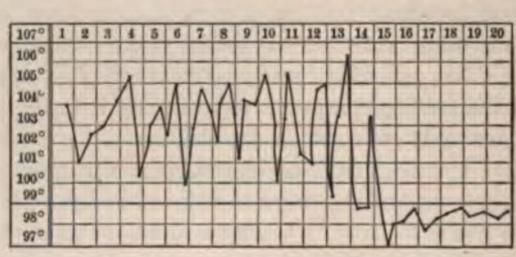


Fig. 104.—Lobar pneumonia with remittent temperature.

History.—Female, eighteen months old; in fair condition; sudden onset; repeated examinations of chest made, but no abnormal signs until the ninth day, when there were very rude respiration and slight dulness at the right apex, in front; on the twelfth day all the signs of consolidation at the same point, no râles; four days after the crisis the lungs were clear.

These wide fluctuations often lead to great difficulty in diagnosis, particularly if the physical signs appear late, as they not infrequently do. It is possible that some of them are to be explained by mixed infection.

The following chart (Fig. 105) illustrates three features which are often seen in pneumonia: (1) A temperature which early in the disease is steadily high and as the day of crisis approaches becomes remittent; (2) a secondary rise after being normal for twenty-four hours, which was due in this instance to an extension of the disease to a new part of the lung; (3) a fall to a point considerably below normal at the time of the crisis. In this case the temperature fell in the course of eighteen hours from 105° to 95° F., and later still lower; it was two days before it finally remained at the normal point. A fall to 96·5° or 97° F. at the time of crisis is not uncommon.

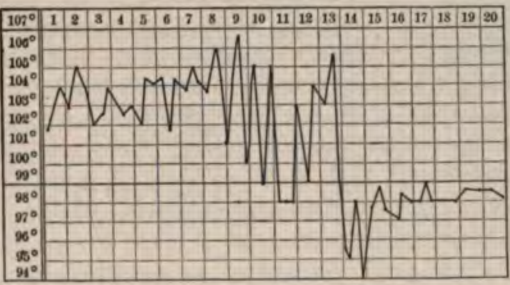


Fig. 105.—Lobar pneumonia with subnormal temperature after the crisis.

History.—Female, nineteen months old; fairly healthy; sudden onset; symptoms typical but physical signs delayed; consolidation in left mammary region on the eighth day; on the ninth in right lung middle lobe; on the eleventh day a pseudo-critical drop, followed after twenty-four hours of apyrexia by a further rise, which was accompanied by signs of extension of the disease in the right lung. Resolution rapid after crisis.

In the foregoing cases the fever terminated by crisis. In Fig. 106 is shown one ending by lysis. This is a mode of termination much more frequent in young children than in those who are older. Thus, in ninety-three of my own cases, nearly all of which were under three years of age, the fever ended by crisis in forty-nine, and by lysis in forty-four; while in five hundred and twenty-two collected cases, the majority of which were in older children, three hundred and ninety-six ended by crisis, and one hundred and twenty-six by lysis.

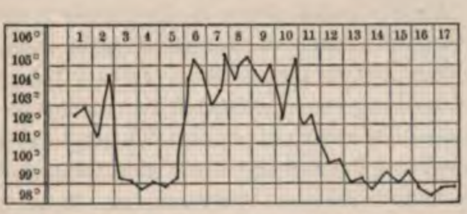


Fig. 106.—Abortive pneumonia in left lung, followed by typical pneumonia in right lung, terminating by lysis.

History.—Male, seventeen months old; healthy; sudden onset; on the second day disseminated fine râles in both lungs behind, and over left lower very feeble respiration, high-pitched—i. e., some bronchitis, with congestion (?) of left base. On the third, fourth, and fifth days, general symptoms gone and signs nearly disappeared. On the sixth day all symptoms of pneumonia, and on the seventh distinct consolidation of right base, rest of chest clear. Subsequent course typical; resolution rapid and complete.

The following table shows the day of crisis in five hundred and sixty-seven cases of lobar pneumonia in children who recovered:

The Day of Crisis.

Second day	3 cases.
Third "	22 "
Fourth "	43 "
Fifth "	88 "
Sixth "	83 "
Seventh "	132 "
Eighth "	73 "
Ninth "	55 "
Tenth "	22 "
Eleventh day	18 cases.
Twelfth "	7 "
Thirteenth day	8 "
Fourteenth "	7 "
Fifteenth "	1 case.
Eighteenth "	3 cases.
Twenty-first day	1 case.
Twenty-sixth "	1 "
	567

From this table it will be seen that the most frequent critical day is the seventh, and that in 66 per cent of the cases it was from the fifth to the eighth day. The causes of a post-critical rise in the temperature are chiefly two—extension of the disease to a new area, or the development of pleurisy, which is apt to be purulent. Less frequently it is due to meningitis, pericarditis, gastro-enteritis, or malaria. In fatal cases the temperature is generally high until the end. In general, it may be said that the temperature is considerably higher in children than in adults; in the majority of cases it reaches 105° F., the usual range being from 102° to 105° F. In fifteen of one hundred and thirty-seven cases, or 11 per cent, it reached 106° F. or over.

Gastro-enteric symptoms.—These are more common in infants than in older children. At the onset there is frequently vomiting, sometimes also diarrhœa. A continuance of the vomiting is rare, and is generally due to improper feeding or medication. It may be a very serious complication. Diarrhœa is also rare, except at the onset and in summer cases. It is sometimes seen at the time of crisis. Throughout the disease there are anorexia, coated tongue, and the usual symptoms of high fever.

Nervous symptoms.—Cerebral symptoms are frequent and very often misleading. In seven of my cases the pneumonia was ushered in by convulsions. These differ in no respect from convulsions from other causes, and may be repeated two or three times in the course of the first twenty-four hours. They are sometimes followed by drowsiness or stupor, sometimes by active delirium. Cerebral symptoms may predominate for several days. There may be opisthotonus, dilated or contracted pupils, irregular pulse, retracted abdomen, and, in fact, almost every symptom of meningitis. Occasionally the decubitus *en chien de fusil*, or gun-hammer position, is assumed. These are often described as cases of *cerebral pneumonia*, and in many of them pneumonia is not suspected until the fourth or fifth day of the disease, sometimes not until the crisis occurs, when the rapid disappearance of all these nervous symptoms indicates their origin. Early convulsions are not generally followed by an especially severe type of the disease, only one of seven cases beginning in this way proving fatal. On the other hand, cases with late convulsions are usually fatal. In two of the three cases in which I have noted them, the convulsions ushered in an attack of meningitis.

Delirium is much more frequent than convulsions, and is seen in nearly one fourth of the cases. Generally it is slight, and noticed only at night or when the temperature is very high. It is usually mild, but may be low and muttering, like that of typhoid, or wild and active, like that of cerebro-spinal meningitis. It is most pronounced at the height of the disease. Other nervous symptoms belonging to the typhoid state, such as incontinence of urine or fæces, muscular twitchings, and tremor of the tongue or protrusion, are occasionally seen, but only in the worst forms of the disease.

There is no relation between the seat of the disease in the lungs and the occurrence of cerebral symptoms. They are more frequent in children under five years than in those who are older, and depend upon the suddenness of the invasion, the intensity of the infection, and the susceptibility of the child. Late in the disease they may indicate exhaustion, toxæmia, or complicating meningitis. They are frequently associated with very high temperature and extensive disease. The usual nervous symptoms—restlessness, headache, sleeplessness, etc.—are nearly always proportionate to the height of the temperature.

Urine.—Throughout the febrile period of the disease the urine is scanty, high-coloured, with a high specific gravity, and usually loaded with urates. In a small number of cases a trace of albumin may be found, and occasionally a few hyaline casts. Evidences of serious renal disease I have seldom found in lobar pneumonia, and in the experience of all observers it is extremely rare in early life.

Skin.—The face, in pneumonia, is usually flushed, sometimes on both sides and sometimes only on one; in other cases it is pale, but not indicative of pain. Cyanosis is rare except toward the close of the disease and is usually a sign of respiratory failure. Herpes of the lips or face is quite frequent.

Blood.—The leucocyte count is of considerable value both from a diagnostic and a prognostic standpoint. For a discussion of this subject see the chapter on Diseases of the Blood.

Physical Signs.—The earliest signs in pneumonia are due to the acute congestion of the affected lung or lobe, in consequence of which less air enters this portion and more air the rest of the lungs. Percussion gives diminished resonance or slight dulness over the affected area, and exaggerated resonance over the remainder of this lung and over the opposite lung. Auscultation over the affected lobe gives feeble respiratory murmur, rather high in pitch; sometimes there may be absence of all breath-sounds so complete as to suggest fluid. The normal respiratory murmur over the healthy portions of the lungs is intensified. In children this exaggerated breathing is not infrequently mistaken for bronchial breathing, and the physician may be led into the error of locating the pneumonia upon the wrong side. Exaggerated breathing does not differ from normal breathing except in intensity, and is heard only on inspiration. Bronchial breathing is higher in pitch, and is heard with nearly equal intensity, both on expiration and inspiration. If the chest is frequently auscultated, crepitant râles (Figs. 107 and 108) may usually be heard at some period at the end of full inspiration, but often they are present but for a few hours, and they may be missed altogether.

In the second stage, that of consolidation (Fig. 109), no air enters the affected part of the lung. Upon palpation there is found here exaggerated vocal fremitus, and on percussion there is marked dulness, but very rarely flatness. Over the rest of this lung there is exaggerated, sometimes even tympanitic, resonance; this is especially frequent at the apex of the lung in front, when there is consolidation at the base behind. Under these conditions cracked-pot resonance may sometimes be obtained. Over the healthy lung there is exaggerated resonance. On auscultation over the consolidated portion there are bronchial breathing and bronchial voice, the area over which they are heard being sharply defined. Râles are usually absent, but there may be pleuritic friction sounds.

In the stage of resolution there is a gradual disappearance of the signs of consolidation. The pure bronchial is replaced by broncho-vesicular breathing, the vesicular element gradually predominating. Moist râles of all varieties are heard. Usually the most persistent signs are slight dulness or diminished resonance, with a respiratory murmur which is feebler than normal and a little higher in pitch; sometimes there are also dry friction sounds. These signs may persist for two or three weeks.

Exceptional physical signs.—While in the majority of cases the signs of consolidation are distinct on or before the fourth day, in not a few they may be delayed much longer. Of eighty-two cases in which the day was noted on which consolidation was found, it was not until the fifth day or later in one fourth the number. In six of them, although carefully and repeatedly examined, no consolidation was found until the seventh day or later and in one case not until the twelfth day. It has been customary to look upon these cases of delayed or concealed physical signs as cases of central pneumonia. That pneumonia may exist in the centre of a lung for a number of days is, to my mind, extremely improbable. At autopsy, superficial pneumonia I have very frequently seen, but central pneumonia never. There are two regions in which pneumonia may exist and yet not be accessible by our means of physical examination, viz., at the apex of the lung in the part covered by the shoulder, and along the posterior border of the lung where it lies against the vertebræ. In either

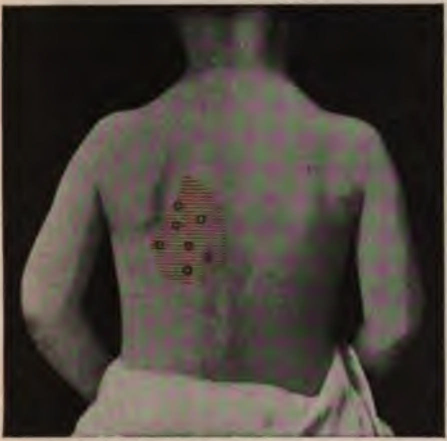


FIG. 107.—First stage. Congestion of left lower lobe, with crepitant râles. Feeble breathing of a rude character, with slight dulness.

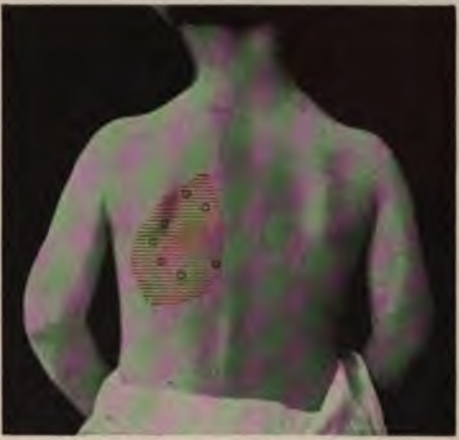


FIG. 108.—In the centre of the area, a small spot of pure bronchial breathing and voice; surrounding this an occasional crepitant râle, with broncho-vesicular breathing and slight dulness.

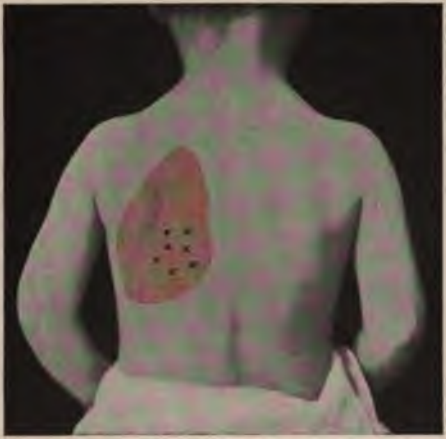


FIG. 109.—Second stage. Complete consolidation of left lower lobe. Pure bronchial breathing and bronchial voice; marked dulness; increased vocal fremitus, and at the lower part a few friction sounds.

NOTE.—During resolution the signs take the inverse order: those of Fig. 109 give place to those of Fig. 108, and these in turn to those of Fig. 107. In addition, many coarse râles may be heard.

of these situations pneumonia may be present without our being able to find it. It is quite common in cases with late physical signs that the first distinctive evidences of disease are found high in the axilla, or beneath the clavicle in front, and these regions should be closely watched in doubtful cases. Sometimes the delay is best explained by assuming that constitutional symptoms due to a pneumococcus infection, may be present for several days before the development of the local lesion in the lung.

Complications.—The occurrence of dry pleurisy over the consolidated portion of the lung is so constant that it can hardly be considered a complication. A slight serous exudation of two or three ounces is not uncommon, but more than this is very rare in young children. In the most severe cases of pleurisy there is an excessive exudation of fibrin and pus. This occurred in eight per cent of my cases. This variety is known clinically as pleuro-pneumonia, and will be considered separately. Pericarditis is rare; it was seen only twice in the series of cases reported, being associated with pleuro-pneumonia of the left side. It rarely gives rise to any new symptoms. Endocarditis was not seen in my cases, though it occasionally occurs. Meningitis is rare, and generally develops late in the disease. It is nearly always ushered in by repeated attacks of vomiting or convulsions. Its course is short and progressive. Peritonitis causes few new symptoms except abdominal distention, pain, and tenderness.

Course and Termination.—In the great majority of cases lobar pneumonia terminates either in perfect recovery or in death. When ending in recovery, resolution commonly begins immediately upon the cessation of the fever, and is complete in about a week. Delayed resolution is not common in children; chronic pneumonia and tuberculosis are rare sequelæ, but empyema is very common. Its symptoms sometimes develop immediately after the pneumonia, the temperature continuing high; or there may be an interval of a few days before the development of the pleural symptoms. Some pleuritic adhesions probably remain in every case in which there has been much dry pleurisy, and when severe and extensive, these may be the cause of subsequent symptoms, like any other dry pleurisy.

Death from uncomplicated pneumonia may be due to exhaustion, or to heart failure, with or without failure of the respiration. The signs of heart failure sometimes develop quite rapidly in cases which are apparently doing well. The symptoms are: coldness of the hands and feet, then of the legs and arms; a rapid, compressible, and sometimes irregular pulse; muscular weakness and pallor, but usually no cyanosis. The symptoms of respiratory failure are: very rapid superficial respirations, sometimes 100 a minute; blueness of the lips and finger nails; often a leaden hue of the whole body; there are loud tracheal râles, and recession of all the soft parts of the chest on inspiration.

Death may result early in the disease, where the pneumonia has spread rapidly, involving both lungs. The earliest deaths I have seen were on the fourth day, and were due to a failure of the heart and respiration. In most of the uncomplicated fatal cases, death results from heart failure at about the time of the crisis. In the complicated cases death usually occurs in the second week. I once knew fatal meningitis to develop at the end of the fourth week.

Diagnosis.—The most characteristic differences between broncho- and lobar pneumonia are shown in the following table:

BRONCHO-PNEUMONIA.	LOBAR PNEUMONIA.
1. More than half the cases secondary.	1. Almost always primary.
2. Under three, chiefly under two years.	2. Most common between three and eight years.
3. Occurs more frequently in delicate and debilitated children.	3. More often in those previously healthy.
4. Bacteria—in primary cases, usually the pneumococcus; in secondary cases, usually mixed infection.	4. The pneumococcus, very often alone.
5. Products of inflammation chiefly cellular; process often diffuse.	5. Chiefly fibrin; process circumscribed.
6. Onset often gradual, sometimes insidious, especially when secondary.	6. Onset sudden, with well-marked symptoms.
7. No typical course; fever often lasts three or four weeks; rarely terminates by crisis.	7. Typical course; crisis usually from fifth to eighth day.
8. Involves both lungs as a rule, most frequently lower lobes posteriorly.	8. Usually one lobe or a part of a lobe; left base most frequently, right apex next.
9. Signs of bronchitis mingled with those of consolidation; râles in other parts of the same lung, or in the opposite lung, throughout the disease.	9. Râles only early, and during resolution; frequently no signs in opposite lung.
10. Consolidation later—fourth to seventh day; there may be none; apt to be incomplete; shades off gradually.	10. Consolidation earlier; second or third day. Consolidation complete; area usually sharply defined.
11. Resolution slow, one week to two months; often incomplete; strong tendency to become chronic.	11. Resolution rapid, usually complete within a week.
12. Relapses and second attacks frequent.	12. Both are rare.
13. Sequelæ: Empyema, chronic interstitial pneumonia, sometimes tuberculosis.	13. No sequelæ except empyema.
14. Prognosis always serious from the age and the circumstances under which disease occurs.	14. Prognosis good; rarely fatal except from complications—empyema, meningitis, pericarditis.
15. Hospital mortality 50 per cent of primary cases, 65 per cent of all cases.	15. Mortality 4 per cent of all cases.

In the majority of cases the symptoms are plain and the physical signs so typical that it is difficult to overlook pneumonia if any degree of care is used in the examination of the patient. The characteristic features are the sudden onset, with vomiting, convulsions, or chill; prostration; rapid respiration, with the expiratory moan; a temperature of 102° to 105° F.; cough and thoracic pain; and the physical signs of a rapidly developing, circumscribed consolidation in one lobe or a portion of a lobe. The difficulties in diagnosis are due to the great variation that is seen in the general symptoms, and to the late appearance of the physical signs. The error usually made is to mistake pneumonia for some other disease, rather than to mistake some other disease for pneumonia. On account of its frequency in children, pneumonia should always be excluded before accepting any other explanation of a continuously high temperature. It is surprising to find how often obscure and indefinite symptoms accompanied by high fever, are due to pneumonia. The rule should be followed, in all cases of acute illness, of making a thorough examination of the chest daily until the diagnosis is clear. If to high temperature rapid respiration is added, one should always suspect the lungs, no matter what the other symptoms may be. It not infrequently happens that the general symptoms are quite characteristic and yet the physical signs appear late. In such cases pneumonia should always be looked for high in the axilla or just beneath the clavicle, since it is particularly in the cases of apex pneumonia that this obscurity is likely to exist. If frequent and thorough examinations of the chest are made, very few cases will be overlooked.

In their onset, scarlet fever, tonsillitis, and gastro-enteritis may all resemble pneumonia. Scarlet fever is recognised by the sore throat and the characteristic eruption on the second day; tonsillitis, by the local symptoms. In infancy, pneumonia often begins with vomiting and sometimes there is also diarrhœa, which may lead one to mistake the disease for gastro-enteritis. The constitutional symptoms of influenza often closely resemble those of pneumonia; the diagnosis is frequently in doubt for several days until definite physical signs of pneumonia make their appearance. Malaria is distinguished from lobar pneumonia by the points mentioned in the diagnosis of broncho-pneumonia. From all other general diseases, pneumonia is to be differentiated by the physical signs.

Pneumonia with marked cerebral symptoms sometimes resembles cerebro-spinal meningitis. In both we may have the abrupt onset, convulsions, delirium or stupor, opisthotonus, and prostration. In pneumonia the temperature is more often steadily high than in meningitis; the pulse is never slow and intermittent; the respiration is rapid; the stupor is usually less profound; and there are no localized paralyses. In meningitis there is usually a steady increase in the severity of the nervous symptoms for the first three or four days; in pneumonia they are as a rule most marked during the first twenty-four or forty-eight hours, and then gradually diminish, always subsiding completely at the crisis. While most of the individual symptoms belonging to meningitis may be present, they are usually less severe and less persistent in pneumonia.

The question sometimes arises, in a case of pneumonia, whether the cerebral symptoms are functional, or whether meningitis also exists. If the nervous symptoms are present from the beginning, there is probably no meningitis. If they develop suddenly during the course or toward the close of the disease, meningitis should be suspected.

Lobar pneumonia is to be differentiated from a pleuritic effusion. The most common mistake which I have seen made is to confound empyema with unresolved pneumonia. The latter is very infrequent, so that the probabilities are always strongly in favour of the diagnosis of empyema. In pneumonia rarely, if ever, is the whole lung affected. There is increased local fremitus, dulness, bronchial voice and breathing, and occasional râles of friction sounds. In empyema the whole lung is often affected, there is displacement of the heart, flatness on percussion, diminished or absent vocal fremitus, and although bronchial voice and breathing are present, they are usually distant and feeble. There are no râles or friction sounds. In doubtful cases an exploratory puncture should always be made. Serous effusions give the same physical signs as empyema, but are relatively rare.

Prognosis.—There is probably no disease in which the patient appears so ill, and yet so often recovers completely, as in lobar pneumonia in a child over three years old. Of 1,295 collected cases, chiefly from hospital practice, there were but 39 deaths, a mortality of three per cent. In 187 cases of my own there were 21 deaths, a mortality of eleven per cent. Only one of the fatal cases was over two years old. The difference between the mortality among my cases and the general mortality given, is due to the fact that a large proportion of the first group were observed in children under two years, while of the collected cases the vast majority were in older children. Combining the above figures, we have a total of 1,482 cases with 60 deaths, a mortality of four per cent. In nearly all my cases death was due either to complications or to very extensive disease, as when both lungs were involved, or nearly the whole of one lung. In only one case was an uncomplicated pneumonia of a single lobe fatal.

The prognosis depends upon the age of the patient, the presence or absence of complications, and the extent of the disease. These factors are to be taken into consideration rather than any special symptoms. Early convulsions do not materially affect the prognosis. Of seven such cases only one was fatal. Late convulsions are always very unfavourable, indicating either exhaustion, toxæmia, or the development of meningitis.

The occurrence of vomiting, diarrhœa, or marked tympanites late in the disease is always unfavourable.

A temperature range between 102° and 105° F. is the rule, and within these limits the fever does not affect the prognosis. Even very high temperature does not increase the danger from the disease as much as might be expected. Of fifteen cases in which the temperature reached 106° F. or over, all but three recovered; while of six cases in which it was 106.5° or over, only one died. The highest recorded temperature in my cases—107.5° F.—was in a patient who recovered. A transient rise, even though the temperature may go very high, is seldom serious. Much more serious is a fever which remains steadily above 105° F., as in most cases this accompanies either very extensive disease or pleuro-pneumonia. The continuance of the fever after the tenth day is a bad symptom, for, although the crisis may be postponed until the twelfth day and occur normally, such a prolonged temperature is apt to be an indication of a new focus of disease or the development of complications. In a severe attack, the extension of the disease to a new lobe after the fifth day is always unfavourable. If resolution does not begin soon after the temperature becomes normal a relapse should then be apprehended, or the development of empyema, or some other complication.

Treatment.—In the treatment of lobar pneumonia in children, several cardinal facts are to be kept in mind. It is a self-limited disease, having a strong tendency to recovery in the great majority of cases regardless of the treatment adopted. The fatal cases are almost always in children under three years of age; the rare deaths in older ones are usually due to complications. I believe that there is no means of treatment by which we can abort pneumonia or shorten its course. It follows, therefore, that the indications are, so far as possible, to make the patient comfortable during his illness, to prevent complications, and to treat the individual symptoms as they arise.

In perhaps the majority of cases, hygienic treatment is all that is required. The patient should be kept in bed, no matter how mild the attack; he should be lightly covered, disturbed just as little as possible, and allowed plenty of fresh air in the room. An open window is desirable even though the room temperature is constantly as low as 60° F. Food should be given at regular intervals, seldom oftener than every three hours. It should not be forced when the patient is suffering only from thirst, especially early in the attack when the appetite is often completely lost. Water should be allowed freely at all times.

These measures, careful nursing, an occasional dose of codeine (gr. $\frac{1}{16}$ to a child of three years) when the patient is very restless, fretful, or sleepless, and cold sponging when the temperature makes him uncomfortable, are usually all that is necessary, except to keep a sharp lookout for complications.

Special symptoms may require treatment. When not severe, the nervous symptoms may be controlled by codeine alone or in combination with phenacetine or the bromides. Sometimes sponging with warm water is better than drugs. Severe nervous symptoms, such as delirium, stupor, great restlessness with impending convulsions, when associated with high temperature, call for ice to the head, cold sponging, or the cold pack or bath. Pain, if moderate, may be relieved by counter-irritation by a mustard paste or by a hot poultice; if severe, codeine may be used in addition. The cough is rarely severe enough to require treatment. When it is so severe as to prevent sleep, small doses of Dover's powder or codeine should be given. Antipyretic measures are not necessarily called for even if the temperature is very high. Some nervous children are less disturbed by the temperature than by the means used to reduce it. Under such conditions the temperature should be closely watched, but not necessarily interfered with unless other symptoms develop. The nervous symptoms are a better guide than the thermometer to the use of antipyretics. Cold I believe to be the safest and most certain antipyretic we possess. It may be used as a cold sponge bath, the cold pack or an ice bag to the chest. There is no objection to the bath except the prejudice of the laity. While cold is applied to the trunk the extremities should be closely watched, and heat applied if necessary. The duration of the pack or bath, and the frequency of their use, will depend upon the individual case. In the majority of cases stimulants are not required. They are called for when the pulse is weak, compressible, and rapid, when the face is pale and the extremities are cold. The same stimulants are to be employed, and in the same way, as in broncho-pneumonia. Cardiac stimulants are usually required in larger quantity at the time of and just after the crisis. Respiratory stimulants are indicated as in broncho-pneumonia.

PLEURO-PNEUMONIA.—Under this term are included cases of pneumonia with an excessive amount of pleurisy, the two processes uniting to produce a single clinical type of disease.

In nearly all cases of lobar pneumonia there is a certain amount of inflammation of the pulmonary pleura, and also in those cases of broncho-pneumonia which are accompanied by any marked degree of consolidation. In both of these the pleurisy is usually coextensive with the consolidation. But in certain cases, in both forms of pneumonia, the amount of pleurisy is excessive, and this so modifies the symptoms and course of the disease as to require for them a separate consideration. In some it appears that the inflammatory process begins almost simultaneously in the lung and in the pleura; while in others the pleurisy follows the pneumonia. These cases are, I believe, almost invariably due to the pneumococcus, although in some there is a mixed infection.

In 398 hospital cases of pneumonia there were 27, or 6.8 per cent, which could be classed as pleuro-pneumonia, the diagnosis being confirmed either by autopsy or operation. Of 190 fatal cases, 12·5 per cent were pleuro-pneumonia. Most of these hospital patients were under three years of age, and the disease is, I think, more frequent at this period than in older children.

Lesions.—Of these 27 cases, 17 were classed as broncho-pneumonia and 10 as lobar pneumonia. The left lung was more frequently affected than the right in the proportion of three to two. In most of the cases the pleura covering the entire lung was involved, even though the pneumonia affected but a single lobe, or only a part of a lobe. In nearly half the cases both lungs were involved, but one to a very much less extent than the other. In a small number of cases the pleurisy was limited to the posterior surface of the lung, stopping at the axillary line.

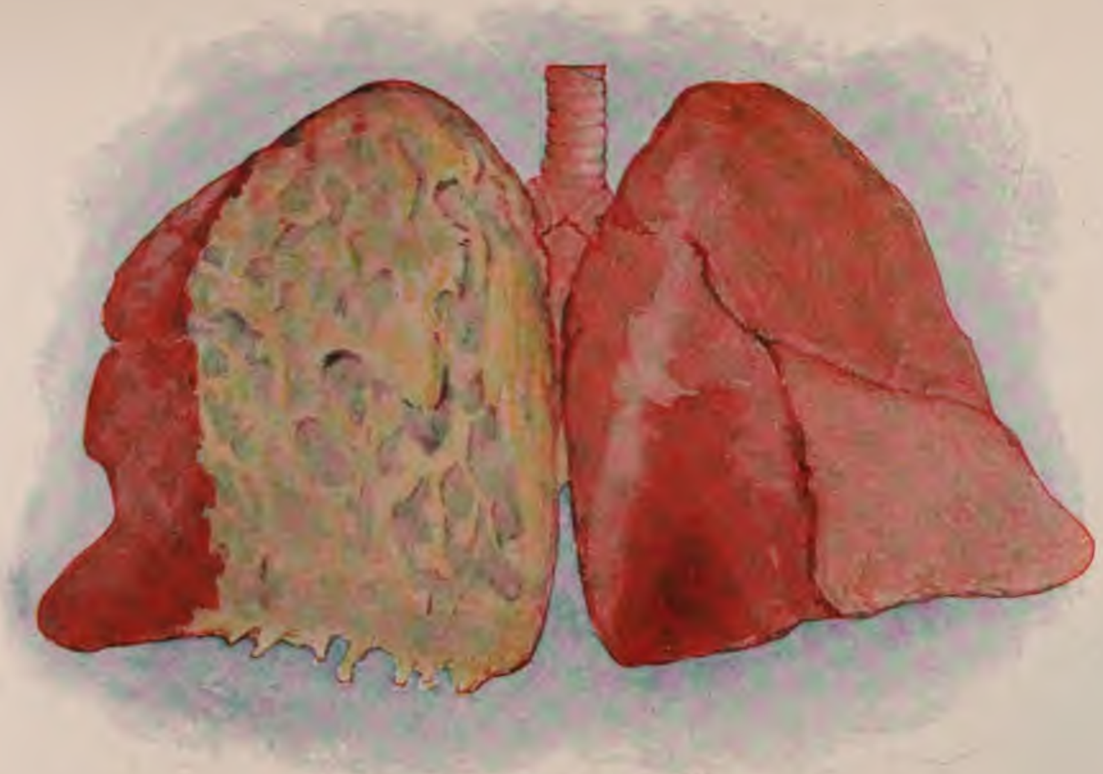
In pleuro-pneumonia both the visceral and the parietal pleura are coated with a layer of yellowish-green fibrin, in thick, shaggy masses, by which the lung is adherent to the chest wall, the diaphragm, and the pericardium (Plate XII). The exudation varies between one eighth and one half an inch in thickness. It can often be stripped from the lung or scraped from the chest wall by the handful. In its meshes small pockets may form, which contain only a few drops, or sometimes a drachm of pus, or less frequently serum. This is the condition in which the lung is usually found where death has occurred at the height of the disease. If the process has lasted longer, larger collections of pus may be present. The lung itself shows the usual changes of pneumonia, and if there has been any considerable accumulation of fluid, there are in addition the evidences of compression.

With pleuro-pneumonia of the left side, the pericardium is occasionally involved. This was seen in two of my cases, the lesions closely resembling those of the pleura. In two cases there was also meningitis, and in one peritonitis, the exudation in all cases having the same characteristics.

An inflammation of the intensity described is very often fatal in the acute stage, if the patient is a child under two years old. Occasionally at this age, and very frequently in older children, we see the later stages of the process. The most frequent course is for more and more pus to be poured out from the inflamed pleura until the chest is filled, the case becoming thus one of empyema. Sometimes the fluid is serous instead of purulent, but this is very rare in infancy. Under other circumstances the exudation is partly absorbed, but the greater part becomes organized so as to form a thick jacket of fibrous tissue which binds the lobe or lung to the chest wall, and interferes seriously with its subsequent full expansion. Chronic interstitial pneumonia may follow.

Symptoms.—There is little which distinguishes a case of pleuro-pneumonia except the severity of all the constitutional symptoms; the tem-

PLATE XII.



ACUTE PLEURO-PNEUMONIA.

The lungs have been separated in front and spread out to show the whole external surface as seen from behind. The left lung, with the exception of a narrow strip along its anterior border, is completely covered with a thick, ragged exudation of fibrin. The left lower lobe was hepatized; the right lower lobe deeply congested.

From a child one year old, who died in the New York Infant Asylum.

perature is often higher, the prostration greater, and the patient in every way impresses one as being more seriously ill than with ordinary pneumonia. Sometimes the thoracic pain is more severe and more constant than is usual in pneumonia. The diagnosis, however, is to be made by the physical signs.

In the early stage the pleuritic friction sounds are unusually prominent; after two or three days the signs of consolidation come out clearly in most cases, but still accompanied by loud friction sounds. After the fibrinous exudation is very abundant, the signs are often obscure and confusing, and there may be at no time well-defined signs of consolidation. There is usually a mingling of the signs of consolidation with those of effusion. There is marked dulness, and sometimes flatness. The vocal fremitus is apt to be diminished, and it may be absent. Bronchial voice and breathing are heard, but they are not distinct as in consolidation; they are, however, feeble and distant, as over fluid. There are usually coarse, moist, crackling pleuritic sounds, but these may be absent. The signs may be found over one entire lung, or they may be limited to the posterior region, and even to a single lobe. They resemble those present over fluid, with one exception—viz., the heart is not displaced. If an exploratory puncture is made, nothing is found; occasionally the exploring needle happens to strike one of the small pockets of pus in the meshes of the fibrin, and a few drops of clear pus are withdrawn. If an incision is made under the supposition that the case is one of empyema, no more pus may be found, the surgeon coming upon the pulmonary adhesions as soon as the chest is opened. There is scarcely any condition in the chest giving signs more puzzling than those just enumerated. They are, however, easily explained by the pathological conditions present.

Prognosis.—The prognosis in pleuro-pneumonia is much worse than in simple pneumonia. In infants the outlook is very bad, the majority of cases dying during the acute stage, usually in the second week. Very young children may be overwhelmed with the extent and the intensity of the inflammation, and die in four or five days. In children over two years old the most frequent result is for the case to go on to empyema, which with proper treatment usually terminates in recovery. Where there is organization of the fibrin with the production of extensive adhesions, the ultimate result is often not so favourable as when empyema develops. Convalescence is usually slow, and the patients are liable to exacerbations of pleurisy; they may suffer for years from the partial crippling of one lung.

Diagnosis.—This is to be made only by the physical signs. A differential diagnosis from fluid in the chest can in some cases be made only by an exploratory puncture.

Treatment.—Cases of pleuro-pneumonia require no special treatment. In general they are to be managed like the ordinary cases of pneumonia of the severe type. In some, the excessive pain may call for more active counter-irritation and a freer use of opium than in other forms of pneumonia, and the greater prostration may require that stimulants be given earlier and in larger quantities.

HYPOSTATIC PNEUMONIA.

This can not often be recognised clinically, but it is very frequently seen upon the post-mortem table. It is present in some degree in almost every case where an infant has died of chronic disease. It is particularly frequent in those who have died of marasmus. It is sometimes described as "strip pneumonia," on account of its position. It invariably occupies a strip along the posterior border of both lungs, and usually of both the uper and lower lobes. This is from one to two inches wide, of a uniform dark-red colour, and is sharply outlined. The pleura is not involved, and the remainder of the lung may be normal, congested, or slightly emphysematous. On section, it is seen that the pneumonic area is quite superficial, rarely involving the lung to a greater depth than half an inch. Under the microscope there is found a distention of the small blood-vessels in the affected area, and the air vesicles are filled with many red blood cells, epithelial cells, and a few leucocytes. Between the areas of consolidation are groups of air vesicles which are normal, congested, or collapsed. It is a lobular rather than a broncho-pneumonia. The lesions in this form of pneumonia are probably the result of venous stasis, owing to the child's recumbent position.

At autopsy the condition may be confounded with atelectasis; this, however, is almost invariably more marked in the interior of the lung, while pneumonia is always more marked upon the surface. The two conditions are sometimes associated. Little significance is to be attached to the finding of hypostatic pneumonia at autopsy, and it alone should never be regarded as a sufficient cause of death, although it is perhaps the only lesion present. During life it may give rise to fine moist râles, which are heard along the spine, usually upon both sides; but there is neither dulness nor bronchial breathing.

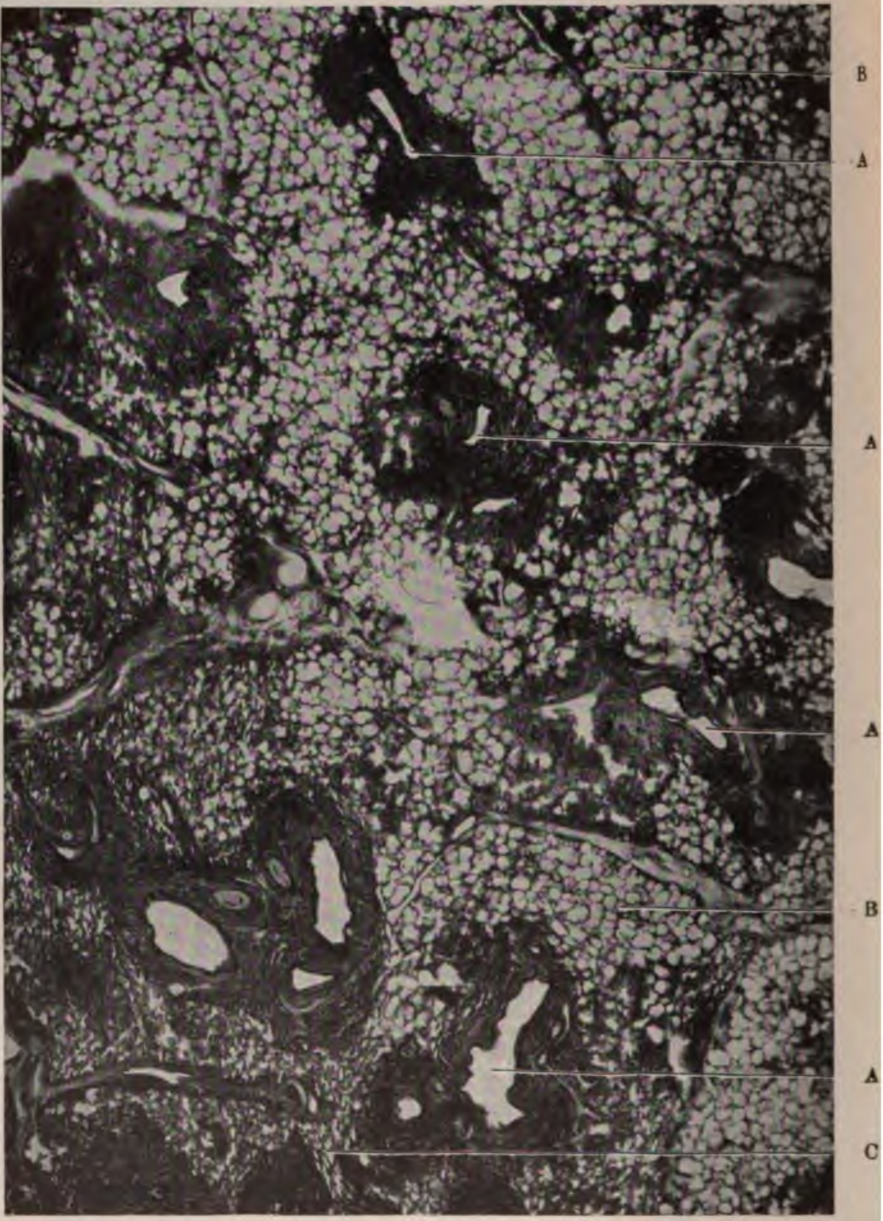
The treatment is that of the primary disease.

CHRONIC BRONCHO-PNEUMONIA—CHRONIC INTERSTITIAL PNEUMONIA—BRONCHIECTASIS.

Chronic broncho-pneumonia is an inflammation of the connective-tissue framework of the lung, involving the stroma, the alveolar septa, the walls of the bronchi, and the pleura. It is usually accompanied by cylindrical dilatation of the bronchi—bronchiectasis.

Etiology.—In children, as in adults, this process is most frequently associated with pulmonary tuberculosis; but in early life it is not an in-

PLATE XIII.



CHRONIC BRONCHO-PNEUMONIA.

In the greater part of the specimen the disease is limited to the vicinity of the small bronchi, A A A, each of which is surrounded by a zone of new connective tissue, the result of the inflammatory process, the intervening lung tissue, B B, being normal. In the lower left-hand portion, the disease is more diffuse; the air vesicles, C, between the areas of new connective tissue are greatly compressed, and in some places entirely obliterated. (After Delafield.)

frequent condition apart from tuberculosis. The non-tuberculous cases, as a rule, are preceded by an attack of acute broncho-pneumonia, sometimes by several such attacks, separated by longer or shorter intervals.

Lesions.—The part of the lung affected may be an entire lobe, but usually it is a portion of one lobe, or there are areas in more than one lobe. There are dense connective-tissue adhesions binding the diseased part to the chest wall, to the diaphragm and to the pericardium, often so firmly that the lung is torn on removal. The affected lung is smaller than in health; it is hard, tough, and fibrous. Surrounding the fibrous portions are emphysematous areas. On section, the process is seen to be somewhat irregularly distributed through the lung, the lesion being usually most marked in the vicinity of the smaller bronchi, and sometimes seen only there, the intervening lung being nearly normal (Plate XIII). In some portions, where the process is most advanced, almost all trace of lung tissue has disappeared, the part resembling a solid fibrous tumour, through which run the bronchial tubes, usually much dilated. In places this dilatation may be sufficient to form cavities of considerable size. The bronchial glands are often enlarged to the size of a hazelnut, and they may be tuberculous.

Upon examination with the microscope, the pleura is found greatly thickened, with bands of new fibrous tissue passing from it into the lung. The walls of the small bronchi are in most places thicker than normal, but elsewhere they have undergone cylindrical dilatation, and are filled with pus. The walls of the alveoli show a marked proliferation of the connective-tissue elements, and the alveoli are filled with organized inflammatory products, so that they are nearly or quite obliterated. The stroma is much increased in amount throughout the affected lung.

Symptoms.—In most of the cases there is a history of an attack of acute broncho-pneumonia, from which the child made a slow convalescence, remaining pale, anæmic, and sometimes wasted for several months. Improvement then took place in the general symptoms, the appetite and strength returned, and in many cases the lost weight was nearly or quite regained. However, neither the pulmonary symptoms nor the physical signs entirely disappeared. There remained a dry, hard cough, which at times was severe. Pains in the chest were occasionally complained of, and perhaps shortness of breath on exertion was noticed.

Examination shows a persistence of the dulness on percussion, with a rude or broncho-vesicular respiratory murmur of very feeble intensity. Little change may take place in these signs for months; then an acute attack of bronchitis or broncho-pneumonia may occur. If the latter, the same lung is affected, and a fresh consolidation is added to the previous disease. This attack may not be very severe, but it drags on for several weeks, with slight fever and little or no change in the physical signs. Partial resolution may then take place, but the lung is left much more seriously crippled than before. Often there is a history of several such attacks, each one leaving the lung a little worse than it found it.

The characteristic physical signs of chronic broncho-pneumonia are not usually present until the process has continued for many months. They may be found over part of a lobe, or over an entire lobe, or even the greater part of one lung. On inspection, there is seen in a well-marked case, retraction of the chest, which is especially noticeable when the disease is situated at the apex of the lung. The vocal fremitus is usually increased, but it may not be abnormal. There is marked dulness, often flatness, over the affected area, with exaggerated resonance over the rest of the lung. The area of flatness shades off gradually. The most striking thing on auscultation is the very feeble respiratory murmur; in many cases the lung is almost silent. Râles and friction sounds are usually absent except during an acute exacerbation of the symptoms, when they may be heard as in any attack of broncho-pneumonia. In recent cases there is no displacement of the heart; in those of long standing it may be drawn far to the affected side by contraction of the adhesions.

When the lesions are once present complete recovery is impossible, and there is always a tendency for them to increase rapidly or slowly, according to the child's vigour of constitution, its surroundings, and the frequency with which exacerbations occur. If the disease is extensive the patient often succumbs to some intercurrent disease or to an acute attack of pneumonia; if limited in area, the process may be arrested and the patient recover, always, however, to be more or less embarrassed because of the crippling of a part of one lung. Not a small number of these children ultimately die of tuberculosis, and in such cases it is always a difficult matter to decide whether tuberculosis was present from the beginning, or whether there was subsequent infection.

The cases in which bronchiectasis is the most important condition are not common. The only characteristic additional symptom is a copious muco-purulent expectoration which is usually very fetid. It may amount to several ounces a day, and is expelled after paroxysms of coughing which usually occur in the morning. This may continue for months or even years, and yet these patients are generally without fever, seldom lose weight, and may give the appearance of being in very good health. It is rare that the physical signs of a cavity are present.

Prognosis.—This depends on the extent of the disease, the patient's age and constitution, and on our ability to prevent by treatment, climatic and otherwise, the occurrence of acute exacerbations. Under the most favourable conditions, a few patients may recover completely so far as symptoms are concerned; but the majority remain at best delicate during childhood, or even throughout life.

Diagnosis.—The most important thing is to distinguish between the simple and the tuberculous cases, and this, it must be confessed, is in the majority impossible. I have repeatedly seen a process proved at autopsy to be simple, which all who had observed the case had unhesitatingly pronounced to be tuberculous, and quite as often the opposite has been true. If the family history is good, if the patient lives in the country, if his symptoms begin with a well-defined acute attack of pneumonia, if the seat of disease is the base posteriorly, and if the examination of the sputum is negative, the process is probably simple. If the family history is doubtful or is positively tuberculous, if the patient lives in the city, and especially if he is an inmate of an institution or if his home is among the tenements, if the initial symptoms are indefinite, if the disease is situated anteriorly, the process is probably tuberculous. The discovery of tubercle bacilli in the sputum is, of course, conclusive.

Treatment.—Nothing has any essential influence upon the disease except change of climate. This should be the same as for tuberculous cases. The treatment of the patient has for its object the maintenance of the general nutrition at its highest point, by careful feeding, judicious exercise, and by most of the measures enumerated in the chapter on Malnutrition. Cod-liver oil should be given throughout every winter season. The cough may be treated as in cases of chronic bronchitis.

Cases of bronchiectasis may obtain considerable relief from inhalations of creosote. They should not be operated upon.

ABSCESS OF THE LUNG.

Multiple small abscesses are not uncommon as a termination of acute broncho-pneumonia, in which connection they have already been considered. Larger non-tuberculous abscesses of the lung are rare, very obscure in their symptoms, and apt to be mistaken for localized empyema, sometimes for interstitial pneumonia with bronchiectasis. Three such cases have come under my observation.* One was discovered at autopsy, the other two were recognized during life and successfully treated by operation. Other examples in young children have been reported by Huber and by Hedges. The cause of these single abscesses is usually a previous attack of acute primary pneumonia, less frequently an inflammation excited by a foreign body in the lung.

An abscess due to a foreign body is usually accompanied by wasting, and a widely fluctuating temperature of a hectic type—symptoms suggestive of a rapidly advancing tuberculous process. If the abscess follows an ordinary pneumonia the course is generally less intense. The constitutional symptoms differ little from those of empyema. There is an irregular type of fever, sometimes quite high, but more often only from 99° to 101° or 102° F., a moderate cough, not much wasting and generally not very marked prostration. A leucocytosis of 30,000 to 50,000 is usually present. The physical signs are somewhat confusing and are a combination of those present in effusion and consolidation. There is an area of flatness shading off into dulness. The vocal fremitus may be increased or it may be diminished. The respiratory murmur is very feeble or absent over the abscess, often it is broncho-vesicular in character. Friction sounds and râles are usually present. The heart is slightly or not at all displaced. If an exploratory needle is introduced, pus may not be found even by repeated punctures; or it may be obtained at one time and not at another, although introduced in the same intercostal space, the difference in result being due to the direction in which the needle is passed into the lung. When pus is found, the diagnosis of a localized empyema is generally regarded as established, and it is not until the chest is opened that the mistake is discovered. The operator then comes upon the lung, which may or may not be adherent. If the abscess follows an acute pneumonia the pus may show a pure culture of the pneumococcus. If it is due to a foreign body, there is invariably mixed infection, and the pus is apt to be fetid.

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* Archives of Pediatrics, January, 1904.

When not treated surgically abscess of the lung may rupture into the pleural cavity, producing a secondary empyema, or spontaneous evacuation may take place through a bronchus and recovery follow. When the cause is a foreign body rapid recovery often follows its expulsion by coughing. If the diagnosis is made and proper surgical treatment is instituted, recovery occurs in probably the majority of cases.

The general plan of treatment should be the same as in empyema. In a small proportion of cases aspiration may suffice for a cure. However, incision is usually necessary. If the pleura is not adherent, adhesions should be excited by packing the thoracic wound with gauze, and after a few days a second operation may be done. The lung should be opened with a blunt instrument, following the line of the exploring needle, and a drainage-tube inserted as in empyema, the subsequent treatment being the same as for that disease.

GANGRENE OF THE LUNG.

Pulmonary gangrene is rare in children, although probably more common than in adults. It is most frequently associated with pneumonia. It is usually circumscribed, and seldom diagnosticated during life.

Etiology.—All my cases have been in children under three years old, the youngest an infant of four months. Gangrene occurs for the most part in children who are ill-conditioned, feeble, or cachectic, and often follows one of the infectious diseases, particularly measles. Of nine cases which have come under my personal observation, six complicated acute broncho-pneumonia and one, lobar pneumonia. It has been present in three per cent of my autopsies upon cases of pneumonia. The immediate cause of the necrotic process is interference with the circulation in a part of the lung, which is usually due to thrombosis or embolism of some of the branches of the pulmonary artery. To this there is added the entrance of putrefactive bacteria. In some cases pulmonary gangrene may begin as a septic thrombosis, this infection originating in some process in a distant part of the body.

Lesions.—The lower lobes are more frequently affected than the upper, and the surface of the lung rather than the central portions.

Two forms of gangrene may be seen: the diffuse form, which affects a whole lobe, or even a whole lung; and the circumscribed form, which occurs in a number of small scattered areas. The latter is the variety usually seen in children. In the diffuse form the lung is of a dirty green or brown colour, moist, and emits a gangrenous odour. In the circumscribed form, when occurring in pneumonia, the parts affected are of a gray or green colour, usually wedge-shaped, with the base at the surface of the lung. In the early stage they are not softened, and have no gangrenous odour; later, both these conditions may be present, and masses of necrotic lung tissue may be found in a cavity with ragged walls, partly filled with fetid pus. Careful dissection will reveal, in many cases, the presence of thrombi in the vessels leading to the gangrenous parts.

Symptoms.—There are but two distinctive symptoms of pulmonary gangrene: the gangrenous odour of the breath, and the expectoration of masses of necrotic lung tissue. In the cases associated with acute pneumonia, which include the majority of those seen, death nearly always takes place before there is any separation of the sloughs, and even before very active decomposition in the necrotic areas has occurred. Both the peculiar symptoms are therefore wanting, and the diagnosis is made only at the autopsy. This has been true of nearly all the cases which have come under my own observation. But these patients, with one exception, were infants. In older children, particularly in cases secondary to the entrance of a foreign body, the characteristic symptoms are more frequently seen, and there may be a third symptom—hæmorrhage. This is present in about one fourth of the cases (Rilliet and Barthez), and may be fatal. The general symptoms associated with gangrene are those of profound asthenia, resembling the typhoid condition.

From what has been said, it will be evident that the diagnosis is very difficult. If the characteristic odour of the breath is present, conditions in the mouth from which it might arise must be excluded. The physical signs differ in no respect from those of ordinary cases of pneumonia. The termination is almost always in death. This is due not only to the condition itself, but to the circumstances in which it is seen.

Treatment.—The general treatment should be supporting and stimulating, as in all severe cases of pneumonia. For the local process but little can be done, except the inhalation of antiseptics, of which creosote and turpentine are undoubtedly the best.

ACQUIRED ATELECTASIS—PULMONARY COLLAPSE.

These terms are applied to a state of the lung resembling the fœtal condition, but occurring in a lung which has once been expanded. It may be due to compression or to obstruction.

Collapse from Compression.—The principal cause of this form is pleuritic effusion. It may also be produced by pneumothorax, enlargement of the heart, pericardial effusion, deformities of the chest from rickets or Pott's disease, and tumours of the mediastinum or the thoracic wall. In these conditions, on account of the external pressure, the air vesicles are not filled, although the bronchi are pervious. After collapse has existed for a considerable time, changes may take place in the lung which render expansion difficult or impossible. Unless, however, there are pleuritic adhesions, expansion often takes place readily after many weeks and even months. The symptoms and signs are those of the original disease.

Treatment is available chiefly in that form which follows pleuritic effusion, and will be considered in the chapter on Empyema.

Collapse from Obstruction.—This is due to two factors: blocking of either the large or small bronchial tubes, and feeble inspiratory force. The importance of collapse from obstruction in the acute diseases of the lung in infancy has, I think, been exaggerated. Whenever a large or small bronchus is completely obstructed by a foreign body, the portion of the lung to which the bronchus is distributed gradually becomes collapsed. If it is one of the primary bronchi which is occluded, a whole lung may be collapsed; if one of the lobar divisions, an entire lobe; if one of the smaller divisions, only a small area. The collapse does not take place immediately, but the contents of the air vesicles are gradually absorbed by the blood. The collapsed portion is slightly depressed below the surface of the lung. It is of a dark-red colour, very vascular, and to the naked eye resembles a pneumonic area, which it may subsequently become.

Many writers explain the development of broncho-pneumonia from bronchitis of the smaller tubes, through the intervention of pulmonary collapse, assuming that the obstruction of the small bronchi from swelling of their walls and the accumulation of secretion, produces the same result as the plugging of a bronchus by a foreign body. In my own autopsies I have found little support for this theory. In acute bronchitis of the smaller tubes the lumen is narrowed, but seldom enough to prevent the entrance of air. The result is usually emphysema, not atelectasis. Such, at least, has been the condition I have most frequently found in autopsies in the earliest stage of broncho-pneumonia following bronchitis of the fine tubes. There are very often groups of collapsed air vesicles surrounding pneumonic areas, but these are neither an essential nor a very important part of the lesion. Collapse of a large part of the lung, or even of a lobe, I have never seen, either in pertussis or in acute bronchitis.

There is seen in delicate or rachitic infants a form of collapse which comes on very gradually. It is accompanied by bronchitis affecting the tubes in the dependent part of the lung. It may resemble the congenital form of atelectasis. Under the microscope there is almost invariably found accompanying the collapse, lobular pneumonia and bronchitis of the tubes in the affected regions.

The symptoms of acquired atelectasis are much the same as in the persistent congenital form. The respiration is rapid, and there may be inspiratory dyspnœa with deep recession of the chest walls, especially if there is rickets. There is also cyanosis of variable intensity. The temperature is not elevated, but frequently is subnormal. The physical signs are very uncertain. There is usually feeble respiratory murmur over the affected areas, occasionally accompanied by moist râles. The essential point of difference between these cases and those of congenital atelectasis is that in the former the patients are often strong at birth, crying and breathing well, giving no signs of anything wrong in the lungs until the general nutrition has suffered from some other cause.

The following is a fairly typical case: A female infant thirteen months old had been under observation for several months before death. During this period she suffered a great part of the time from mild bronchitis. The chest was extremely rachitic. The respiration was always accelerated, and on inspiration the lateral recession of the chest was at times extreme. There was occasionally seen slight cyanosis, and during the last few weeks it was constant. Death occurred quite suddenly. At autopsy there was found very marked vesicular emphysema of both lungs in front. Nearly the whole of both lower lobes were in a condition of collapse, and of a uniform grayish-purple colour. The posterior portion of the upper lobes was similarly affected, but to a less degree. With moderate force all of the collapsed areas could be completely inflated. Bronchitis was present, but the pleura was normal.

The treatment of these cases is the same as that outlined in the chapter upon Congenital Atelectasis (page 74).

EMPHYSEMA.

Pulmonary emphysema consists primarily in overdistention of the air vesicles. It may result in their rupture and the escape of air into the interlobular connective tissue of the lung. In infancy and childhood emphysema is usually associated with acute processes.

Etiology.—Cases of emphysema are divided into two groups which are due to quite different causes. In one group it is compensatory, and consists in overdistention of the air vesicles in certain parts of the lungs because the full expansion of other parts is prevented either because they are consolidated, as in pneumonia or tuberculosis, bound down by adhesions from old pleurisy, or subjected to external pressure, as from chest deformities due to Pott's disease or rickets. In these conditions it is probable that the emphysema is produced during inspiration. It may also be produced by the artificial inflation of the lungs of the newly born.

In the second group of cases emphysema is produced by obstructive expiratory dyspnœa or cough. It is seen in all forms of laryngeal stenosis, in acute bronchitis and broncho-pneumonia, in asthma, pertussis, and occasionally it is produced by any condition which requires deep inspiration and holding the breath. A case has been reported to me which occurred in a little boy, who, while playing that he was a steam engine, would hold his breath for a long time and then issue short, forcible expiratory puffs. In bronchitis the obstruction may be caused by swelling of the mucous membrane or by an accumulation of secretion. In this group of cases air enters the lung, but as it can not readily escape, the air vesicles are distended, sometimes to such a degree that their resiliency is almost entirely lost.

Lesions.—The most common form in early life is acute vesicular emphysema, which occurs when the force distending the air cells is only moderate. In this form there is dilatation of the vesicles with very slight structural changes, there being usually rupture of a few alveolar septa only (Fig. 90). Although the dilatation may be quite marked, the emphysema is not permanent. The parts most affected are the upper lobes, particularly the anterior borders. In appearance the emphysematous lung is pale, sometimes almost white. The areas are prominent, and do not collapse upon opening the chest. With a lens, or even with the naked eye, the individual air vesicles can often be distinguished as minute pearly bodies, at times resembling miliary tubercles. When the disease is secondary to acute bronchitis or laryngeal stenosis it may affect nearly the whole of both lungs.

With a greater distending force rupture of many of the air vesicles results, and this may give rise to interstitial or interlobular emphysema. At times blebs are formed, varying in size from a pin's head to a cherry. These are usually seen at the anterior border or at the root of the lung on its inner surface. Again, the air finds its way between the lobules, dissecting them apart in all directions throughout the lung. Sometimes a large part of the surface of both lungs is seamed with irregular deep crevasses containing air, the largest being an inch or more in length and nearly one fourth of an inch wide. The most severe cases occur in pertussis. On two or three occasions I have seen this form of emphysema, once to an extreme degree, where children had died from diseases unconnected with the respiratory tract, and where no history could be obtained which threw any light upon the etiology of the emphysema. Rupture of the blebs which form at the root of the lung may lead to emphysema of the mediastinum, or even of the subcutaneous connective tissue of the body. This is occasionally seen in whooping-cough and in laryngeal stenosis. The primary or substantive form of emphysema seen in adult life rarely if ever occurs in childhood.

Symptoms.—Emphysema occurring in acute pulmonary diseases gives rise to no peculiar symptoms and to no physical signs except exaggerated resonance upon percussion. If the patients recover from the original disease, the emphysema undoubtedly disappears completely in the course of a few weeks or months. Acute interlobular emphysema can not be diagnosticated during life. The lesion is of such a nature that complete recovery is impossible, although improvement often takes place.

The treatment of emphysema is that of the disease with which it is associated.

CHAPTER VI.

PLEURISY.

ALL the common forms of inflammation of the pleura are seen in childhood. In the great majority of cases they are secondary to disease of the lung itself. Serous effusions are much less frequent than in adults, and under three years they are extremely rare. Purulent effusion (empyema) is, however, much more often seen than in adult life, and it is the most important variety of pleurisy with which the physician has to deal.

Whether inflammation of the pleura ever occurs as a strictly primary disease is still a mooted point. Cases are occasionally observed clinically in which both the serous and purulent forms of the disease appear to be primary, but these are extremely rare. Acute pleurisy may, however, follow inflammation of the lung so rapidly that it is not easy to determine that the lung was first affected. In infants, extension from the lung is almost the sole cause. It occurs both with lobar and broncho-pneumonia, existing to some degree in nearly every case in which there is consolidation of the lung. Next in frequency to simple pneumonia as a cause of pleurisy are the tuberculous processes of the lung. Tuberculous pleurisy without tuberculosis of the lungs or the bronchial glands is of doubtful occurrence. Acute pleurisy is not an infrequent complication of the infectious diseases, particularly scarlet and typhoid fevers, measles, and influenza. In most of these cases also it is secondary to disease of the lung. Pleurisy in older children occasionally follows cold and exposure, although it is doubtful whether in any case this is the only cause. In them also it may occur as a complication of rheumatism.

The most important cause of acute pleurisy being extension from pneumonia, it follows that it is most frequent in the cold season, that it occurs more often in males than in females, and between the ages of one and five years. It may, however, be seen at all ages, and may even occur in intra-uterine life. The youngest case in which I have found extensive pleuritic adhesions as an evidence of previous inflammation was in an infant of three months, who died at the Randall's Island Hospital. In this case firm connective tissue adhesions were found over the whole of both lungs.

DRY PLEURISY.

In infants and young children this usually accompanies pneumonia or tuberculous processes in the lung. In older children it may be primary.

Lesions.—On account of the frequency with which this occurs in pneumonia we have an opportunity of observing it in all stages. In the mildest varieties it affects only the pulmonary pleura, and occurs over the pneumonic areas. The pleura is injected, has lost its lustre, and appears dull or roughened. This is due to an exudation of fibrin upon its surface. If the process continues, more fibrin is poured out, and there are in addition swelling and a proliferation of the connective-tissue cells, and an exudation of leucocytes from the blood-vessels. The pleura is then coated with a layer of fibrin of variable thickness, in which are entangled pus cells and new connective-tissue cells. The layer of fibrin varies from the thickness of tissue paper to that of an ordinary book cover. In recent cases it may easily be stripped off, while in older ones it becomes organized and is firmly adherent. The colour of the exudate varies with the number of pus cells. It is gray, grayish-yellow, or yellowish-green, according as these cells are few or numerous. As a rule, dry pleurisy is localized, but the two opposing surfaces are affected. Part of the exudate is usually absorbed, but it is doubtful if complete recovery occurs, there being left behind some adhesions between the visceral and parietal layers.

In some cases of dry pleurisy there is an excessive exudation of pus cells. These cases are most common in young children, and usually occur with pneumonia, constituting what is known as "pleuro-pneumonia." The process is essentially the same as in the cases just mentioned, yet the gross appearance differs very much from that of ordinary dry pleurisy. The lesions have already been described under the head of Pleuro-Pneumonia.

In the dry form of tuberculous pleurisy there may be only an exudation of fibrin, or the pleura may be covered with gray tubercles and yellow tuberculous nodules. These are not only seen upon the pleura, but develop in the exudation. In this form, which is usually chronic, great thickening of the pleura may take place. Both the serous and purulent effusions occurring in conjunction with tuberculosis are likely to be sacculated because of the previous existence of adhesions.

After nearly every case of dry pleurisy there probably remains some slight thickening of the pleura. In certain cases there follows a chronic inflammation of the pleura with the production of new connective tissue, which results in thickening and adhesions, which may be so extensive as to entirely obliterate the pleural cavity. Either one or both sides may be affected. This form is extremely rare in childhood.

Symptoms.—As an independent clinical disease, acute dry pleurisy has no existence in infancy or early childhood. The cases which are occasionally so diagnosticated have in my experience invariably proven to be broncho-pneumonia. In children from ten to fourteen years old, dry pleurisy may occur under the same conditions as in adults.

The symptoms are sharp, localized pain, increased by full inspiration, sometimes tenderness upon pressure, and a short, teasing cough. The pain is not always felt upon the affected side, and it may be referred to the abdomen. Upon physical examination, dry pleurisy is recognised by the presence of a pleuritic friction sound. This is usually of a moist, crackling character, generally localized, and heard both on inspiration and expiration. It is quite superficial, and not changed by coughing. This form of pleurisy, as a rule, runs a course of a few days or a week, without constitutional symptoms. When dry pleurisy occurs as a complication of pneumonia it is recognised by the signs just mentioned; but it usually causes no new symptoms except pain.

Treatment.—The treatment consists in counter-irritation by mustard, iodine, or blisters, according to the severity of the inflammation, and in the use of opium. Severe pain can sometimes be relieved by firmly encircling the chest with a broad band of adhesive plaster.

PLEURISY WITH SEROUS EFFUSION.

This form of pleurisy is infrequent in children, and under three years it is very rare. It may occur as a complication of pneumonia, nephritis, acute rheumatism, scarlet fever, or any of the other acute infectious diseases. It may be tuberculous. In rare cases it appears to be primary. Bacteria are occasionally present in the exudation, even in cases which do not become purulent, but their number is usually small. The pneumococcus, the streptococcus, and the tubercle bacillus are the forms most often seen.

Lesions.—The early changes are much the same as in dry pleurisy, but in addition serum is poured out from the blood-vessels, in some cases almost from the beginning of the inflammation. This may be small in amount, or it may fill the pleural cavity. The lesions are similar to those seen in adults, except that in children there is apt to be more fibrin. The process usually terminates in absorption of the serum, but, as in dry pleurisy, more or less extensive adhesions are left behind from the fibrinous exudation.

Symptoms.—The small serous effusions of one or two ounces, occurring with the dry pleurisy that complicates pneumonia, rarely cause either symptoms or physical signs by which they can be recognised. In the present connection only those cases will be discussed in which the amount of effusion is considerable. This form of pleurisy sometimes follows a well-defined attack of pneumonia. Other cases come on with acute febrile symptoms somewhat resembling those of pneumonia, but with all the symptoms less severe, except the pain. After an illness of only two or three days the chest may be found full of fluid. In a third class the disease comes on insidiously, with little or no fever, and often with no distinct pulmonary symptoms except shortness of breath. There are general weakness, sometimes loss of flesh, anæmia, and moderate prostration; but usually the patients are not sick enough to go to bed. The symptoms of pleurisy with effusion vary greatly. When it occurs as a complication of some acute infectious disease, it is often latent, and the diagnosis is to be made only by the physical examination of the chest.

The usual course of the disease is for the fluid to disappear gradually by absorption, the case going on to spontaneous recovery. Serious symptoms resulting from pressure upon the heart and lungs are not common, but may occur when the fluid accumulates rapidly; hence they are most likely to be seen early in the attack. There may be great dyspnœa, sometimes orthopnœa, cyanosis, weak pulse, and even attacks of syncope. Death may occur with these symptoms. In certain cases there is seen no tendency to spontaneous absorption, and the exudation may remain stationary for months. There may then be fever, usually slight but sometimes quite regular, with a decline in the general health, pallor and anæmia, which may strongly suggest the existence of pus, although this is not present. Others are regarded as cases of tuberculosis.

Physical Signs.—The signs in the chest are essentially the same whether the fluid is serous or purulent. On inspection, there is diminished movement of the affected side, sometimes bulging of the intercostal spaces, and if the effusion is large, an increase in the measurement of the affected side of the chest. The apex beat of the heart will usually be considerably displaced if the effusion is upon the left side. It may be found at the epigastrium, at the right border of the sternum, or even in the right mammary line. In disease of the right side the displacement is less, and occurs only with a large effusion. It may then be found in or near the left axillary line. On palpation, the vocal fremitus is usually diminished or absent, but it may be but little changed. Percussion gives marked dulness or flatness. In a large effusion this is over the entire lung. There is also a sensation of increased resistance appreciable by the percussing finger. With a smaller effusion there is usually flatness over the lower part of the chest and dulness or tympanitic resonance above; sometimes dulness is found behind and tympanitic resonance at the apex in front. The line of flatness may change with the position of the patient. The signs on auscultation are variable, and probably lead to more frequent mistakes in diagnosis than in any other pulmonary affection. Bronchial breathing and bronchial voice over the fluid are the rule in children; they are generally more distinct the greater the effusion. Absence of both voice and breathing is sometimes met with, but it is exceptional. The bronchial breathing over fluid usually differs from that over consolidation, in that it is feebler and distant; in some cases, however, it is indistinguishable from that heard over consolidation. Friction sounds may be heard above the level of the fluid, or when the fluid is subsiding, and there may be bronchial râles.

Diagnosis.—The most reliable signs for diagnosis are displacement of the heart, flatness on percussion, absence of râles and friction sounds, and (usually distant) bronchial breathing. In an infant, flatness should always lead one to suspect fluid. If there is flatness over one entire lung, the existence of fluid is almost certain. Between serous and purulent effusions a positive diagnosis is possible only by the use of the exploring needle. This should be employed in every case, as for treatment it is important to know at once whether or not we have a purulent effusion to deal with. The amount of fluid in serous pleurisy is generally less than in the purulent variety.

Pleurisy is further to be differentiated from pneumonia, and from tuberculosis. From pneumonia, the acute cases are distinguished by the lower temperature, the less severe prostration, and the fact that all the general symptoms are milder, but especially by the physical signs. The differential diagnosis by the physical signs between effusion and the various forms of consolidation is considered under the head of Empyema.

Prognosis.—These cases, as a rule, terminate in recovery, death being very infrequent. In cases coming on without definite cause there should always exist a suspicion of tuberculosis, and hence every patient should be closely watched for the development of the other signs of that disease.

Treatment.—In the great majority of cases, only symptomatic treatment is required during the acute period. The patient should be kept in bed, and pain relieved by opium, counter-irritation, or hot poultices. After the fever has ceased the patient may be allowed to sit up, but all exertion should be carefully avoided if the effusion is large. Sudden death has often occurred when this rule has been violated. The patient should in suitable weather be kept in the open air as much as possible. In the course of a few weeks the effusion usually subsides under simple tonic treatment. Absorption may sometimes be hastened by counter-irritation and diuretics; but convalescence is apt to be slow, and it may be several months before the health is entirely restored.

The removal of the fluid by operation is indicated in the acute stage when it is accumulating so rapidly as to endanger life from the pressure upon the heart and lungs; also when there is no tendency to absorption after from two to three weeks of constitutional treatment. In such cases nothing is to be gained by waiting, and harm may be done to the lung by the delay. The usual method is by aspiration. In the acute stage enough should be removed to relieve the patient's symptoms, aspiration being repeated if necessary in twelve or twenty-four hours. In the sub-acute stage the removal of a portion of the fluid may be all that is required, spontaneous absorption of the remainder often taking place then quite promptly. A few cases of serous pleurisy have been incised and drained as cases of empyema. Scharlau (New York) operated on such a case in an infant two years old. The effusion came on acutely and was excessive, the chest having refilled very quickly after aspiration. The chest was incised and drained and the patient recovered in five days. In chronic cases, in which there are slight fever and a gradual failure of general health, the operation of incision is by some preferred to aspiration.

EMPYEMA.

Fully nine tenths of the cases of empyema in children under five years either occur with or follow pneumonia, being usually the sequel of the form described as pleuro-pneumonia. In some of these cases, however, the pleurisy masks the pneumonia, so that the former appears to be the primary disease. Tuberculosis is a rare cause in early childhood, but becomes more frequent after the seventh year. Empyema may complicate scarlet fever, measles, or any of the other acute infectious diseases. It is met with in pyæmia from all causes. It may occur in the newly born as the result of infection through the umbilical wound or the skin. It is seen with suppurative inflammations of the joints and in osteo-myelitis. It may complicate suppurative processes in the abdomen, such as appendicitis or purulent peritonitis. Among the local causes may be mentioned traumatism, necrosis of a rib, and the rupture into the pleural cavity of abscesses originating in the mediastinum, in the thoracic wall, or below the diaphragm.

Bacteriology.—Much light upon the etiology of empyema has been thrown by the bacteriological investigations of the past few years, especially by the work of Fraenkel, Weichselbaum, Levy, and Netter in Europe, and Prudden and Koplik in this country. Bacteriologically, we may divide the cases into several groups:

1. Those containing the pneumococcus (micrococcus lanceolatus), usually in pure culture. This is the largest group, and includes nearly all the cases secondary to pneumonia. The pleura is usually involved by direct infection from the lung.

2. Those containing other pyogenic germs, particularly the strepto-

coccus and the staphylococcus. Of these the streptococcus is the most important. It may be found alone, but is usually associated with the pneumococcus. This combination is likely to be found in cases secondary to the pneumonia which occurs with the infectious diseases. The streptococcus and staphylococcus occur in the pleurisy of pyæmia, and usually also when the disease is due to the rupture of abscesses into the pleural cavity.

3. The cases due to tuberculosis. In this group the presence of the tubercle bacillus is very often difficult to demonstrate, and it may be absent. From this fact the statement is made by Levy that, if no bacteria can be found in a purulent exudate, tuberculosis should always be suspected. It is not, however, safe to conclude that under these circumstances tuberculosis is always present.

Of nineteen successive cases of empyema occurring in my own practice, the pneumococcus was found alone in fourteen; the streptococcus alone in three; the pneumococcus and streptococcus in one; and the staphylococcus alone in one.

Lesions.—Empyema is an inflammation with the production of serum, fibrin, and pus. In most of the cases—and the younger the child the more frequent its occurrence—it succeeds pleuro-pneumonia. There is first an exudation of fibrin with an excess of pus cells. As the process continues, more and more pus is poured out, with serum. At first the fluid collects in small pockets formed by the slight adhesions. As it accumulates these are broken down, and the pleural cavity may be filled with pus. If the original inflammation involved but a portion of the pleura the empyema may be sacculated. This is often seen even in infants. Sacculated empyema is usually posterior, but may be in any part of the chest. In very rare cases there may be several sacs containing pus, separated by septa. This I have never seen in empyema following pneumonia. The cases just described are those in which, in infants and young children, the pneumococcus is regularly found. The amount of fibrin is large, covers both surfaces of the pleura, and many large masses float in the fluid. The pus is usually thick, creamy, and odourless. In another group of cases the evidences of inflammation of the pleura are much less marked, and in some they may be slight. There is but little fibrin in the exudate, and adhesions are rare. In this form the streptococcus or the staphylococcus are the organisms usually found. In these cases the inflammation may be purulent from the outset, and the pus is thinner than in the preceding variety. It is rare that empyema in a young child results from a serous effusion which has been gradually converted into a purulent one. I can recall but a single instance.

Even when the fluid is moderate in quantity it is not all at the bottom of the chest, but is generally distributed over a considerable part of its surface, and its depth at the middle and upper part of the chest may be only half an inch, or even less. When the accumulation is larger, the lung does not float on the surface of the fluid, but the fluid surrounds the lung, which is compressed on all sides (Fig. 110). The heart is displaced; the diaphragm and the abdominal viscera are somewhat depressed, and there may be bulging of the chest on the affected side. The amount of fluid in ordinary cases is from half a pint to two pints, although in neglected cases it may accumulate until it amounts to four or five pints. The effect upon the lung will depend upon the amount of fluid and the duration of the compression. When the quantity is small, or when the pressure is removed early, the lung in most cases readily expands, air being forced into it from the opposite lung, especially during the act of coughing. If the pressure is great and has been long continued, the adhesions over the lung may become so dense and firm that expansion is difficult, and can at best be only partial. In such cases recession of the chest wall occurs. In very old cases, expansion is still further interfered with by the changes taking place in the lung itself, usually a low grade of interstitial pneumonia.

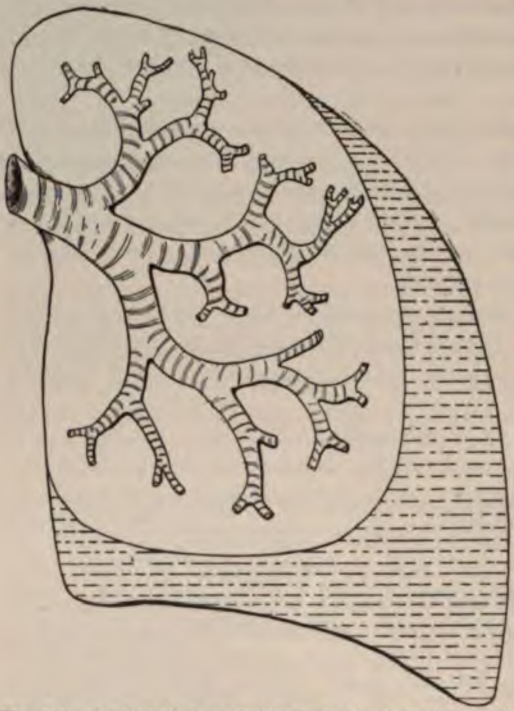

FIG. 110.—Section of a lung to illustrate the distribution of the fluid in the chest in a moderately large effusion (diagrammatic).

In cases of empyema receiving proper surgical treatment reasonably early, full expansion of the lung occurs, and, with the exception of adhesions, recovery may be complete. Although wide in extent, the adhesions are not usually strong enough to interfere seriously with the function of the lung. In cases receiving no treatment, absorption of the pus is possible, but is not to be expected. It generally seeks an external outlet; the lung may be perforated and the pus evacuated through the bronchi, or external rupture may occur, generally in the neighbourhood of the nipple. In still other cases the pus may burrow along the spine, or through the diaphragm may reach the peritonæum.

Empyema is more often of the left than of the right side, the proportion being about three to two. It is double in about three per cent of all cases, but much oftener in infants. The most serious complication in young children is pericarditis, usually with empyema of the left side; in older children the most frequent complication is pulmonary tuberculosis.

Symptoms.—When it occurs as a sequel of pneumonia, the symptoms of empyema may follow those of the original disease without any intermission; or after the temperature has been normal or nearly so for several days it may rise again, sometimes quite suddenly, but more often gradually. With this accession of fever there are other symptoms pointing to an increase in the thoracic disease. (See Figs. 111 and 112.) After scarlet fever or other infectious diseases, the onset of empyema is often signalized by cough, rapid breathing, and the other usual symptoms of pulmonary disease. In the cases where empyema appears to be primary, the onset is sudden, with high temperature and general and local symptoms resembling those of pneumonia. After such a beginning, the

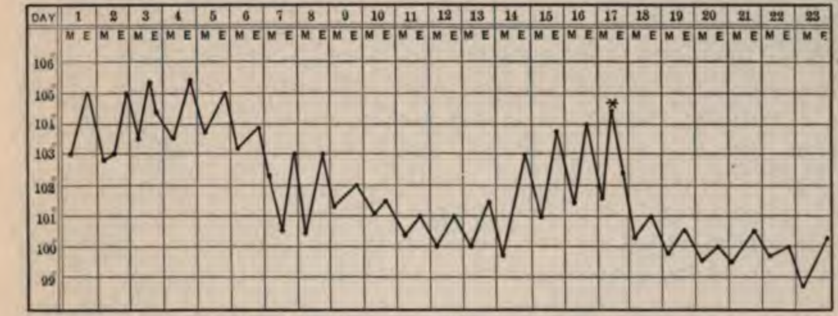

FIG. 111.—Empyema following pneumonia.

Private patient, girl, eight years old; severe pneumonia terminating by lysis; development of empyema indicated by secondary temperature; operation on seventeenth day; recovery.

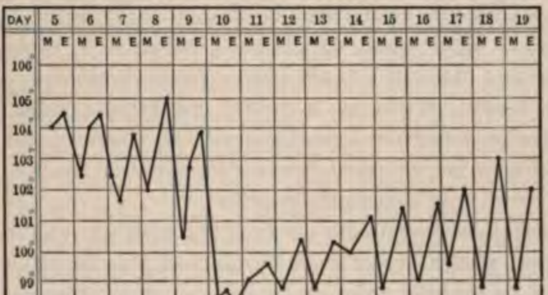

FIG. 112.—Empyema following pneumonia.

Hospital patient, two years old; single-lobe pneumonia with crisis on ninth day; no resolution, but instead gradual development of signs of empyema closely following the temperature curve.

chest may be found full of pus by the third or fourth day. In older children empyema may come on with gradual, and even insidious symptoms, there being only slight fever, dyspnœa, and cachexia. Marked leucocytosis, 30,000 to 50,000, is almost invariably present.

Whatever may have been the mode of onset, when the pus has been in the chest for some time the symptoms are fairly uniform. There is cachexia, pallor, anæmia, and prostration which is generally sufficient to keep the child in bed. The respirations are always accelerated, being usually from forty to seventy a minute. Cough is present; there is dyspnœa, sometimes marked, but more often it is scarcely noticeable. Fever is exceedingly variable; it is not usually above 102° or 103° F.; in many cases it is not over 100° F., and it may be absent altogether. A typical hectic temperature with sweating, is in my experience very rare. The pulse is rapid but of fair strength. There is loss of flesh, sometimes even emaciation and anorexia; occasionally there is diarrhœa. In chronic cases the general symptoms closely resemble those of tuberculosis. There may be clubbing of the fingers, albuminuria, swelling of the feet, and often marked lateral curvature of the spine.

Diagnosis.—The physical signs do not differ essentially from those present in serous effusions. If the patient is under three years of age, the fluid is almost certain to be purulent; and from the third to the seventh year, pus is much more often found than serum. Marked leucocytosis always makes pus more probable. In every case in which fluid is suspected the exploring needle should be used, because of the great importance of an early diagnosis. The skin should be washed and the needle sterilized. Pus may not be found because the needle is too small, too short, or because it is introduced too far into the chest; for when the layer of pus is thin, the needle may be pushed through this into the lung.

The physical signs upon which most reliance is to be placed are, marked dulness or flatness on percussion, feeble breathing, and displacement of the heart. When in a young child these signs are present, whether general or localized, a needle should be inserted, and if pus is not found at the first trial, repeated punctures should be made until the presence or absence of fluid is definitely settled.

Empyema is most frequently confounded with unresolved pneumonia. The differential points are that in unresolved pneumonia the dulness is usually over a single lobe, râles or friction sounds are heard, and there is no displacement of the heart; empyema may give flatness over the whole lung, or over the lower half of the chest in front and behind, râles and friction sounds are absent over this area, and the heart is usually displaced. In both conditions we may get bronchial breathing and voice. The confusion of acute pneumonia or tuberculosis with empyema, generally arises from placing too much reliance upon auscultation. In pleuro-pneumonia, with an excessive exudation of fibrin, the signs may be identical with those of empyema, except that the heart is not displaced. I once saw pulmonary tuberculosis, with caseation of an entire lobe, which gave signs that were identical with those of a sacculated empyema. It is by the exploring needle, and by that alone, that empyema is positively differentiated from these pulmonary conditions.

There are some other thoracic diseases from which the diagnosis may be even more difficult. A large pericardial effusion gives signs which are in some cases identical with those of empyema of the left side. Marked displacement of the heart to the right is always a strong point in favour of empyema; besides, such pericardial effusions are extremely rare in young children. A pulmonary abscess of considerable size—also a rare condition—gives signs identical with those of localized empyema, and is only distinguished from it by autopsy or operation. Abscesses from broken-down tuberculous glands may give signs resembling those of localized empyema, and may point like an empyema between the ribs in the upper part of the chest. The constitutional symptoms of empyema may at times resemble typhoid fever or malaria; but it is distinguished from them by the physical signs.

Prognosis.—The outcome of a case of empyema depends chiefly upon the cause, the age and general condition of the patient, the duration of the symptoms, the presence or absence of serious complications, and the treatment. The best results are obtained in the cases that follow pneumonia. Tuberculosis before the seventh year is an exceedingly infrequent cause, and gangrene of the lung and general pyæmia are both rare causes in early life. It is these three conditions that make the prognosis of the disease in adults so serious. The mortality in infants under one year, particularly hospital cases, is high—fully 75 per cent—not only because of the tender age, but because of the wretched general condition of most of these patients. Empyema in children over two years old seen reasonably early—i. e., within six or eight weeks—and receiving proper treatment, almost invariably terminates in recovery, unless the disease is double or serious complications exist. Great delay in operation makes the prognosis worse, because the more difficult the expansion of the lung the more tedious is the disease, and the greater the likelihood of a sinus remaining. With proper early treatment these patients not only recover, but in most cases the recovery is surprisingly complete. Retraction of the chest and its resulting lateral curvature of the spine are rare, and seen only in neglected cases. In very many of the cases I have seen, in which a reasonably early operation was done, it was impossible, after the lapse of two or three years, to detect any difference whatever in the physical signs of the two sides of the chest. There are few serious diseases the treatment of which is more satisfactory than that of acute empyema following pneumonia.

Spontaneous recovery in empyema may take place by absorption; but this is so rare that it is not to be expected. The pus may be evacuated spontaneously through a bronchus, rupture having taken place through the visceral pleura. When this occurs, a large amount of pus may be coughed up in a few hours, usually followed by immediate, but not always lasting, improvement. This is the most favourable of the natural terminations. External opening may take place, usually about the nipple. There is an area of redness, then a fluctuating tumour, and finally the pointing of an abscess. The discharge may continue for months, or even for years. External opening rarely occurs until the disease has lasted several months. Of 19 cases of empyema in children collected by Schmidt, in which a spontaneous discharge of pus occurred either externally or through a bronchus, there were 17 deaths and 2 recoveries. Empyema may burrow behind the diaphragm into the abdominal cavity, appearing as a psoas abscess; it may burrow posteriorly into the lumbar region; it may rupture into the œsophagus, or through the diaphragm into the peritoneal cavity. All these conditions, however, are very rare. The chances of spontaneous cure in empyema are small. Of 32 cases, reported by Rilliet and Barthez, which received no surgical treatment, 21 proved fatal. The statistics of empyema before the general adoption of surgical treatment are simply appalling. Patients were either worn out by the protracted suppuration, or died from amyloid degeneration, pneumonia, or tuberculosis.

Treatment.—The medical treatment relates to the patient only; the disease is always to be treated surgically. Like any other acute abscess, empyema requires free incision and drainage with proper aseptic precautions.

Aspiration as a means of cure has been almost entirely given up in New York. Unquestionably it sometimes suffices to cure empyema, most frequently when it is localized. How often this occurs is shown by the following statistics: Of 139 cases which I collected that were treated by aspiration, 25 were cured, 8 of these by a single aspiration; 13 died, and the remaining 101 were afterward subjected to other treatment. The objections to aspiration are, that it is not possible to remove all the pus; that it affords no opportunity for the removal of the large fibrinous masses; and, finally, that it is only a possible means of cure. The terror caused by repeated aspirations is almost as great as that of incision without anæsthesia. Aspiration, therefore, is to be advised in children only for temporary relief when the amount of fluid is large and the symptoms are urgent.

Simple incision and drainage.—If possible I prefer to delay operation until the period of most acute inflammation has subsided, as shown by lower temperature and stationary physical signs. This is usually seen two or three weeks after the pleural invasion. Such delay is not admissible if either the local condition or the temperature points to a steady increase in the disease; nor when the general symptoms indicate increasing prostration or sepsis. The dangers attendant upon general anæsthesia are considerable, and in most cases it is better not to employ it. I have known of four deaths on the table during operation, and in several other cases have seen very alarming symptoms occur. Chloroform is more to be feared than ether. We should therefore rely upon local anæsthesia obtained by cocaine or by a spray of chloride of ethyl or ether. The most favourable point for incision is the posterior axillary line in the seventh intercostal space upon the right side, the eighth upon the left. In a case of a localized empyema, the lowest point at which pus can be obtained by puncture should be chosen. The incision is made in the middle of the intercostal space. No matter what has been found by puncture on previous occasions, the exploring needle should always be used at the time of operation and at the site of the incision before the latter is made. The cutaneous incision should be an inch and a half long, and the opening in the pleura made large enough to allow the little finger of the operator to pass into the pleural cavity. The hæmorrhage is very rarely sufficient to require a ligature. The wound may be held open by forceps or a tracheal dilator, and as much of the fibrin as possible removed at the time; or, if the patient's condition is bad, the tube may be immediately inserted and the dressings applied. The drainage tube should be of heavy rubber, fenestrated, three eighths or half an inch in diameter and four or five inches long. It is passed into the deepest pocket of the empyema. To secure it from slipping into the cavity, its outer end should be transfixed by a large safety-pin before its introduction. It is usually advisable for the first few days to insert two tubes side by side. This diminishes the danger of stopping the discharge by the plugging of the tube with fibrin. Gauze is placed over the wound beneath the safety-pin, and a compress of the same over the opening of the tube, the dressing being completed by a large mass of absorbent cotton and a snug roller bandage. The pus now slowly escapes into the dressing as the lung expands. When there is no reason for haste during the operation, a larger part of the pus may be removed before the application of the dressing. This should be allowed to escape slowly, the opening being closed from time to time by a compress. Ten or fifteen minutes may be consumed in evacuating the pus.

Both the original operation and the subsequent dressings should be done with strict aseptic precautions on account of the danger of secondary infection, the occurrence of which adds to the severity and prolongs the course of the disease. For the first day or two the dressings should be changed twice daily, then once a day for ten days or two weeks, and later at longer intervals. After the third day the second tube may be omitted and the remaining one gradually shortened. Usually by the end of the third week, and often before, the tube may be dispensed with altogether, the tract being kept open by a small roll of rubber tissue. The time of redressing and the removal of the tube is determined by the amount of discharge and the temperature. While this does not usually rise after the second day unless the drainage is imperfect, there are a number of conditions which may cause it to do so. The most important are: pneumonia, either a continuance of the old process or lighting up of a new one; abscess of the lung; empyema of the opposite side; pericarditis; tuberculosis; abscess from a necrosed rib; or some cause outside the chest — otitis, malaria, indigestion, or the onset of some other disease. The drainage should always be first suspected. The tube is often blocked by masses of fibrin, even when one of large size is used. At each dressing it is well to remove it to see if it is clear. The mistake is often made of allowing the tube to remain for too long a time, so that a sinus is kept open which would otherwise heal. Another mistake is that of allowing a very large tube to remain for too long a time; this may cause erosion of the periosteum and even necrosis of a rib. Washing out the pleural cavity is indicated only in cases in which the pus is foul. A single washing for the purpose of removing fibrin is the routine practice of some surgeons. For this a warm sterilized salt solution should be used. Personally I have not found this necessary. Repeated irrigations should not, I think, be employed. The usual duration of the discharge in cases treated by simple incision is from three to six weeks, the average being about five weeks.

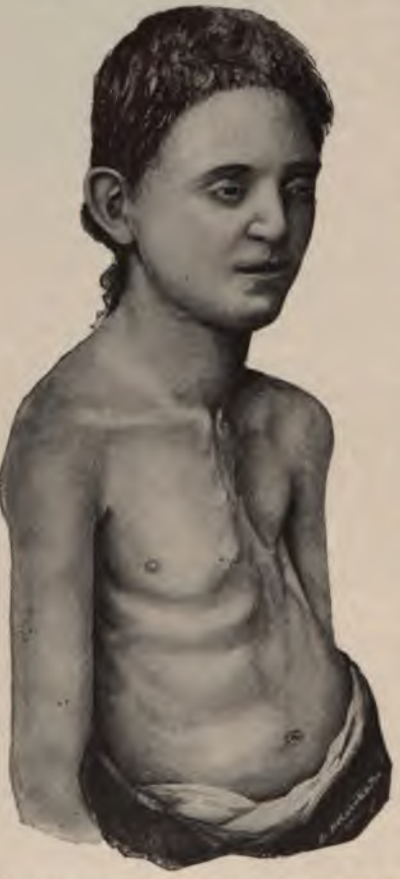

FIG. 113.—Deformity after an old empyema of the left side for which Estlander's operation was performed. Portions of five ribs were removed. (From a photograph seven years after operation.)

Resection of a rib.—Many of the best surgeons favour this as a routine procedure, with the belief that with the larger opening which is thus made, more perfect drainage is secured, that masses of fibrin can be removed with greater facility, and that it is altogether a more certain and efficient means of treatment than is a simple incision. While admitting some of the advantages claimed, my own experience has been that in the great majority of recent cases in young children, simple incision with drainage is all that is required. Rib resection is necessary whenever good drainage can not be secured by simple incision; especially if there is overlapping of the ribs, or if the intercostal spaces are very narrow. These are usually the cases in which the disease has lasted much longer than the average time. One inch of rib is all that it is necessary to remove. The periosteum is preserved, and there is rarely any permanent deformity.

In chronic cases, or those which have been long neglected, some further operative treatment is often necessary. The lung is so bound down by firm adhesions that further expansion is impossible, and even after the chest has receded to its utmost, so that the ribs are in contact, there still remains a cavity which can not close. For such cases the only hope is in an operation by which portions of several ribs are removed, thus allowing a greater collapse of the chest wall. This is known as thoracoplasty, or Estlander's operation. The operation is of itself a serious one, and only to be advised as a last resort in inveterate cases. Such an operation is, of course, always followed by very great deformity (Fig. 113).

Methods of inducing expansion of the lung.—In most of the cases, particularly the recent ones, complete expansion of the lung takes place without any difficulty, the chief agent being the cough. In some cases this may be insufficient. The apparatus, devised by James (New York), shown in the accompanying cut (Fig. 114) serves at the same time as a toy for the child's amusement and as a most efficient means of inducing forced expiration. One bottle is placed a few inches higher than the other, and the child blows a coloured fluid from the lower into the higher bottle, allowing it to siphon back. Blowing soap bubbles often answers the same purpose.

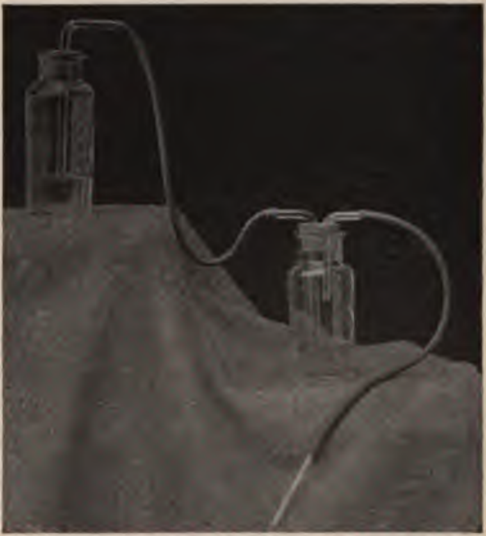
FIG. 114.—James's apparatus for expanding the lung after empyema.

SECTION V.

DISEASES OF THE CIRCULATORY SYSTEM.

CHAPTER I.

PECULIARITIES OF THE HEART AND CIRCULATION IN EARLY LIFE.

The Fœtal Circulation.—During the latter part of fœtal life the circulation may be briefly described as follows: The purified blood comes from the placenta through the umbilical vein. Entering the body, it divides at the under surface of the liver into two branches, the smaller one, the ductus venosus, communicating directly with the inferior vena cava; the larger branch joining the portal vein, so that its blood traverses the liver, and then enters the inferior vena cava through the hepatic vein. From the inferior vena cava the blood enters the right auricle, like that returned from the head and upper extremities by the superior vena cava. A part of the blood now passes directly into the left auricle through the foramen ovale; the remainder, through the tricuspid orifice into the right ventricle. As the requirements of the pulmonary circulation are not great, only a small part of the blood is sent through the pulmonary artery to the lungs; the greater portion passes from the pulmonary artery through the ductus arteriosus into the aorta, joining here the blood from the left ventricle. The blood thus finds its way from the right heart to the left, only in small part by way of the lungs, the greater part passing directly from the right auricle to the left, or from the right ventricle into the aorta through the ductus arteriosus. From the aorta, the blood reaches the placenta through the umbilical arteries, which are a continuation of the hypogastric arteries, which in turn are given off from the internal iliacs.

Changes in the Circulation at Birth.—With the ligation of the umbilical cord, the circulation through the umbilical vein and arteries and the ductus venosus ceases. With the establishment of respiration and the consequent increased demands made by the pulmonary circulation, the blood ceases almost at once to pass through the ductus arteriosus, and very soon through the foramen ovale. The umbilical vessels during the first few days of life are filled with small thrombi, which become organized. By the end of the first week, these vessels, as well as the ductus venosus, are usually closed at their extremities, although they may remain patulous throughout the greater part of their extent for several weeks. They subsequently atrophy to the condition of small fibrous cords. For some weeks before birth the circulation through the foramen ovale is slight, it being gradually obstructed by the growth of a septum which nearly fills the space at birth. After the first week of extra-uterine life very little, if any, blood passes through it, although complete closure of the foramen often does not take place until the middle of the first year. In fully one fourth of the autopsies I have made upon infants under six months old, there have been found minute openings at the margin of the foramen ovale, but they are usually oblique, and closed by the valvular curtain so as effectually to obstruct the current of blood. The ductus arteriosus is first closed by a clot, which becomes organized and blends with the products of a proliferating arteritis. It is rarely found open after the tenth day, and by the twentieth it is almost invariably obliterated.

The Pulse.—The pulse in early life is not only more frequent, but it is very much more variable than in adults. The following is the average pulse-rate in healthy children during sleep or perfect quiet:

Six to twelve months...........................	105 to 115 per minute.
Two to six years................................	90 " 105 " "
Seven to ten years..............................	80 " 90 " "
Eleven to fourteen years........................	75 " 85 " "

The pulse is a little more frequent in females than in males, and more frequent when sitting than when lying down. Muscular exercise or excitement increases the pulse-rate by from twenty to fifty beats. Very trivial causes disturb not only the frequency but the force of the pulse. The pulse in young infants may be irregular even in health and during sleep. When rapid, it is frequently irregular without any meaning. No dicrotism is seen in the pulse wave of early infancy, according to Blanche.*

The circulation is much more active in infancy than in later childhood; thus, according to Vierordt, the entire round of the circulation is accomplished in the newly born in twelve seconds; at three years, in fifteen seconds; in the adult, in twenty-two seconds.

Size and Growth.—The relative size of the heart is slightly greater in infancy than in later life, it being smallest at about the seventh year. The average weight at the different periods of life is as follows: †

AGE.	Ounces.	Grammes.	Ratio to body weight.
Birth............................	0·50	14	1 to 225
1 year...........................	1·25	35	
2 years..........................	1·87	53	
3 "	2·25	64	
7 "	2·80	80	1 to 280
14 "	5·84	166	1 to 222
Adult............................	8·50	241	1 to 226

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* See tracings in Archives of Pædiatrics, vol. v, p. 732.

† The figures in infancy are from one hundred and fifty-five observations made in the New York Infant Asylum; the others are taken from Sahli.

The growth of the heart is rapid during the first three years, and nearly proportionate to that of the body. It is slowest from the third to the tenth year, and most rapid from the eleventh to the fifteenth year. At birth, the thickness of the right ventricle is very nearly the same as that of the left, the ratio being 6 : 7. The left ventricle, however, grows very much more rapidly than the right, so that at the end of the second year the ratio is 1 : 2, which is nearly that of the rest of childhood.

Position of the Apex Beat.—In the infant the heart is placed somewhat higher, and occupies a position a little nearer the horizontal than in the adult. This is partly due to the higher position of the diaphragm. The apex beat is therefore higher and farther to the left than in adult life. According to the observations of Wassilewski and Starck, whose combined examinations with reference to this point were made upon over 2,100 children, the apex beat is, as a rule, outside the mammary line until the fourth year; if it is less than one third of an inch beyond the nipple, it can not be considered abnormal. From the fourth to the ninth year, the apex beat is in or near the mammary line. After the thirteenth year, under normal conditions, it is invariably within that line. During the first year the apex beat is usually found in the fourth intercostal space; from the first to the seventh year, it is found with about equal frequency in the fourth and the fifth spaces; after the seventh it is usually, and after the thirteenth year it is always, when normal, in the fifth space. The position of the apex beat may be considerably modified by severe deformities of the chest resulting from rickets, Pott's disease, or lateral curvature of the spine.

Examination of the Heart.—*Inspection.*—Bulging of the præcordia is a frequent and important sign of cardiac disease during childhood. The cardiac impulse is generally weaker than in the adult, and often it is difficult to locate the apex beat owing to the thick layer of adipose tissue covering the chest.

Palpation.—This is usually a much more satisfactory method than is inspection for determining the position of the apex beat. For this purpose the child should be in the sitting posture, with the body inclined slightly forward. Great displacement of the apex beat is always significant, and should lead one to suspect pleuritic effusion; lesser degrees of displacement to the left indicate hypertrophy, especially of the left ventricle; to the right, hypertrophy of the right ventricle, usually with a congenital malformation.

Percussion.—This is best done by means of the percussion hammer. A light blow should be used, on account of the thinness and elasticity of the chest walls. The outline of the area of "relative cardiac dulness," especially in small children, is proportionately larger than in the adult. This may lead to the mistaken opinion that the heart is enlarged, when it is really of normal size. According to Sahli,* the limits of this area are as follows: Above, the second space or lower border of the second costal cartilage; to the right, at the para-sternal line, sometimes slightly beyond it; to the left, at or slightly beyond the mammary line, this depending upon the age of the child. The lower border is indeterminable on account of the liver.

The area of "absolute cardiac dulness," or that part of the heart uncovered by the lung, resembles in shape the same area in the adult, but it is relatively larger. Its upper limit is the upper border of the third intercostal space, sometimes the third costal cartilage; it extends to the left to a point between the para-sternal and the mammary lines, and to the right as far as the left border of the sternum. These two areas will be readily understood by reference to the accompanying diagram (Fig. 115).

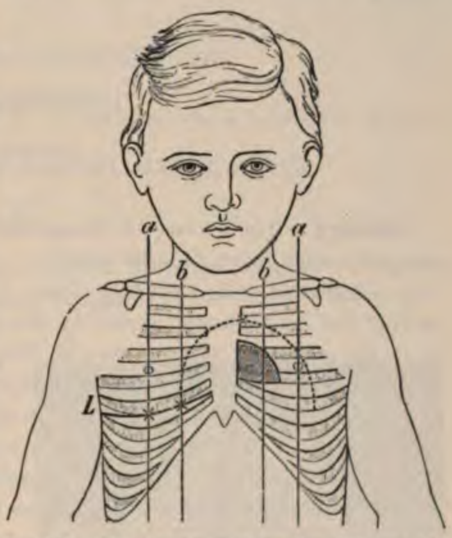

Fig. 115.—Showing areas of cardiac dulness: *a* is the mammary line; *b*, the para-sternal line; *L*, the upper border of the liver. The space enclosed by the dotted line represents the area of relative dulness; the heavily shaded area, that of absolute dulness. (After Sahli, slightly modified by Unger.)

Auscultation.—This is of little value unless the child is quiet. The preferable position is the sitting posture. For an accurate diagnosis the stethoscope is indispensable, but auscultation should always be practised with the naked ear as well. The rhythm and rapidity of the child's heart action are much more easily disturbed than are the adult's, and such disturbances are consequently much less significant. The rapidity of the heart in infancy is ordinarily so great as to make it practically impossible to distinguish between diastolic and presystolic murmurs. Normally, the loudest sound is the first sound at the apex; the weakest sound is the second sound at the aortic orifice. According to Hochsinger, the accentuation of the child's heart-sounds is upon the first sound, and not upon the second, as in the adult.

In consequence of the small size and the thin walls of the chest, all sounds, both normal and pathological, appear relatively louder than in the adult, and the area of diffusion is therefore much greater. Thus it is a frequent occurrence for murmurs to be heard all over the chest both in front and behind.

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* Topographische Percussion im Kindesalter, 1882.

Reduplication of the heart sounds, in consequence of the valves of the two sides not closing exactly together, is not uncommon in children, and may be due simply to excitement. During the first four years of life nearly all the abnormal murmurs heard are systolic.

Accidental murmurs may be due to anæmia and other blood conditions, and, although not so common as in older patients, they are by no means rare even in infants.

CHAPTER II.

CONGENITAL ANOMALIES OF THE HEART.

Etiology.—The causes of congenital anomalies of the heart may be grouped under three general heads:

1. Malformations resulting from imperfect development of certain parts of the heart, most frequently one of the septa. Either the ventricular or the auricular septum may be affected, or that dividing the pulmonary artery from the aorta. Such failure in development perpetuates conditions which are normal in the early months of fœtal life. There may also be atresia of any one of the orifices, absence of one or more of the valvular leaflets, or of any one of the large vessels.

2. Fœtal endocarditis. The effects of this condition vary according to the time of its occurrence. It is almost invariably of the right side, most frequently affecting the pulmonic valves. Valvular disease in fœtal life leads not only to hypertrophy and dilatation, but also interferes with the normal development of the heart by preventing the closure of the auricular or ventricular septum or the ductus arteriosus, these being kept open by way of compensation.

3. Persistence of fœtal conditions, such as the foramen ovale or ductus arteriosus. This may be the result of valvular disease, as previously stated, or of some condition of the lungs, such as atelectasis.

Lesions.—In the following table are given the lesions found in two hundred and forty-two cases, which I have collected from medical literature:

Frequency of the different lesions in 242 autopsies upon cases of congenital cardiac anomaly.

Defect in the ventricular septum	149	cases;	the only	lesion in	5	cases.
Defect in the auricular septum, or patent foramen ovale	126	“	“	“	9	“
Pulmonic stenosis or atresia	108	“	“	“	6	“
Patent ductus arteriosus	68	“	“	“	3	“

Abnormalities in the origin of the great vessels.	45	cases;	the only	lesion in	0	cases.
Pulmonic insufficiency	17	"	"	"	0	"
Tricuspid insufficiency	6	"	"	"	0	"
Tricuspid stenosis or atresia	3	"	"	"	0	"
Mitral insufficiency	1	"	"	"	0	"
Mitral stenosis or atresia	6	"	"	"	0	"
Aortic insufficiency	1	"	"	"	0	"
Aortic stenosis or atresia	6	"	"	"	0	"
Transposition of the heart	2	"	"	"	0	"
Ectocardia	1	"	"	"	0	"

The most frequent associated lesions.

Pulmonic stenosis, with defect in the ventricular septum	92	cases;	the only	lesion in	20	cases.
ulmonic stenosis, with defect in the auricular septum	52	"	"	"	8	"
Defects in both septa	82	"	"	"	17	"
Pulmonic stenosis and defects in both septa	36	"	"	"	21	"

From this table it will be seen that, in the great majority of cases, several lesions are present, the most frequent combinations being pulmonary stenosis with defective ventricular septum, pulmonic stenosis with defective auricular septum, the three lesions associated, or the first two with a patent ductus arteriosus.

Defect in the ventricular septum.—This is the most frequent lesion in congenital cardiac disease, and in half the cases was associated with pulmonic stenosis. The defect is generally at the upper part of the septum (Fig. 116). It is usually from one fourth to one half an inch in diameter, but not infrequently there is a large defect, and the septum may be entirely absent, the heart then consisting of but three cavities—two auricles and one ventricle. If the auricular septum also is wanting, as is often the case, the heart has but two cavities. Frequently there are also abnormalities in the origin of the great vessels. The pulmonary artery and the aorta may be given off from the common ventricle, or the aorta may arise partly from one ventricle and partly from the other. If pulmonic stenosis or atresia is present, the opening in the

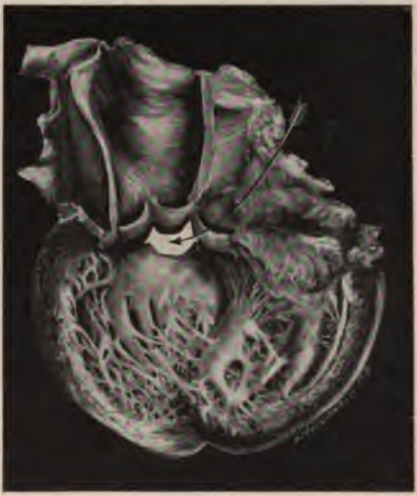

FIG. 116.—Congenital cardiac disease. The left ventricle is shown with a defect in the ventricular septum, the opening being just beneath the aortic valve. (From a patient dying in the Babies' Hospital.)

ventricular septum is conservative, affording a channel for the passage of blood from the right to the left side of the heart.

Patent foramen ovale, or defect in the auricular septum.—Although this is one of the most common congenital malformations, it is not one of the most important. It rarely occurs alone, but is frequently found with pulmonic stenosis or a defect in the ventricular septum. Small oblique openings in the auricular septum—usually at the foramen ovale—are not infrequently met with in autopsies upon young infants, but they are of no importance. In pathological conditions the opening is from one fourth to one inch in diameter, and there may be more than one opening. A defect in this septum is frequently secondary to pulmonic stenosis, or it may be a failure in development. A patent foramen ovale may be due to atelectasis.

Patent ductus arteriosus.—As a solitary lesion this is rare, but it is frequently associated with pulmonic stenosis, usually with a defect in one or both septa. It is then one of the channels by which the blood may find its way to the lungs when the pulmonary orifice is obstructed. It is not a malformation, but simply the persistence of a fœtal condition usually necessitated by other changes in the heart.

Pulmonic stenosis.—This is one of the most frequent and most important lesions. It may be due to fœtal endocarditis, or to a malformation. If the former, there is usually stenosis; if the latter, there may be atresia. It is often a primary lesion, and when marked it is always accompanied by other changes, most frequently by a defect in one or both septa or by a patent ductus arteriosus. This is important, as being more constantly associated with cyanosis than is any other congenital lesion. The amount of obstruction varies from a slight narrowing of the orifice to complete atresia. If there is atresia, the pulmonary artery is very small, and may be rudimentary.

Pulmonic insufficiency.—This lesion is relatively rare. It is usually the result of fœtal endocarditis, but there may be absence of the pulmonary valve. It is most frequently associated with a defect in the ventricular septum.

Tricuspid, mitral, and aortic disease are all very infrequent and usually seen in cases with multiple defects. Atresia or stenosis is much more common than insufficiency.

Abnormalities in the origin of the large vessels.—These are quite frequent; but, as will be seen from the table, they are always associated with other lesions. Three forms are seen: (1) Transposition of the large vessels —the pulmonary artery is given off from the left, and the aorta from the right ventricle. (2) Both arteries arise from a common trunk. This is usually due to an incomplete development of the lower part of the septum dividing the two arteries. Usually the pulmonary artery appears to be a branch of the aorta. This condition is frequently associated with other abnormalities, often with so large a defect in the ventricular septum that there is really but one ventricle. (3) The aorta has an abnormal origin, arising from the right ventricle, or partly from both ventricles. This also is associated with a large defect in the ventricular septum. When described as arising from both ventricles, the aorta is usually given off directly above the line of the septum.

An abnormality in the number of valvular segments is quite frequent, but seldom impairs the valve's function. In rare cases a valve is rudimentary, and it may be absent, generally at the pulmonic or tricuspid orifice. Absence of the right auricle and absence of the pericardium have been recorded; also opening of the pulmonary veins into the right auricle, and a single pulmonary artery. In one case in the series there was ectocardia, this being associated with a congenital fissure of the sternum. I once saw a very remarkable instance of congenital cardiac displacement; the heart was situated in the abdominal cavity. Its pulsations could be plainly seen and felt just above the umbilicus.

Transposition of the heart, or true dextro-cardia, was recorded but twice in this series of cases. It was, however, simulated in several others, including one of my own, where the apex beat was to the right of the sternum. There was in this case great hypertrophy of the right ventricle with a rudimentary ventricular septum.

Secondary lesions.—In congenital malformations the right heart is usually found hypertrophied, since there remain one or more of the fœtal conditions in which the greater part of the work is thrown upon the right ventricle. Such hypertrophy is in most cases accompanied by some dilatation. Changes in the wall of the left heart alone are exceedingly rare. In four cases there was evidence of malignant endocarditis, which was the cause of death, all but one of these patients being adults.

Symptoms.—The symptoms of congenital cardiac disease are usually manifested soon after birth. Of 128 cases in which the time of the first symptoms was noted, they were congenital, or appeared during the first month, in 85; after one month and during the first year, in 18; from one to sixteen years, in 15; while in 10 no symptoms were observed until after puberty. Congenital cardiac disease is one of the causes, but not a frequent one, of death during the first days of life.

The most striking objective symptom is cyanosis. This is present in over four-fifths of the severe cases; but cyanosis may be absent, even with serious lesions. It may be slight and noticed only upon exertion, as upon coughing or crying, or it may be intense and constant, giving the skin a dark, leaden colour, and the mucous membrane of the mouth a raspberry hue. The view that cyanosis depends upon an admixture of arterial and venous blood is generally discredited. In the great majority of the cases at least, the explanation is a deficient oxidation of the blood in the lungs, owing to some interference with the sternum in the second or third intercostal space, and widely diffused, often being audible all over the chest. In the great majority of cases this is heard alone; in a smaller number a double murmur is present. A systolic murmur may be due to pulmonic stenosis, deficient ventricular septum, patent ductus arteriosus, mitral regurgitation, tricuspid regurgitation, or aortic stenosis. Since these conditions are very often associated, it is difficult to tell upon which one the murmur depends. In a young child, a loud murmur at the base with cyanosis, almost always means congenital disease.

Enlargement of the right heart, chiefly from ventricular hypertrophy, is present in most of the cases.

A diagnosis of the precise nature of the malformation is very difficult, and in the great majority of cases only a probable diagnosis is possible. Nearly all the cases are complex, and the variety of combinations is very great. A study of the histories and autopsies of the cases in this series reveals many apparently contradictory facts. Loud murmurs are sometimes heard which are difficult to explain by the lesions, and murmurs may be absent when there is every reason from the post-mortem findings for expecting their presence. Certain lesions like aortic stenosis, mitral stenosis, and mitral regurgitation may be accompanied by the same signs as in acquired disease. With reference to the other conditions, I can not do better than give the more frequent clinical symptoms with the results of the autopsies in the series of cases which I have collected.

A systolic murmur at the base, with cyanosis.—This was the most common combination met with, and was present in about one third of the entire number. In over 80 per cent of the cases with these symptoms, pulmonic stenosis was found. The remainder were complicated cases of quite a wide variety. Pulmonic stenosis was usually associated with a defect in one of the cardiac septa, or a patent ductus arteriosus.

A systolic murmur without cyanosis.—In this series of autopsies this was not a frequent combination, being noted but six times. It is usually dependent upon a defect in the ventricular septum without pulmonic stenosis. Clinically, however, this is more often seen. The murmur is generally loudest at the left margin of the sternum at the third space. There is a striking absence of all other symptoms. I have watched a number of such patients for many years who have remained in perfect health.

A systolic murmur at the apex with cyanosis.—Of the six cases with this combination, all were examples of complex malformation, the most frequent lesions being a defect in the auricular septum, transposition of the great vessels, and patent ductus arteriosus.

Cyanosis without murmurs was noted fourteen times. It indicates either pulmonic atresia or the transposition or irregular origin of the great vessels.

Diastolic murmurs were heard in two cases, and depended upon pulmonic insufficiency.

Absence of both cyanosis and murmurs was recorded in five cases. The lesions found were: atresia of the aorta, both arteries arising from the right ventricle, or defective septa.

The only cases, therefore, in which a fairly certain anatomical diagnosis can be made are those of pulmonic stenosis with a deficient ventricular septum.

Diagnosis of congenital from acquired disease.—Congenital disease may be suspected if the patient is under two years of age; if there is no history of previous rheumatism; if the murmur is atypical in its location, character, or transmission; if there is a very loud murmur at the base, and if there is evidence of enlargement of the right heart. If cyanosis and clubbing of the fingers are present the diagnosis is certain.

Especially difficult are the cases without cyanosis seen in older children. Absence of hypertrophy of the left ventricle, continued absence of subjective symptoms, even with a very loud murmur, and a lesion which does not increase, all point strongly to a congenital malformation.

Diagnosis of congenital from anæmic murmurs.—This is often a more difficult matter than to decide between congenital and acquired disease. From a murmur alone one should be very cautious in making a diagnosis of cardiac malformation in a very anæmic infant. Anæmic murmurs are systolic, basic, unaccompanied by enlargement of the heart; usually heard in the carotids, often in the subclavian arteries, but are seldom so loud as those due to malformations. In some cases it may be necessary to watch the effect of treatment before deciding the question.

Prognosis.—Of 225 cases, 60 per cent were fatal before the end of the fifth year, and nearly one-half of these during the first two months; while 16 per cent of the cases lived over sixteen years, and 8 per cent over thirty years. The prognosis in any given case is to be made from the general condition of the patient and how well the circulation is carried on, rather than from the intensity of the cyanosis or the character of the murmur, although extreme cyanosis is always unfavourable.

In the cases fatal soon after birth the usual lesions are large defects in the septa, transposition of the great vessels, or pulmonic atresia. In five of twenty-three cases dying thus early, the heart had but two cavities. Lesions which are compatible with the longest life are minor septum defects, and pulmonic stenosis which can be compensated for by hypertrophy of the right ventricle. Many exceptional instances are recorded in which patients have lived a long time in spite of extreme deformities. One child with transposition of the pulmonary artery and aorta lived two and a half years. Tiedmann's case lived eleven years with a heart consisting of three cavities —two auricles and one ventricle—and with constant cyanosis. In three cases reported by Rokitansky, the patients lived over forty years with rudimentary auricular septa and no cyanosis mentioned. Gelpke's case had cyanosis, and lived twenty-seven years with rudimentary auricular and ventricular septa, and with no tricuspid opening.

Treatment.—No treatment is of the slightest avail in diminishing the amount of deformity or promoting the closure of any of the abnormal openings. All cases are to be treated symptomatically.

CHAPTER III.

PERICARDITIS.

INFLAMMATION of the pericardium is a rare disease in infancy and early childhood, only two cases being seen in seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum. In later childhood the disease is more frequent. In its etiology, symptoms, and course it resembles quite closely the same disease in adults.

Etiology.—Of 69 cases of pericarditis in children under fourteen years of age, 24 occurred before the third year, 12 between the third and seventh years, and 33 between the seventh and fourteenth years. It has been seen in the newly born, and has been found even in the fœtus.

Pericarditis is almost invariably a secondary disease, following (1) pleurisy or pleuro-pneumonia; (2) acute rheumatism; (3) acute infectious diseases, especially scarlet fever; (4) pyæmia; (5) tuberculosis; (6) local conditions. The relative importance of these causes differs with the age of the child. In infancy and early childhood most of the cases complicate disease of the lung or pleura, usually of the left side. After the fourth year rheumatism takes the first place as an etiological factor. Pericarditis is then generally associated with endocarditis, and may precede or follow the articular manifestations of rheumatism. Following scarlet fever, pericarditis generally occurs in connection with nephritis or multiple joint inflammations. In typhoid fever, also, it is usually associated with pneumonia or joint lesions. Pyæmia may be a cause in the newly born, or it may occur in connection with disease of the bones or joints in older children; in both it is usually associated with similar lesions of other serous membranes. Tuberculous pericarditis is more frequent after the third year, and is generally secondary to pulmonary tuberculosis. Among the local causes may be mentioned traumatism, ulceration of a foreign body from the œsophagus into the pericardium, disease of the sternum, ribs, or vertebræ, and abscesses resulting from cheesy bronchial lymph nodes.

Lesions.—1. *Pericardial transudations*, or an increase in the normal pericardial fluid, are met with in many conditions in which there is a very marked degree of anæmia, general dropsy, or a weak heart, particularly of the right side. Generally from one and a half to two ounces of a clear serum are found in the pericardial sac.

2. *External or mediastinal pericarditis* is always associated with mediastinal pleurisy, and results in more or less extensive adhesions of the pericardial and pleural surfaces, with an increase in the connective tissue of the mediastinum. It is often a tuberculous process. When severe, it may cause compression of the large blood-vessels, and seldom in any other way produces symptoms. With this form there may be inflammation of the internal layer of the pericardium. It is only inflammation of the internal layer which is ordinarily considered as pericarditis, the other form being preferably classed as *mediastinitis.*

3. *Dry pericarditis.*—This may be either general or localized. If the latter, it is more often seen at the base than at the apex of the heart. The two opposing surfaces are usually involved. As a result of the inflammation they are coated with fibrin, which may be partly absorbed, but usually leaves behind bands of adhesions of greater or less extent. From repeated attacks there may result complete obliteration of the pericardial sac.

4. *The sero-fibrinous form—pericarditis with effusion.*—This is the most common variety. The heart appears roughened from the exudate which often completely covers it, forming bands which extend from one surface to the other. The serum may be clear, or contain flakes of lymph, and varies in amount from a few ounces to a pint. In cases terminating in recovery there is gradual absorption of the serum and part of the fibrin, but adhesions more or less extensive always remain.

5. *Purulent pericarditis.*—If the inflammation is set up by a foreign body ulcerating into the sac, by the rupture of a mediastinal abscess, or by general pyæmia, the process may be purulent from the outset. More frequently, however, in purulent pericarditis there is first an abundant exudation of fibrin with pus cells in its meshes, and subsequently the pouring out of fluid pus, precisely as in empyema, with which it is very often associated. If death occurs in the early stage, both surfaces of the pericardium are found coated with a thick exudate of greenish-yellow lymph, but little or no fluid pus may be present. At a later period the pericardial sac contains pus, which may vary in amount from a few ounces to one or two pints. Purulent pericarditis, which is secondary to pneumonia or pleurisy, is usually due to the pneumococcus. In other cases any of the pyogenic germs may be found.

6. *Pericarditis with an effusion of blood* is very rare in children. It may occur from the rupture of organized adhesions or in certain blood states such as purpura, and very rarely in tuberculosis.

Pericarditis complicating pneumonia and pleurisy is generally fibrinous or fibrino-purulent; that with rheumatism is sero-fibrinous, and often accompanied by endocarditis. With acute tuberculosis there is usually only a deposit of miliary tubercles, or there may be a small serous or sero-sanguinolent effusion. In chronic cases there may be a tuberculous inflammation with the formation of caseous nodules, new connective tissue, and extensive adhesions. This generally occurs in connection with pulmonary tuberculosis—sometimes with tuberculous peritonitis.

In any form of pericarditis complete recovery, so far as pathological conditions are concerned, is rare—if, indeed, it ever occurs. Generally adhesions remain, which may be in the form of a few thin connective-tissue bands, or so extensive as to produce almost entire obliteration of the pericardial sac. Such adhesions are usually followed by secondary changes. The growth and development of the heart are interfered with, and there may be sufficient pressure upon the coronary vessels to lead to degeneration of the muscular walls and dilatation of the heart. With large fluid exudations there may be an interference with the systemic circulation, enlargement of the spleen and liver, and sometimes general dropsy.

Symptoms.—A pericardial transudation, or dropsy of the pericardium, is very rarely large enough to make a diagnosis possible.

External pericarditis is seldom recognised during life, there being no symptoms except those of the pleurisy with which it is associated. Occasionally there may be heard, particularly if the inflammation is anterior, a pleuritic friction sound which is increased with the systole of the heart. The pulse may be weak during inspiration, and there may be an increased area of cardiac dulness. If the inflammation is chiefly posterior, it causes only the symptoms of mediastinitis, which is recognised principally by its pressure effects upon the great vessels. It may produce œdema of the face or of the lower extremities, ascites, enlargement of the liver and spleen, but rarely albuminuria. It is usually progressive, and lasts from a few months to two or three years, according to its cause.

Inflammation of the internal layer is the only form usually described as pericarditis. This is very frequently overlooked, not only on account of its rarity, but from the obscurity of its symptoms. The difficulty in diagnosis is particularly great in young children. The symptoms are few, and many of them are equivocal. As this disease is nearly always secondary, the physician should be on the watch for it in infants with pleurisy or pleuro-pneumonia of the left side, and in older children in the course of articular rheumatism. Localized pain and tenderness may be present, and also a certain amount of embarrassment of the heart's action, usually manifested by præcordial distress, palpitation, and slight irregularity of the pulse. There may be dyspnœa, and if there is a large effusion present there may be orthopnœa and cyanosis. Sometimes there is delirium. When pericarditis follows pleurisy or pleuro-pneumonia there are frequently no new symptoms added.

The physical signs in older children resemble those in adults. In dry pericarditis there is usually heard a double friction sound over the præcordial space, the area being generally small and near the base of the heart. The sound is not transmitted, and bears no relation to the respiratory movements. After effusion has taken place the apex beat may be displaced upward, diffused, and somewhat indistinct, or it may not be found at all. There may be bulging of the chest wall. On palpation, there is an absence of vocal fremitus over an area usually occupied by the lung. Percussion gives an area of marked dulness or flatness of triangular shape, the base being below and the apex above. The normal area of cardiac dulness is increased in all directions, and this dulness extends beyond the limits of the heart. On auscultation, the heart sounds are feeble and distant. Friction sounds disappear as serum is poured out, and reappear as it is absorbed. Endocardial murmurs may also be present. In infants, physical signs are often entirely wanting, or the normal sounds may be feeble, distant, or absent.

The usual duration of acute pericarditis is from one to three weeks. The ordinary dry form, with its resulting adhesions, may be followed by a subacute or chronic form of the disease. In the sero-fibrinous form the serum is usually absorbed quite promptly, and only adhesions are left, or a chronic inflammation follows, with exacerbations in each recurrence of rheumatism. In the purulent form of the disease in young children, death is the most frequent termination. If the pus is evacuated, or spontaneous opening takes place, there may be recovery, but always with more or less extensive adhesions remaining.

Prognosis.—Of thirty-five cases in Steffen's collection, only six recovered. This statement is to be taken rather as evidence of the great difficulty of diagnosis than of a very high mortality, although the disease is always a serious one. The prognosis depends chiefly upon the exciting cause. When due to pyæmia or the acute infectious diseases, or when extending from pleurisy or pneumonia, the prognosis is bad. Here it is usually the primary disease rather than the pericarditis which is the cause of death; the latter may be the case, however, if the effusion is large. The cases in which the pericarditis itself is the most important disease are those depending upon rheumatism. Although immediate danger to life may not often be great, yet the remote consequences of the disease, by reason of adhesions and subsequent dilatation, are frequently very serious.

Diagnosis.—Owing to the very rapid action of the heart in children, acute dry pericarditis presents difficulties of diagnosis in early life which are not met with in the adult. The disease is fortunately so rare under three years, that in ordinary practice it need seldom be considered. In older children the diagnosis is to be made by essentially the same signs as in adults. Pericarditis with effusion is to be diagnosticated from dilatation of the heart and from pleuritic effusions. From dilatation, the diagnosis is not often difficult in childhood, for this is not a common condition, and is rarely extreme except in advanced valvular disease. From pleuritic effusions the diagnosis is at times almost impossible. Signs pointing to a sacculated empyema of the left side anteriorly should always be regarded with suspicion, particularly if the apex beat is not displaced to the right, and if the heart sounds are very feeble. When empyema and pericarditis coexist, it may be impossible to recognise the condition. The diagnosis between serous and purulent effusions can be made only by aspiration. Fluid effusions in infants are almost invariably purulent, and so also are they in the majority of cases in older children, unless due to rheumatism.

Treatment.—In the early part of an attack of acute pericarditis the patient should be kept in bed and as quiet as possible, and hot poultices or counter-irritation by mustard used over the heart. Sometimes an ice bag may with advantage be substituted. Excessive heart action may be controlled by aconite, and severe pain requires usually opium. If the disease is due to rheumatism, anti-rheumatic remedies should be employed. Serous effusions usually subside under simple tonic treatment. If absorption is slow, it may be hastened by counter-irritation. When a large effusion forms rapidly there may be danger of death from syncope. Symptoms which indicate an unfavourable termination are cyanosis, weak, irregular pulse, and great dyspnœa, or orthopnœa. Under these conditions aspiration may afford temporary relief, and free diuresis should be induced by citrate of potash and caffein. The inhalation of oxygen is at times of great value in cases presenting such urgent symptoms. If pus is shown to be present by puncture, incision and drainage should be practised, as in empyema. The results of aspiration in such cases are extremely unfavourable. Of eighteen cases of aspiration of the pericardium collected by Keating, only four recovered. In puncturing the pericardium the point usually selected is a little to the left of the border of the sternum in the fifth intercostal space, the needle being directed upward and outward.

CHRONIC PERICARDITIS WITH ADHESIONS.

This is not a very uncommon condition. It may be general or localized. The youngest case which has come under my observation was in a female child sixteen months old, who died from acute broncho-pneumonia. The adhesions were old and general, the pericardial sac being completely obliterated. Chronic adhesive pericarditis may follow single or repeated attacks of acute rheumatic pericarditis; or there may be no history of any prior attack, the condition being apparently chronic from the beginning. Osler has reported a case in which a similar lesion of the peritonæum was present. The pericardium may become very greatly thickened and its cavity obliterated; it may be adherent externally to the pleura, diaphragm, or chest wall. Other changes are usually present in the heart. It is often the seat of chronic myocarditis; the cavities may be greatly dilated, and the heart walls very much hypertrophied. Valvular lesions may be present.

Partial adhesions cause no symptoms by which they can be recognised, and even general adhesions sufficient to obliterate the pericardial sac may be found at autopsy when not suspected during life. This is one of the conditions in which, after it has led to considerable dilatation of the heart, sudden death sometimes occurs.

The heart is almost invariably much enlarged, chiefly from dilatation. On inspection, there may be bulging of the chest wall, with a diffused and often feeble or absent apex beat. The characteristic signs are a systolic retraction of the chest at or near the apex of the heart, sometimes at the tip of the sternum. This is due to the external pericardial adhesions, and is often better appreciated by palpation than by inspection. It is followed by a rapid rebound, associated with diastolic collapse of the jugular veins. A similar retraction, according to Broadbent, is to be seen behind in the infra-scapular region, sometimes on the left and sometimes on the right side. Percussion shows an increase in the cardiac dulness in all directions. The position of the apex and the percussion outline of the heart do not change with the posture of the patient, and the cardiac dulness is but little affected by full inspiration. A systolic murmur is often present. The diagnosis of adherent pericardium always presents difficulties, but it can be made with tolerable certainty in a considerable proportion of the cases. On account of the enlargement of the heart and the frequency of murmurs, it is usually mistaken for valvular disease. The lesion is a permanent one, and tends to increase. The treatment is symptomatic.

CHAPTER IV.

ENDOCARDITIS AND VALVULAR DISEASE.

ACUTE SIMPLE ENDOCARDITIS.

ACUTE endocarditis may occur even in fœtal life. At this period it usually affects the right side of the heart, and is one of the important causes of congenital malformations. In infancy, acute endocarditis is exceedingly rare, not a single instance being found in over one thousand autopsies upon children under three years of age of which I have records. From the third to the fifth year it is not so rare, and after this period it is quite common. Of 95 cases observed by Steffen, 15 occurred before the sixth year, and 80 between the sixth and fourteenth years.

Acute endocarditis may be primary, but it is much more frequently a secondary disease. The primary cases have been the subject of much discussion, but I agree with those who regard the great majority of these as rheumatic. Cheadle (London) has well said that we are to look upon endocarditis in children not as a complication of rheumatism, so much as a manifestation—often the first—of that disease. Sometimes endocarditis occurs alone, and sometimes it is associated with chorea without articular symptoms; but the latter almost invariably appear sooner or later. Endocarditis is seen as a frequent complication both of acute and of subacute articular rheumatism. The proportion of rheumatic cases in which it occurs is much larger in children than in adults. Compared with rheumatism, all other causes of acute endocarditis are very infrequent. It is seen occasionally in the course of nearly all the acute infectious diseases, most often with scarlet fever, and it sometimes complicates pleurisy and pneumonia, being usually associated with pericarditis. It may follow acute tonsillitis. In infectious diseases, and in pleurisy and pneumonia, the endocarditis is probably excited by pathogenic germs. Fraenkel and Sanger have found the staphylococcus in cases of simple endocarditis, and cultures by others have shown the presence of other pyogenic organisms, including the pneumococcus.

Lesions.—Acute inflammation may affect any part of the endocardium, but in extra-uterine life it usually affects the valves of the left side, involving the mitral much more frequently than the aortic valve. Steffen's figures give only four examples of aortic disease in ninety-five cases. (Compare statistics of valvular disease.)

The pathological changes consist first in an extensive growth of new connective-tissue cells and an infiltration of round cells beneath the endothelial layer. This results in the formation of small masses of granulation-tissue upon the valves or the endocardium of the heart wall, and upon these there is deposited fibrin from the blood. In this way the tiny wart-like excrescences known as vegetations are produced. Bacteria may also be caught in the exudate. As a consequence of the inflammation, the valve is swollen, somewhat shortened, and consequently insufficient. The results of the process may be ulceration of this new-formed tissue, which in ordinary cases is small in amount, or organization and cicatrization. Masses of fibrin may be detached from the vegetations and swept into the general circulation, lodging as emboli in the kidneys, spleen, brain, or other organs. This is not common in acute endocarditis, at least not in the first attacks.

In the milder forms of inflammation it is possible for complete recovery to take place, with the exception of a slight valvular thickening, not enough, however, to interfere in any way with the function of the valves. But this result is rare. In most cases they remain slightly insufficient, as the least serious consequence of the inflammation. Unfortunately, it more often happens that an acute inflammation which may not be at first serious, proves the beginning of the progressive changes of a chronic inflammation, the full effects of which are not seen for years. Chronic inflammation may follow the first attack immediately, or after a considerable interval, or occur after several acute attacks.

Symptoms.—When acute endocarditis occurs as a primary disease, or when it is the only manifestation of rheumatism, it usually begins abruptly with rather severe general symptoms—high temperature, often 102° to 105° F., prostration, exaggerated heart action, restlessness, and sometimes dyspnœa. There is nothing distinctive about these symptoms, and it is not until the heart is examined that the disease is recognised. If the heart is not watched, the diagnosis is not made, and there may be no suspicion of the nature of the attack until some time afterward, when the existence of valvular disease is discovered. If the heart is carefully examined from day to day, nothing abnormal may be found until the third or fourth day, or even later, when there is heard the characteristic soft, blowing, systolic murmur at the apex. The murmur is generally transmitted to the left. It may be accompanied by a thrill and by an accentuated pulmonic second sound, and later there may be evidence of slight dilatation with the usual signs of some degree of cardiac insufficiency. The murmur gradually increases in intensity until the maximum is reached, and then in most cases somewhat subsides.

Acute endocarditis sometimes occurs in the course of, or simultaneously with an attack of chorea, with symptoms quite similar to those above described. Finlayson (Glasgow) has called attention to endocarditis as a frequent cause of obscure fever in choreic patients, either when occurring alone or with articular symptoms. It may develop at any time during the choreic attack or subsequent to it. When endocarditis occurs as a complication of articular rheumatism, there may be an increase in the temperature and in the severity of the general symptoms, but rarely anything more definite. Endocarditis complicating other diseases is recognised only by the physical signs.

The usual duration of acute endocarditis is from one to three weeks, the febrile symptoms frequently subsiding in a few days and the cardiac symptoms slowly diminishing.

The attack may terminate fatally in the course of a few weeks, owing to the rapid development of acute dilatation, accompanied by the usual signs of cardiac insufficiency, with dropsy, cyanosis, and often pulmonary complications. Cerebral embolism may occur, which usually produces hemiplegia, but rarely results fatally. If emboli lodge in the spleen or kidneys, they may lead to swelling of the spleen or to hæmaturia. The patient may recover with a murmur which lasts but a few weeks and gradually disappears—a rare result. Usually there is a persistent murmur, with the subsequent development of the ordinary signs of valvular disease. Lastly, there may be recurrent attacks of inflammation, with the ultimate development of chronic valvular disease.

Diagnosis.—The diagnosis of acute endocarditis is very frequently not made; not because it is difficult, but because in young children the heart is not examined as frequently and as carefully as it should be. The symptoms are few and not diagnostic. It is therefore very important that not only in chorea and rheumatism, but in all acute febrile attacks, particularly those of obscure origin, the heart should be watched. Endocarditis affecting the wall of the heart can not be diagnosticated. The murmur of valvular endocarditis may be confounded with pericarditis, or with functional murmurs occurring in the course of febrile attacks, or with those of anæmic origin. From pericarditis it is distinguished by the fact that the murmur is single, has a soft blowing character, is usually located at the apex, is transmitted beyond the border of the heart, and is diminished by a full inspiration. Murmurs are often heard late in acute infectious diseases, especially diphtheria, scarlet fever, and typhoid, which closely simulate those of acute endocarditis. They are most frequently due to a relative insufficiency at the mitral orifice, generally caused by dilatation of the left ventricle. This produces a systolic murmur at the apex, transmitted to the left, often accompanied by an accentuated second pulmonic sound. A differential diagnosis between these conditions is often impossible except by following the course of the disease.

Prognosis.—The danger to life in acute endocarditis is not often great, as the disease seldom proves fatal. However, death may occur when it is associated with chorea, but here usually when an acute process is ingrafted upon an old valvular disease. In other cases, death results from complications, particularly pneumonia. Only the progress of the case enables one to decide how extensive is the damage which has been done to the valves. There is always the danger of recurrent attacks.

Treatment.—The most important thing in the management of these cases, and the one frequently overlooked, is to secure for the heart as complete rest as possible, not only during the period of acute inflammation, but for several succeeding weeks. With children this can be accomplished only by keeping them in bed, after mild attacks for at least a month, after severe attacks for three months. It is during this early period of the disease that changes take place most rapidly in the heart walls, and the gravest results sometimes follow the neglect of these precautions. Children are often allowed out of bed as soon as the fever has subsided, and the heart disease is unnoticed until a grave amount of dilatation has developed, with dropsy, palpitation, shortness of breath, slight cyanosis, irregular pulse, and cough. All the so-called primary cases, as well as those occurring with chorea and articular symptoms, should have the benefit of anti-rheumatic remedies, as this is the only plan which offers any chance of limiting the inflammation, although the effect upon the heart is rarely striking. Excessive cardiac action is sometimes allayed by aconite, sometimes best by opium. All children who have once suffered from endocarditis should be protected as much as possible from subsequent attacks of rheumatism.

MALIGNANT ENDOCARDITIS.

Malignant or ulcerative endocarditis is a rare disease in childhood. The youngest case I have found reported is that of Harris, which occurred in a boy four years old, and affected the right side of the heart. It was secondary to a cardiac malformation. Of the cases thus far reported in early life, about twenty-five in number, the great proportion have been in children over ten years of age, in whom the disease does not differ essentially from the adult type. For the most exhaustive study of this subject we are indebted to Osler's Gulstonian Lectures.

Malignant endocarditis rarely occurs as a primary affection. Of the acute diseases, it is most frequently secondary to pneumonia, next to rheumatism and meningitis. It may be met with in any infectious disease or septic process. In 75 per cent of the cases, according to Osler, it is ingrafted upon a previous valvular disease. In my series of collected cases of congenital malformations of the heart, there were four deaths from malignant endocarditis, all but one, however, occurring in adult life.

The bacteria most frequently associated are the staphylococcus and streptococcus, and, in the cases complicating pneumonia, the pneumococcus. These micro-organisms are believed to play an important part in the production of the disease. Circulating in the blood, they lodge upon the endocardium of the valves, all the more readily when the valves are previously diseased.

Lesions.—Malignant endocarditis may result in the production of vegetations which subsequently break down, or there may be superficial ulceration affecting only the endocardium, or deeper ulceration involving the valve, the septum, or even the heart wall. In other cases there is suppuration of the deeper tissues of the valve first affected, with the production of small abscesses at the base of the vegetations. These conditions may lead to large perforations, or even to the destruction of the valve, to valvular aneurisms, or to abscesses of the heart wall. According to Osler, the different parts of the heart are affected in the following order: mitral valve, aortic, mitral and aortic combined, tricuspid and pulmonic valves, and the cardiac wall. The secondary lesions of malignant endocarditis are due to emboli. These are most frequent in the spleen and kidney, next in the brain, intestines, and skin, and, if the right side of the heart is diseased, in the lungs. These emboli lead to the formation of red or white infarctions, to hæmorrhages, or to multiple abscesses in the various organs and tissues in which they lodge.

Symptoms.—Malignant endocarditis presents a great variety of symptoms, making the diagnosis extremely difficult in perhaps the majority of cases. There is generally a remittent type of fever, sometimes repeated rigors, profuse sweating, low delirium, stupor or coma, and extreme prostration. In many cases there is a fine petechial eruption upon the skin;

diarrhœa is also frequent. The cerebral symptoms may be so prominent as to suggest meningitis. There is usually a cardiac murmur, the location of which depends upon the seat of disease. It is most frequently the murmur of mitral regurgitation. This murmur is sometimes faint, and may be absent. The spleen is in most cases enlarged. From the emboli there may be hemiplegia, rapid swelling of the spleen, bloody urine, cough, and symptoms of pneumonia. The disease lasts from a few days to six weeks, death being the almost invariable termination. It is due to exhaustion or to some embolic process.

Diagnosis.—The most characteristic features of malignant endocarditis are the development of pyæmic or typhoid symptoms with a petechial eruption, in a patient who has previously had valvular disease. Malignant endocarditis is differentiated from typhoid fever by its sudden onset, irregular temperature, recurring chills, profuse sweats, petechial eruption, and dyspnœa. It may be confounded with malarial fever.

Treatment.—This is entirely symptomatic; no known measures have any influence upon the disease itself.

CHRONIC VALVULAR DISEASE.

Chronic valvular disease of the heart in children is usually the result of endocarditis; in a small number of cases it depends upon congenital malformation; but the degenerative lesions to which many adult cases are due have no place in early life.

Lesions.—The changes of chronic endocarditis may be briefly described as follows: The valvular segments are thickened by the production of new connective tissue, the contraction of which results in retraction, shortening, puckering, and imperfect closure of the valves. The valvular leaflets may adhere to each other, so that the opening is very much narrowed. This is sometimes reduced to a funnel-shaped orifice barely admitting the tip of the finger, and it may even be much smaller. The leaflets are sometimes adherent to the wall of the heart; the chordæ tendineæ are shortened, and sometimes entirely disappear; and, finally, the valves may be the seat of calcareous deposits. These changes take place very slowly, requiring many years for their full development. From time to time there may be attacks of acute inflammation. The changes described may bring about (1) valvular insufficiency, owing to imperfect closure, causing a regurgitation of blood through the opening guarded by the valve; or (2) stenosis, with such a narrowing of the opening that the outflow of blood is obstructed. In early life it is usually the mitral valve that is affected.

Of 141 cases in children under fourteen years old, observed clinically by Dr. F. M. Crandall and myself, the mitral valve was alone affected in 79 per cent; the aortic valve alone in 3 per cent; and both were associated in 18 per cent. Lesions of the aortic valve in early life are therefore comparatively rare.

Following valvular lesions, important changes take place in the wall and cavities of the heart: these are hypertrophy and dilatation.

Hypertrophy.—This consists in an increase in the thickness of the heart wall, due to an increase in the size and number of the muscular fibres. It is principally of the ventricles, and is always conservative. It may continue indefinitely, or it may be followed by degeneration and dilatation. Hypertrophy occurs as a result of any obstructive lesion at one of the cardiac orifices, in renal disease when the obstruction is in the small arteries, also when extra work is thrown upon the ventricles as a result of regurgitation, and it may follow primary dilatation.

Dilatation.—This consists in an enlargement of the cavities of the heart, usually with thinning of their walls. It is generally most marked in the auricles. Primary dilatation is produced by regurgitation of blood into any of the cavities as a result of valvular insufficiency. This may to a slight extent be regarded as a conservative lesion. Secondary dilatation, or that resulting from degeneration of the cardiac muscle, is always injurious. It is usually caused by imperfect nutrition of the heart which may be due to local or general causes. In most of the cases both hypertrophy and dilatation continue for a long time. So long as hypertrophy predominates, the circulation may be well carried on; but when dilatation comes to exceed hypertrophy, there are signs of great embarrassment to the circulation and of cardiac insufficiency.

There are other lesions accompanying chronic valvular disease, depending upon obstruction to the venous circulation. If this obstruction is in the pulmonary veins, it leads to congestion of the lungs, chronic bronchitis, or chronic pneumonia; if of the systemic venous circulation, it leads to chronic congestion of the spleen, liver, kidneys, peritonæum, and sometimes to general dropsy.

Etiology.—The following table gives the age and sex in the cases observed by Dr. Crandall and myself:

AGE.	1 year.	2 years.	3 years.	4 years.	5 years.	6 years.	7 years.	8 years.	9 years.	10 years.	11 years.	12 years.	13 years.	14 years.	Totals.
Males......	..	1	2	2	4	6	4	9	8	6	5	7	6	1	55, or 38%
Females....	..	1	3	5	7	9	10	3	11	12	14	4	2	3	90, " 62%
Total....	..	2	5	7	11	15	14	12	19	18	19	11	8	4	145

The difference in sex is very nearly the same as in my cases of rheumatism. Sturges, in 100 cases of chronic endocarditis gives 56 per cent females and 44 per cent males. Sansom's figures alone give a predominance of males.

The chronic endocarditis of early life is, as a rule, secondary to the acute or subacute form. Its etiological factors are therefore those of acute endocarditis. Of 117 cases in my own series, 93, or 80 per cent, gave a history of previous rheumatism—7 cases of chorea without articular symptoms being included as rheumatic. Of the 31 cases which at the first examination gave no history of rheumatism, 8 subsequently developed articular rheumatism, and 2 chorea, so that nearly 90 per cent of this series of cases presented, to my mind, conclusive evidence of a rheumatic diathesis. Thirty per cent had chorea previously, or developed it while under observation. The more closely I study cases of rheumatism, chorea, and valvular disease, and the longer the patients are kept under observation, the deeper becomes my conviction of the very close relationship between these three conditions in childhood. The percentage of rheumatic cases in this series is considerably larger than that given by many writers, but it corresponds very closely with Cheadle's careful observations. Valvular disease is occasionally traced to an attack of endocarditis complicating scarlet fever, and in rare cases to that occurring with other infectious diseases.

Symptoms.—The symptoms of chronic valvular disease in most cases come on slowly, often insidiously, and frequently there are none until the disease has lasted a long time, the condition being discovered by accident. The course of valvular disease is usually divided into two periods, the first being that while compensation is present, and the second after compensation has failed. The duration of the stage of compensation is indefinite; it may last a lifetime. The only subjective symptom that is of much diagnostic value is shortness of breath on exertion. Occasionally other symptoms are present, such as præcordial pain, attacks of palpitation, headache, epistaxis, anæmia, and cough. These are rarely constant, but come on when the patient's general condition for any reason is below normal. As a rule, there is in young subjects a tendency to an increase in the disease, although this is often slow, and may be interrupted by long periods in which the process appears to be stationary. At such times the patients either have no symptoms, or suffer only from a slight amount of inconvenience on marked exertion.

Failure in compensation is generally brought about by one of the following causes: There may be an intercurrent attack of acute endocarditis, which in a short time leads to a very great increase in the heart's disability. It may be due to additional work thrown upon the heart from excessive muscular exertion, or to the strain of a prolonged attack of some acute illness, especially one that is liable to produce changes in the heart muscle, such as typhoid or scarlet fever. It is sometimes the increased work which is physiologically thrown upon the heart at the time of puberty, owing to the rapid growth of the body. It may result from any cause which seriously affects the patient's general nutrition, particularly when this is associated with marked anæmia.

The symptoms indicating failure of compensation are those depending upon a weak heart, with imperfect filling of the arteries and overfilling of the veins. The embarrassment of the pulmonary circulation leads to constant dyspnœa or orthopnœa and cough, sometimes accompanied by profuse expectoration, which may be bloody, and in rare cases there may be larger pulmonary hæmorrhages. The obstruction to the systemic venous circulation leads to dropsy, which begins in the feet. There may be general anasarca and dropsy of the serous cavities, especially the peritonæum and pleura; also enlargement and functional disturbances of the liver, enlargement of the spleen, dyspeptic symptoms, and chronic congestion of the kidney, with scanty urine and albuminuria. There may be dilatation of the superficial veins, with clubbing of the fingers, and cyanosis; and there may be cerebral symptoms, such as headache, dizziness, and fainting attacks. The pulse is small and soft, and the heart's action rapid and irregular.

It is rare to see all the symptoms of cardiac failure in children under ten years, but about the time of puberty they are not uncommon. The symptoms may increase in severity until death occurs, or they may be severe for a time and then nearly disappear, to return again after a longer or shorter interval.* Death may be due to sudden cardiac paralysis, to intercurrent nephritis, pneumonia, embolism, inflammation of the serous membranes, or to œdema of the lungs.

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* The course and termination of these cases of chronic valvular disease is well illustrated by the following history of a little girl who was under my observation for nine years: When first seen she was seven years old, and gave a history of cardiac symptoms for one year. There was then present a loud mitral regurgitant murmur, with considerable hypertrophy. There was general dropsy, and all the symptoms pointed toward acute dilatation. Under treatment, the dropsy and other symptoms disappeared, and she went on comfortably for over a year. In her eighth and ninth years there were frequent attacks of subacute rheumatism, during which time the heart lesion steadily increased in severity. At twelve years there was an eruption of subcutaneous tendinous nodules, which remained for over two years. During this year there was heard for the first time a mitral direct murmur, accompanied by a very marked thrill, mitral stenosis having been gradually brought about by the slowly progressing endocarditis. This murmur gradually increased in intensity from that time, while the mitral regurgitant murmur became less distinct. The apex beat was then in the sixth space, two and a half inches to the left of the nipple. From the twelfth to the fifteenth year she grew very little in height or weight, and showed no signs of maturity, the cardiac symptoms being nearly stationary. In the fifteenth year she developed a marked enlargement of the liver and spleen with general dropsy and all the symptoms of cardiac insufficiency, these being the first symptoms of this character since she was seven years old. There was now heard for the first time an aortic regurgitant murmur in addition to the others formerly present. The symptoms disappeared under treatment in the course of a few months, but six months later returned with greater severity and were accompanied by albuminuria, the patient dying from heart failure in a few weeks. During the last exacerbation there was heard a double aortic as well as a double mitral murmur.

At autopsy the heart weighed fifteen ounces. There was a very great hypertrophy, especially of the right ventricle, which was as thick as the left. All the cavities were much dilated. The most important valvular lesion was mitral stenosis, the orifice not admitting the end of the little finger. The valves were the seat of calcareous deposits. The curtains of the aortic valve were thickened and adherent; there was also thickening of the pulmonic and tricuspid valves.

Clinical Varieties.—Of the 141 cases of valvular disease in children under fourteen years, previously referred to, the following were the forms and combinations recorded. It is to be noted that these figures are based upon clinical and not pathological examinations:

Mitral insufficiency	131 cases;	alone in	99 cases.
Mitral stenosis	17 "	" "	4 "
Aortic insufficiency	9 "	" "	0 "
Aortic stenosis	28 "	" "	3 "
Double mitral	8 "		
Double aortic	1 case.		
Double mitral and double aortic	3 cases.		
Mitral insufficiency and double aortic	3 "		
Mitral insufficiency and aortic stenosis	18 "		
Mitral stenosis and aortic insufficiency	2 "		

Mitral insufficiency.—This is usually the result of attacks of acute endocarditis. It is by far the most frequent form of valvular disease in early life, occurring in 93 per cent of the above cases, and alone in 70 per cent. In mitral insufficiency there is regurgitation of blood from the left ventricle into the left auricle during systole. This is compensated for by hypertrophy of both ventricles. It causes dilatation of the left auricle, increased pressure in the pulmonary veins, afterward in the pulmonary arteries, hypertrophy of the right ventricle, and, finally, there is dilatation of the right ventricle, tricuspid insufficiency, dilatation of the right auricle, and general systemic venous obstruction. Coincident with the changes in the right heart there is hypertrophy of the left ventricle, followed by dilatation.

In mitral insufficiency there is heard a systolic murmur which is synchronous with the apex impulse and with the first sound of the heart, and may in part replace the first sound. It is loudest at the apex, transmitted to the left, and heard with almost equal distinctness at the inferior angle of the left scapula. This is a very diffusible murmur, and may be audible all over the chest. It is accompanied by an accentuation of the pulmonic second sound heard at the left border of the sternum in the second space, and by signs of hypertrophy of the heart. When both these signs are wanting, the existence of mitral insufficiency is somewhat doubtful, as a similar murmur may be of functional or accidental origin. In the early stages of the disease the signs of hypertrophy predominate; in the later stages, those of dilatation.

In hypertrophy of the left ventricle or of the whole heart, the apex beat is displaced downward and to the left.* It may be in the fifth or the sixth space, but rarely lower, and as far to the left as the axillary line. There is often bulging of the præcordia, so marked as to cause a deformity of the chest. The impulse is forcible and heaving, and over a larger space than normal. The area of cardiac dulness is increased in all directions, but particularly downward and to the left. In hypertrophy involving chiefly the right ventricle, there may be bulging of the lower part of the sternum, and the area of dulness is increased to the right, in extreme cases extending from one to one and a half inches beyond the right border of the sternum. The heart sounds in hypertrophy are loud and distinct, and often have a somewhat metallic character. With hypertrophy of the right ventricle there may be reduplication or accentuation of the second sound. The pulse is full and strong.

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* For normal position of the apex in childhood, see page 604.

In dilatation the apex beat is indistinct, diffuse, and undulatory. There is an increase in the area of cardiac dulness, the outline being nearly square. The cardiac sounds are feeble, and murmurs previously present may be lost. The heart's action is irregular, and the pulse small and weak.

Mitral stenosis.—This is apt to occur from repeated attacks of subacute rheumatism, with a slowly progressing endocarditis. It is usually associated with mitral regurgitation. With this lesion there is obstruction to the flow of blood from the left auricle into the left ventricle. It is mainly compensated for by hypertrophy of the right ventricle, but to a certain degree by hypertrophy of the left auricle. The secondary changes following the lesion are hypertrophy of the left auricle followed by dilatation, increased pressure in the pulmonary veins, followed by hypertrophy and dilatation of the right ventricle. The left ventricle is usually normal or small.

Mitral stenosis produces a presystolic murmur which is somewhat prolonged, usually rough in character, and terminates sharply with the first sound of the heart. It is loudest at or near the apex, but is audible over only a small circumscribed area. Quite as constant and important for diagnosis is the presence of a "purring thrill," which is very distinct upon palpation, and terminates sharply as the apex strikes the chest wall. The pulse of mitral stenosis is usually small. The symptoms are few, but those which are present depend chiefly upon pulmonary congestion.

Aortic stenosis.—This is not very common in early life, and rarely occurs as the only lesion, being most frequently associated with mitral insufficiency. It is sometimes a congenital lesion. Aortic stenosis is compensated for by hypertrophy of the left ventricle, which may be complete for a long period, but ultimately it is followed by dilatation of the left ventricle, with mitral insufficiency and its consequences. In aortic stenosis there is an interference with the outflow of blood from the left ventricle into the aorta. It causes a systolic murmur, which is usually loudest at the right border of the sternum in the second space, and is transmitted upward, being distinct in the carotids. The second sound is generally weak. There are associated the signs of marked hypertrophy of the left ventricle.

Aortic obstruction is more frequently confounded with conditions giving accidental or functional murmurs than is any other valvular lesion. Without the signs of hypertrophy of the left ventricle, a positive diagnosis should not be made. On account of the almost perfect compensation, this form of the disease causes fewer symptoms than any other variety, possibly excepting mitral obstruction. The danger of embolism is somewhat greater than in mitral disease.

Aortic insufficiency.—This is one of the rarest valvular lesions in children. In no case on my list did it occur as the only lesion. It causes a regurgitation of blood from the aorta into the left ventricle during diastole. It is compensated for by dilatation and hypertrophy of the left ventricle. The order in which the secondary changes take place is: dilatation followed by hypertrophy of the left ventricle, ultimately followed by further dilatation due to degeneration, this leading to mitral insufficiency with all its remote consequences. The signs of aortic insufficiency are a prolonged diastolic murmur, with, or taking the place of, the second sound of the heart, generally loudest at the left border of the sternum in the second space, and transmitted downward to the apex of the heart or the ensiform cartilage. This is invariably accompanied by signs of hypertrophy and dilatation of the left ventricle, which are usually marked. In the stage of compensation the signs of hypertrophy predominate, and when compensation has failed, the signs of dilatation. A characteristic symptom is the intense throbbing of the carotids, with the sudden distension and complete collapse of their walls, and the "ball-pulse" of Corrigan. Early in the disease there may be headache, flashes of light before the eyes, and other evidences of cerebral congestion. In the late stages there may be fainting attacks. With this lesion compensation may be complete for a long time.

Tricuspid insufficiency.—This is usually secondary to disease of the left side of the heart, occurring in its late stages. It most frequently follows mitral insufficiency, where it is usually due to dilatation of the right ventricle without changes in the valves. It may be secondary to certain diseases of the lungs, such as emphysema, chronic interstitial pneumonia, or chronic pleurisy, and it may be due to congenital malformation. Tricuspid insufficiency gives a systolic murmur, loudest over the lower part of the sternum, but heard usually over a small area. It is generally associated with signs of dilatation of the right ventricle. The jugular veins stand out prominently, and often show systolic pulsation, especially upon the right side. The symptoms associated with tricuspid regurgitation are due to general systemic venous obstruction, already mentioned in connection with mitral insufficiency.

Tricuspid stenosis, pulmonic stenosis, and pulmonic insufficiency are practically unknown in childhood, except in congenital cardiac disease.

Prognosis of Valvular Disease.—Complete recovery from valvular disease is possible only when the lesions are very slight. Few children die from cardiac disease before reaching the age of fourteen years, sudden death being extremely rare. A large proportion of the cases do fairly well up to about the time of puberty, when they begin to lose ground, often failing rapidly. Others do well until a fresh endocarditis is lighted up by an intercurrent attack of rheumatism, after which the disease may make rapid progress. The proportion of children who have serious cardiac lesions before the age of eight years, and reach adult life in good condition is comparatively small.

There are several features of cardiac disease in children, in consequence of which, serious lesions tend to progress more rapidly than in adults. The muscular walls are less resistant, and hence rapid dilatation occurs much more readily than in adult life. The heart must provide not only for constant needs, but for the growth of the body. If the patient's general nutrition is poor during the period of most rapid growth, this tells quickly and seriously upon the heart, and dilatation makes rapid progress; but if the general nutrition continues good the heart may do more than hold its own throughout childhood. The demands made upon the heart at puberty are especially severe, by reason of the rapid growth of the body and the frequency of anæmia and malnutrition. There is always present the danger of rapid advances in the disease from intercurrent attacks of rheumatism, from which children are more likely to suffer than are older subjects. Extensive pericardial adhesions are frequent, and seriously handicap the heart, greatly increasing the tendency to dilatation. The effect upon the heart of poor food, unhygienic surroundings, and general malnutrition is much more marked than in adults.

These unfavourable conditions are in part offset by others in which the child has an advantage over the adult. Disease of the coronary arteries is very rare, and the valvular lesions which are most frequently met with—mitral insufficiency and aortic obstruction—are those which admit of the most complete compensation.

In making a prognosis in any given case, the amount of hypertrophy or dilatation which exists is of much more importance than the location or the special character of the murmur. The condition of the arterial and venous circulation must also be taken into consideration; also how rapidly the disease is progressing, the condition of the patient's general health, and how well circumstances will admit of proper hygienic and general management. The presence of valvular disease in childhood increases the danger from every acute disease, especially pertussis, diphtheria, pneumonia, and scarlet fever.

Diagnosis.—Valvular disease is to be particularly distinguished from conditions in which there are heard functional or accidental murmurs. According to my own experience the latter are quite common even in young children. Mistakes usually arise from attaching too much importance to the presence of murmurs, and too little to the changes in the walls and cavities of the heart, with which valvular disease is almost invariably associated. It is not always possible to decide whether a murmur is organic or functional until the patient has been for some time under observation and treatment, particularly when anæmia is present. The diagnostic points, so far as the murmurs are concerned, are mentioned in connection with anæmic murmurs.

Treatment.—A child who is the subject of a serious chronic valvular disease should be constantly under a physician's observation. Irreparable harm often results from wilful, but more frequently from ignorant, disregard of the simplest and most important rules of life for these patients. At the very least the patient should be carefully examined three or four times each year, in order that the physician may note the progress of the disease, and be able to modify the child's occupation, exercise, and surroundings so as to meet, as far as possible, the changing conditions.

Several distinct conditions may be present which call for quite different management. The essential points may be stated in a few words: for all recent cases and during all exacerbations, rest, complete and prolonged; for deformed valves with good heart walls and perfect compensation, fresh air, moderate exercise, and general tonics; for feeble heart walls, failing compensation and dilatation, rest and specific heart tonics.

During the stage of compensation, treatment directed especially to the heart is rarely necessary. The main purpose should be to maintain the patient's general nutrition at the highest possible point during the period of active growth. To this end, diet, sleep, study, and exercise should receive the most careful attention. If malnutrition and anæmia are allowed to go on unchecked until they become severe, the cardiac disease may make rapid strides, and as much harm be done in a few months as otherwise might not occur in years. The question of exercise and recreation is always a difficult one to settle. Often too little latitude is given, and the heart, like the voluntary muscles, loses its tone. Every form of exercise requiring a prolonged severe strain should be forbidden, particularly swimming and competitive games, like ball and tennis, and others requiring much running; but skating, rowing, mountain-climbing, horseback exercise, gymnastics, and even cycling on the level—all in moderation—may be allowed not only without harm, but with the greatest benefit; but any of these, used immoderately, may be productive of great injury. All exercise should be taken with regularity and system, the amount being carefully measured by the child's condition. If the patient is a boy who must earn his own living, the physician should see to it that the occupation chosen is not one likely to make special demands upon the heart.

Special watchfulness is required at the time of puberty to prevent overpressure in schools, and the development of anæmia or chlorosis. The first symptoms of these conditions should be treated energetically, and if the heart seems to be overtaxed the child should be put to bed. Patients should be so far as possible removed from conditions likely to induce fresh attacks of rheumatism. To this end, if possible, they should spend the winter and spring months in a warm, dry climate.

In the stage of failing compensation, the same general conditions are present as in adults, and they are to be managed in pretty much the same way. When such symptoms are first seen, prolonged rest in bed should be insisted upon as the thing most likely to restore the normal conditions. Cardiac dropsy with low arterial tension and weak pulse, calls for digitalis. An overloaded venous circulation may be relieved by diuretics, or, better, by saline purgatives. Iron and tonics generally are indicated, particularly strychnine and cod-liver oil. In cases of sudden heart failure, nitroglycerin, ether, and ammonia are as valuable as in adults; but better, probably, than any of these is the use of strychnine hypodermically.

MYOCARDITIS.

Disease of the muscular wall of the heart is rare in children, and of comparatively little importance, except in connection with the acute infectious diseases. Myocarditis may, however, occur at any age, even in fœtal life. As seen in children, it is almost invariably a secondary lesion, usually the result of some infectious process. The two diseases which furnish most of the cases are scarlet fever and diphtheria. The most important local cause is pericarditis with adhesions.

Lesions.—In extra-uterine life, myocarditis, as a rule, affects the wall of the left ventricle, the papillary muscles, or the septum. The heart is pale or of a yellowish-white colour, very soft and flabby, and there is frequently dilatation of the cavities. Small ecchymoses may be seen beneath the pericardium.

Two varieties of myocarditis are described: In the parenchymatous form there is a degeneration of the muscle fibre which, according to Romberg, is most frequently albuminous, next fatty, and least frequently hyaline. There is a loss of the transverse striations, and there may be complete disintegration of the fibres. This process may be circumscribed, but it is usually diffuse. In the interstitial form the lesion usually occurs in small, circumscribed areas. There is an infiltration of round cells between the muscular fibres of the heart. The process, when acute, may result in absorption or in the production of small abscesses. There may also be congestion and minute blood extravasations. In chronic cases it may lead to the formation of larger or smaller areas of dense connective tissue resembling cicatrices, in the heart wall. Either the interstitial or the parenchymatous form may occur alone, but in most of the acute cases they are combined. In addition, there is usually some degree of mural endocarditis and inflammation of the pericardium next to the heart wall. Dilatation frequently follows; rarely abscesses may form, which may open into the heart or into the pericardium. Cardiac aneurism, and even rupture, have been known to occur in a child of six years (Hadden's case).

Symptoms.—These are very rarely sufficiently marked to enable one to make a positive diagnosis. In many cases in which advanced lesions have been found at autopsy there have been no symptoms during life, and in others none until the occurrence of sudden death. This is usually from cardiac paralysis, rarely from rupture. In eight cases studied by Romberg, which occurred in the course of diphtheria, not one had cardiac symptoms during life and two died suddenly. When symptoms are present, they are generally those of feeble heart action—a faint apex impulse, a slow, weak pulse of irregular rhythm, pallor, dyspnœa, and attacks of syncope. In the late stages there may be the physical signs of dilatation, with dropsy of the feet or the serous cavities, and scanty urine, sometimes containing albumin.

Diagnosis.—A positive diagnosis of myocarditis is impossible. It may be suspected in the course of diphtheria, scarlet or typhoid fever, when cardiac symptoms like those mentioned occur, and when pericarditis and endocarditis can be excluded by the physical examination.

Treatment.—This is mainly symptomatic. After severe attacks of those infectious diseases in which myocarditis is liable to occur, and at any time when it is suspected, patients should be kept recumbent for several weeks, and special care exercised to prevent any sudden exertion, as death has occurred from so slight a thing as suddenly sitting up in bed. Iron, alcohol, and tonics should be given, the best of all of these being strychnine. Digitalis should be used with caution, and never in large doses. In some cases with symptoms indicating imminent heart failure, more striking benefit follows the use of morphine hypodermically than any other plan of treatment.

ANÆMIC MURMURS.

As already stated, anæmic murmurs are not rare even in infancy. They may be confounded with organic murmurs, either from congenital malformations or acquired disease. I have several times found the heart normal at autopsy in cases where a diagnosis of congenital disease had been unhesitatingly made during life, the murmur having been of anæmic origin. In any anæmic infant, as well as older child, one should hesitate to make a diagnosis either of congenital or acquired organic disease, from the mere presence of a murmur.

An anæmic murmur is usually systolic, generally but not always loudest at the base of the heart, audible in the carotids, often in the subclavians, and occasionally over any large artery. The murmur varies from day to day, and sometimes it is altered by changing the position of the patient. It may be loud enough to be heard over a great part of the chest in front, and even behind. There is frequently present a venous hum in the neck. There are no signs of hypertrophy, nor is there the accentuated second sound so characteristic of mitral disease. The pulse is not usually strong. Anæmic murmurs diminish in intensity and ultimately disappear with improvement in the general condition of the patient. In some cases one must wait for the effects of treatment before giving a positive opinion.

FUNCTIONAL DISORDERS OF THE HEART.

Disturbances in the heart's action unconnected with organic disease, are rare in infants and young children; but after the seventh year they are not uncommon, becoming in fact quite frequent as puberty approaches. One of the most important causes is indigestion; another is overpressure in schools, or anything else leading to nervous exhaustion. In these circumstances it is usually associated with other mental or psychical disturbances. An important predisposing cause is the demand made upon the heart by the rapid growth of the body about the time of puberty, particularly when this is associated with anæmia. In some of the cases there is a definite exciting cause, such as fright or great excitement, and it may be due to the excessive use of tea, coffee, or tobacco, especially in the form of cigarette-smoking. In a few instances it has been traced to masturbation. It may follow any acute disease, such as typhoid fever, malaria, or one of the exanthemata, and occasionally it occurs in the course of these diseases, or with bronchitis or pneumonia.

Symptoms.—The usual manifestations are attacks of palpitation; less frequently there is tachycardia (rapid heart) or bradycardia (slow heart). The majority of children complain more with functional disturbances than with organic disease, certainly while the latter is accompanied by compensation. Attacks of palpitation occur in paroxysms. In the severe form there is usually a sense of oppression in the region of the heart, with some dyspnœa, or even orthopnœa. The pulse is usually rapid, from 120 to 130, and is irregular both as to force and rhythm. The carotids pulsate strongly. The apex impulse is felt over an increased area, the heart sounds are usually strong but irregular, and sometimes a slight murmur is heard. The face is pale or flushed. There may be headache, vertigo, spots before the eyes, and noises in the ears. Sometimes there is slight cyanosis with cold hands and feet, and general perspiration. The frequency of these attacks depends upon the nature of the exciting cause. Their duration is from a few minutes to several hours.

Diagnosis.—Functional disorders are differentiated from organic cardiac disease only by careful and repeated examinations of the heart. In the diagnosis of functional disturbance especial importance is to be attached to a neurotic or neurasthenic condition of the patient, to the presence of some adequate exciting cause, the absence of evidence of enlargement of the heart, and the fact that the pulmonic second sound is not increased.

Prognosis.—This in most cases is favourable, for with improvement in the patient's general condition, with the growth of the body, and in girls with the establishment of menstruation, the attacks usually disappear.

Treatment.—During the attacks, digitalis in moderate doses should be given, also bromides or valerian. The curative treatment is to be directed toward the cause. Where no special cause can be discovered a general tonic plan of treatment should be adopted, with careful regulation of the patient's diet, exercise, and mode of life. All stimulating food, tea, coffee, and tobacco should be prohibited. Anæmia should receive its appropriate remedies. The hours of sleep and study, and the amount and character of exercise allowed, should be carefully regulated. Between attacks no treatment of the heart is necessary.

DISEASES OF THE BLOOD-VESSELS.

Abnormally Small Arteries (*Arterial hypoplasia*).—This condition is not a very common one, but it has attracted a good deal of attention, having been studied especially by Virchow. The only thing which is abnormal in the circulatory system may be that the aorta, and sometimes all the large vessels are only two thirds or three fourths their usual calibre, or even less. This may interfere seriously with the growth and development of the body, especially of the genital organs, although this result is not a constant one. The condition is found occasionally in cases of chlorosis, and in the congenital cases it may be the chief cause. There is usually associated a certain amount of hypertrophy of the heart. The other symptoms are anæmia, and sometimes an imperfect development of the body. A positive diagnosis during life is impossible.

Aneurism and Atheroma.—In early life chronic disease of the blood-vessels is exceedingly rare, yet a sufficient number of observations have been recorded to show that even young children are not exempt from this form of disease. There had been reported up to 1890 twenty-eight cases of aneurism in patients under twenty years of age (Jacobi).* Of these, however, only twelve were under fourteen years. Sanné † records the youngest case, which occurred in a fœtus born at about the eighth month, in whose body there was found a large aneurism of the abdominal aorta just below the origin of the renal arteries. Of the eleven remaining cases occurring in children under fourteen years, in over one half the number the arch of the aorta was the part affected. In one case the seat was the femoral artery, in another the external iliac, and in still another the abdominal aorta.

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* A. Jacobi, Archives of Pædiatrics, vol. vii, p. 161.

† Sanné, Revue Mensuelle des Maladies des l'Enfance, vol. v, p. 56. In these articles will be found references to most of the reported cases.

Probably the most important etiological factor, as in adult life, is syphilis, but in only a few of the cases reported was the evidence of syphilis conclusive. In two cases there was general tuberculosis. In addition to these general causes, aneurism may be due to some local condition, such as an erosion from bone, an abscess in the neighbourhood, or to embolism. The symptoms and course of aneurism in young children do not differ essentially from those of the disease as seen in adults.

In addition to the cases of aneurism referred to above, I have found reports of seven cases of atheroma in very young subjects. In Sanné's case the patient was but two years old, and patches of atheromatous degeneration were found in several places in the aorta. In Hawkins's case, eleven years old, there was found extensive atheromatous disease of the aorta, subclavian and carotid arteries. In Filatoff's case, atheromatous degeneration affected the arteries at the base of the brain, causing death from cerebral hæmorrhage. It is interesting to note that in this patient, who was only eleven years old, there was also present chronic diffuse nephritis with contracted kidneys. A similar condition of the kidneys and arteries was observed by Dickinson in a girl of six years.

Embolism and Thrombosis.—Embolism has already been referred to in connection with acute endocarditis. It may be seen at any age, even in infancy, but generally occurs in patients over five years old. The emboli are usually swept into the circulation from vegetations upon the valves of the heart. The symptoms which they produce will depend upon the nature of the emboli and the vessels occluded by them. If they lodge in the brain they may cause paralysis or convulsions; if in the spleen, pain and swelling of this organ; if in the kidneys, pain, tenderness, and sometimes hæmaturia; if in the lungs, cough, sometimes accompanied by hæmoptysis and occasionally by a sharp thoracic pain. If the emboli are infectious, they may give rise to abscesses. The pathological results following embolism are similar to those which are seen in adults.

The most frequent form of thrombosis, that occurring in the sinuses of the brain, is discussed in connection with Diseases of the Nervous System. Cardiac thrombi, especially of the right side of the heart, are not infrequently found in patients dying from heart disease, pneumonia, and occasionally also from other acute inflammatory processes and acute infectious diseases, particularly diphtheria. These thrombi are in most cases produced during the last few hours of life, or just at the time of death, and are of no clinical importance. They frequently extend from the heart into the large blood-vessels, particularly the pulmonary artery. Thrombosis occasionally occurs in all the large vascular trunks in childhood as well as in adult life.

Thrombosis of the internal jugular vein.—Pasteur * reports a case in a child two and a half years old, in which the middle of the vein was filled with an organized thrombus, and the lower portion obliterated and reduced to a fibrous cord. The symptoms were swelling, œdema, and cyanosis of the face, and dilatation of the facial vein, but not of the external jugular. There were clubbing of the fingers and œdema of the feet, but not of the arm. The heart was found to be dilated and hypertrophied, but was not the seat of valvular disease. The symptoms had existed since an attack of pneumonia, eighteen months before death.

Thrombosis of the vena cava.—Quite a number of cases are on record where this has occurred as the result of pressure from large abdominal tumours; it has followed new growths of the kidney and large masses of tuberculous lymph nodes. Neurutter and Salmon have recorded a case of thrombosis, apparently of marantic origin, in a child seven years old. The thrombus filled the vena cava, and extended to the origin of the hepatic veins and into both femorals. Death occurred from tuberculosis. In Scudder's case (seventeen years old) there was apparently obliteration (probably congenital) of the inferior vena cava; there was an extensive varicose condition of all the abdominal veins. The symptoms of thrombosis of the vena cava are swelling and œdema of the feet—sometimes of the abdominal walls and the groin—and very great dilatation of the superficial abdominal veins.

Thrombosis of the aorta.—A case has been reported by Leopold in a newly-born child which was delivered by version. The thrombus was of recent origin, and filled the lower aorta, extending into the femoral artery. A case of thrombosis of the aorta occurring in a girl of thirteen years has been reported by Wallis. The aorta was very narrow, and probably the seat of syphilitic disease. The thrombus extended from the origin of the renal arteries to the cœliac axis.

Thrombosis in infectious diseases.—There is occasionally seen in typhoid fever, but more frequently in diphtheria, thrombosis of some of the large venous trunks, usually of one of the lower extremities. The symptoms are pain, localized swelling, and partial paralysis. If the artery is affected, there may be gangrene.

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* Lancet, February 11, 1888.

SECTION VI.

DISEASES OF THE URO-GENITAL SYSTEM.

CHAPTER I.

THE URINE IN INFANCY AND CHILDHOOD.

WHILE a study of the urine is of much less importance in early life than of the symptoms referable either to the digestive or respiratory system, it is deserving of much more attention than it has generally received. In infancy especially it is attended with difficulty, owing to the fact that it is by no means an easy matter to secure specimens for examination.

Methods of Collecting Urine.—In male infants this may be done by placing the penis in the neck of a small bottle which lies between the thighs and is secured in position by pieces of tape passing over the hips and beneath the perinæum. A still better plan is to use in the place of a bottle a condom large enough to include both the scrotum and penis. The urine of female infants can sometimes be collected in a similar way by placing a small cup over the vulva and holding it in place by the napkin. A plan nearly always successful is to put the infant upon a chamber after a long sleep. It should be done on the instant of waking, or the child may be wakened for the purpose. A cold hand over the bladder facilitates matters. A small amount, sufficient to test for albumin, may often be obtained by placing absorbent cotton over the vulva or penis. The most certain of all means, however, is catheterization; in females sometimes nothing else will answer the purpose. A soft rubber catheter, size 6 or 7, American scale (9 or 11 French), should be used for infants.

Daily Quantity.—This is relatively much larger in infants than in older children and in adults, on account of the more active metabolism of the young child and the large amount of water taken with the food. The quantity fluctuates widely from day to day according to the amount of fluid food taken and the activity of the skin and bowels. The following figures are the averages obtained by combining the results of the investigations of Schabanowa, Cruse, Camerer, Pollak, Martin-Ruge, Berti, Schiff, and Herter:

Average Daily Quantity of Urine in Health.

AGE.	Grammes.	Ounces.
First twenty-four hours	0 to 60	0 to 2
Second twenty-four hours	10 " 90	⅓ " 3
Three to six days	90 " 250	3 " 8
Seven days to two months	150 " 400	5 " 13
Two to six months	210 " 500	7 " 16
Six months to two years	250 " 600	8 " 20
Two to five years	500 " 800	16 " 26
Five to eight years	600 " 1,200	20 " 40
Eight to fourteen years	1,000 " 1,500	32 " 48

Frequency of Micturition.—This is greatest in young infants, and diminishes steadily as age advances. In the first two years, during the waking hours, the urine is generally passed as often as twice an hour, while during sleep it is retained from two to six hours. By the third year the urine may be held during sleep for eight or nine hours, and at other times for two or three hours. Such control of the sphincter of the bladder is often obtained at two years, and sometimes even at an earlier period. From slight nervous disturbances or minor ailments of any kind, this control is impaired, and the water may be passed by children of four or five years with the frequency seen in infants.

Physical Characters.—The urine of the newly born is usually highly coloured. During later infancy it is pale and frequently turbid, even when practically normal, owing to the presence of mucus; this turbidity often no amount of filtration will entirely remove. Less frequently turbidity depends upon urates. The urine of the first few days of life often shows a deposit of urates or uric acid in the form of a reddish-yellow stain upon the napkin. The reaction of the urine at this time is usually strongly acid, but throughout the rest of infancy it is faintly acid or neutral.

The specific gravity is higher during the first two days than at any time in infancy on account of the scanty supply of fluid taken; it is usually lowest from the third to the sixth day, but from this time it rises steadily until puberty is reached. The specific gravity will of course vary with the quantity. From the writers already referred to the following figures are taken:

	Specific gravity.
First to third day	1·010 to 1·012
Fourth to tenth day	1·004 " 1·008
Tenth day to sixth month	1·004 " 1·010
Six months to two years	1·006 " 1·012
Two to eight years	1·008 " 1·016
Eight to fourteen years	1·012 " 1·020

Microscopically, the urine of the newly born shows the presence of many squamous epithelial cells, mucus, granular matter, and crystals of uric acid and amorphous or crystalline urates. It is not uncommon to find hyaline and even granular casts. Martin-Ruge found hyaline casts in the urine of fourteen out of twenty-four healthy nursing infants examined during the first week. Granular casts were much less frequent. The microscopical appearances of the normal urine of later infancy and childhood present no peculiarities.

Composition.—*Urea.*—The following figures show the average daily quantity of urea eliminated at the different ages:

Age.	Daily quantity of urea.
First day	0·076 to 0·114 gramme.
Second to seventh day	0·140 " 0·660 "
One to two months	0·90 " 1·40 "
Three to five years	13·09 " 14·01 grammes.
Five to thirteen years	16·05 " 21·03 "

Uric acid.—Few observations have been made upon the elimination of uric acid, but all authorities agree that it is much higher in the newly born than at any subsequent period of life. The quantity is better appreciated by giving the ratio between the uric acid and urea than by the absolute quantity of the former. The figures here given for the newly born are taken from Martin-Ruge; the others are from Herter.

Ratio of Uric Acid to Urea.

In the newly born	1 to 14
Under one year	1 " 60–80
From two to five years	1 " 50–70
From five to fifteen years	1 " 45–60

The inorganic salts (phosphates, chlorides, sulphates) are all present in the urine of the newly born, but in relatively small quantities, increasing as age advances. The colouring matters are also less abundant.

Albumin is often present in the urine during the first days, but usually in small amount. Cruse found it twenty-eight times in ninety observations upon healthy infants; usually the quantity was small, amounting to traces only, but in two cases it was quite large upon the second day. These observations are confirmed by the investigations of Martin-Ruge, and also of Pollak.

Sugar is frequently found in the urine of healthy infants during the first two months. This subject is referred to later under the head of Glycosuria.

FUNCTIONAL OR CYCLIC ALBUMINURIA.

Etiology.—This condition, although a rare one in young children, is occasionally seen between the ages of ten and sixteen years. I shall not in this connection include cases sometimes classed as febrile albuminuria, in which there is usually present the condition described as acute degeneration of the kidneys.

The causes of functional or physiological albuminuria, and the circumstances in which it has been observed, are many and varied. It is much more common in males than in females. In many patients it is regularly cyclic in character, albumin being absent in the urine passed during the night or early morning, but present during the day, diminishing in the evening and absent at bed-time. In a case reported by Tiemann, the morning urine showed no trace of albumin in seventy-eight of eighty-four examinations. At noon albumin was present in ninety-eight of one hundred and thirteen examinations. In certain cases albuminuria is distinctly traceable to cold bathing; in others, to fatigue following excessive muscular exercise; in still others, to dyspeptic conditions. It may be associated with a diet rich in nitrogenous food. Sometimes none of these conditions exist, and there is simply the occasional presence of albumin in the urine.

Many theories have been advanced in explanation of cyclic albuminuria. Sometimes it appears to be clearly traceable to irritation of the kidney by uric acid, urates, or oxalates. Kinnicutt believes this to be one of the prominent causes, and that albuminuria is due to vaso-motor disturbances in the kidney. Delafield compares the exudation of serum from the vessels of the kidney to the dropsy of the feet seen in anæmia. Da Costa believes that it always depends upon slight changes of an evanescent character in the kidney.

Symptoms.—Many of the patients exhibiting cyclic or periodical albuminuria are well nourished, and have no other signs of disease; others show dyspeptic symptoms, and are anæmic and poorly nourished, suffering from headaches and other neuroses. In the cases distinctly periodical the amount of albumin is commonly small. It is not infrequently associated with temporary glycosuria. As a rule, casts are absent, although it is not uncommon to find a few hyaline casts, and occasionally granular casts are also present. A gouty family history exists in a certain proportion of the cases, and some of the patients themselves present other evidences of this diathesis.

Diagnosis.—Pavy mentions the following points as characteristic of physiological or functional albuminuria: (1) The time of its occurrence. The absence of albumin early in the morning, its presence in the forenoon, and diminution toward evening. When this is repeated day after day the diagnosis is, he believes, quite positive. (2) The absence of serious impairment of the general health and of the characteristic symptoms of nephritis, such as dropsy, cardiac hypertrophy, a pulse of high tension, retinal changes, etc. (3) The fact that casts are, as a rule, absent. (4) That crystals of oxalate of lime are present, and the urine is of high specific gravity.

Too much stress is certainly laid by Pavy and many other writers upon the fact that the albumin is found in the urine only at certain times in the day. This is not peculiar to functional albuminuria, as the same thing occurs in many cases of chronic nephritis, especially in the early stages when the amount of albumin present is small. All these cases must be carefully watched for a long time and many observations made, before nephritis can positively be excluded.

Prognosis.—The prognosis in purely functional albuminuria is good. But many patients who for a considerable time were thought to have only functional albuminuria have ultimately developed nephritis. A favourable prognosis is therefore possible only after long observation.

Treatment.—This is to be directed toward the patient's general condition. Dyspeptic symptoms must be relieved, the patient's mode of life regulated, only moderate exercise allowed, and a simple diet given. If the urine is of high specific gravity, and contains oxalate-of-lime crystals, alkalies and mineral waters should be given in addition. Iron is indicated if there is anæmia.

HÆMATURIA.

Hæmaturia is characterized by the presence of red blood-cells in the urine, and is to be distinguished from hæmoglobinuria where only blood pigment is present.

Hæmaturia may result from local or general causes. In infancy it may be due to new growths of the kidney. Such hæmorrhages, though rare, may be abundant, and may be seen early. Hæmaturia may occur also as a symptom of acute nephritis, especially that complicating scarlet fever, or it may result from the irritation of a calculus in the kidney, the ureter, or the bladder. In rare instances its cause is a new growth of the bladder, and it may be due to traumatism. It may sometimes be produced by the irritation of a highly concentrated urine, owing to the fact that too little fluid is taken. I saw a marked example of this in an infant eight months old, where no other explanation could be found. I once saw hæmaturia following uric-acid infarctions in the newly born. It may also occur at this time as one of the symptoms of sepsis. Among the general causes the most important are: the hæmorrhagic disease of the newly born; the blood dyscrasiæ, such as scurvy, purpura, and hæmophilia; and infectious diseases, particularly malaria, typhoid, variola, scarlet fever, and influenza. In most of these cases the amount of blood passed is small. When it is large it may appear in the urine as clear blood, or as clots, or it may impart simply a reddish or smoky colour to the urine. The colour, however, is not so reliable as a microscopical examination. For a simple chemical test guaiacum may be used.

Large hæmorrhages are much more likely to come from the kidneys than from the bladder. The presence of blood casts from the renal tubules, or larger ones from the ureter, are conclusive evidence of the renal origin of the hæmorrhage.

In children, renal hæmorrhage in itself rarely requires treatment; when it does, the same remedies are indicated as in the adult, viz., ergot, gallic acid, and rest in bed. Some obstinate cases have been cured by drinking water from alum springs.

HÆMOGLOBINURIA.

In this condition blood pigment appears in the urine in large quantity, but red blood-cells are very few in number, or are absent altogether. In severe cases the urine may be almost black. There is commonly a small amount of albumin. This condition may be recognised by the appearance of granules of pigment under the microscope, or by Heller's test; the most conclusive means of diagnosis, however, is the spectroscope.

Epidemic hæmoglobinuria (Winckel's disease) has already been described in the chapter on Diseases of the Newly Born. Hæmoglobinuria may be due to certain poisons, as carbolic acid or chlorate of potash, or to certain infectious diseases, as scarlet fever, typhoid fever, malaria, syphilis, and erysipelas.

Paroxysmal hæmoglobinuria occurs in childhood, although it is an exceedingly rare condition. A typical case in a child of four and a half years has been reported by Mackenzie. This was a delicate child of syphilitic parents; the hæmoglobinuria was preceded by fever and chills, without any other evidence of the presence of malaria.

The exact pathology of hæmoglobinuria is at present unknown, and its treatment is very unsatisfactory.

GLYCOSURIA.

By this term is understood the occasional or transient appearance of sugar in the urine. This is not very infrequent in children, and may be met with even during the first month of life. Grósz has published some careful investigations upon the glycosuria of early infancy.* He made many observations upon fifty infants during the first month of life, from which the following conclusions were drawn: Glycosuria is not uncommon in nursing infants; but it is not seen in nursing infants who are perfectly healthy. It occurs particularly with certain disturbances of digestion, whether functional or inflammatory. The sugar found in the urine under these conditions reacts strongly to the reduction test (Fehling's), but not to the fermentation test; sometimes the polariscope shows that it has the power of dextro-rotation. This is believed to be milk sugar, or one of its derivatives. It is not of constant or regular occurrence. It may be produced artificially by increasing the amount of milk sugar above that which can be normally absorbed. This quantity Grósz places at 3·3 grammes for each kilogramme of the body weight. If more than this is given, or if there is diminished capacity for the absorption of sugar, glycosuria occurs.

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* Jahrbuch für Kinderheilkunde, Bd. xxxiv, p. 83.

Koplik has made some observations upon the urine of patients fed chiefly upon infant foods composed largely of sugar. He found sugar in five out of ten cases examined; in three, the sugar responded both to Fehling's and the fermentation test; in two cases to Fehling's test only.

There seems to be no doubt regarding the existence of dietetic glycosuria in infants and in older children. Repeated examinations of the urine are, however, necessary in order to exclude more serious disease.

PYURIA.

Pus in the urine may exist as an acute or a chronic condition. In either case, in a child, it is much more likely to come from the pelvis of the kidney than from any other source. It may, however, come from any part of the genito-urinary tract—the kidney or its pelvis, the ureters, the bladder, the urethra, or the vagina. Sometimes it comes from an outside source, as when an abscess from perinephritis, appendicitis, or caries of the spine opens into the urinary tract.

Coming from the pelvis of the kidney, pus may indicate, if the condition is an acute one, pyelitis, pyelo-nephritis, or pyonephrosis; if it is chronic, it points to renal tuberculosis or calculus. The amount of pus in any of these conditions may be quite large. The urine is turbid and usually acid in reaction. It contains many epithelial cells of the transitional variety. A urine containing much pus is always albuminous. A turbidity due to pus may be mistaken for an excessive deposit of urates; they are distinguished by the microscope and by the fact that urates clear up on heating. It is rare that pus comes from the ureters except in connection with congenital malformations or the impaction of calculi. Pus from the bladder is not usually in large quantity, and may be mixed with mucus. The urine may be alkaline or acid in reaction; there may be associated the symptoms of vesical irritation or of cystitis. Pus from the lower genital tract is rare in children, and its causes may often be recognised by a local examination. When the cause of pyuria is the opening of an abscess into the urinary tract there is generally a sudden appearance of pus in large amount. The pyuria is in most cases of short duration, possibly only a few days, and it may disappear quite rapidly.

The treatment of pyuria depends altogether upon its cause. Improvement in the symptoms nearly always follows the use of urotropin, which may be given in doses of from two to five grains three times a day to a child of five years.

LITHURIA.

Lithuria is a condition in which there is an excessive elimination in the urine of uric acid or of urates. The amount of nitrogen compounds eliminated by the kidneys as uric acid and urea, varies much from day to day with the nature of the food and other conditions. Hence in estimating an excess of uric acid, the absolute quantity eliminated in twenty-four hours is much less significant than the ratio of the uric acid to the urea (page 644). Whenever this ratio is continuously disturbed, the excretion of uric acid may be considered abnormal, except, of course, in grave pathological conditions of the kidney, where there is an insufficient elimination of urea. Regarding the source of uric acid, the theory of Horbaczewski is that most widely accepted, viz., that it results from the destruction of the nuclein of the cells of the body, particularly of the white blood-cells.

For accurate knowledge as to the amount of uric acid eliminated, nothing short of a quantitative chemical analysis can be depended upon. But if amorphous urates are deposited in large amount, uric acid may be considered excessive if the specific gravity is not high (above 1.025). If the specific gravity is high, the precipitation may be explained simply by the concentration of the urine. The deposition of the crystals of uric acid, forming the familiar brick-dust deposit, is not in itself evidence of excessive elimination. For a quantitative clinical test, that of Haycroft is probably the best.*

Lithuria is not a specific condition, but rather a very general symptom associated with many kinds of disturbances of nutrition. It may be found in anæmia, malnutrition, chorea, rheumatism, chronic dyspepsia, and in a great variety of other disorders. Regarding the significance of lithuria, thus much may be positively asserted: The excessive elimination of uric acid when continuous is always evidence of a serious disturbance of nutrition. The gravity of the condition will depend upon the degree of this excess and upon its duration.

The treatment of lithuria is the treatment of the condition upon which it depends. The essential pathological condition is not so much excessive elimination as excessive production.

Urine containing Crystals of Uric Acid in the Form of Brick-Dust Deposit.—This condition is not to be confounded with the one just described. As already stated, such precipitation is not to be taken as evidence of an excess of uric acid, and, in fact, in most of these cases there is no excess. The condition is rather one in which the solvent power of the urine for uric acid is much reduced. Such urine, as a rule, is high-coloured, strongly acid, and may have a high specific gravity.

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* See Haig on Uric Acid in Health and Disease.

This condition also is dependent upon a disturbance of nutrition, and one which is most frequently associated with a gouty diathesis. It is not very common in children except in those of gouty antecedents. In such patients it is only occasionally present, and is usually associated with some other disturbance of nutrition, often of digestion. It is frequently the cause of local irritation of the urinary passages, which is frequently manifested by incontinence of urine.

In my experience these cases are most improved by cutting off sugar from the diet almost entirely, by greatly reducing the amount of starchy food and substituting a diet rich in nitrogen and fat, viz., meat, milk, and cream, together with plenty of outdoor exercise. The continued use of alkaline waters is also of decided advantage in most cases.

INDICANURIA.

Indicanuria is a condition characterized by the presence of indican in the urine. To Herter is due the credit of bringing this subject prominently to the minds of the profession in this country. Indican (indoxyl-potassium sulphate) is derived from indol, which is formed in the intestine by the agency of bacteria from the excessive putrefaction of the proteids. It may also be produced in other parts of the body where putrefactive processes are going on, as in extensive suppuration without drainage, in pulmonary cavities, empyema, etc. Indican is only one of the ethereal sulphates produced in the manner above indicated, and when other conditions like those mentioned are excluded it may be taken as an index of the amount of putrefaction going on in the intestine.

The presence of indican in the urine is demonstrated by adding certain oxidizing agents, which produce an indigo-blue colour.* The existence of indicanuria in children was formerly believed to be pathognomonic of tuberculosis. Later investigations have shown that this is not the case; for in cases of tuberculosis indican is almost as frequently absent as present.

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* The commonly employed test for indican is that known as Jaffé's test. It is described by Herter as follows: Pour into a test-tube equal quantities of urine and strong hydrochloric acid so as to fill the tube to within half an inch of the top, and shake. If there is much indican, a dark blue or purple colour will be produced. Then add sufficient chloroform to completely fill the tube and shake thoroughly. It is important that the chloroform should completely fill the tube so that no air bubbles get in by the agitation. If, after standing, the chloroform assumes a deep-blue or violet colour, there is certainly an excess of indican. The reaction may not appear at first, but may come out after standing several hours, or if slight at first it may increase in intensity. Sometimes, when no reaction is obtained, it may be produced by adding one drop of a saturated solution of chloride of lime or of peroxide of hydrogen. No more than one drop should be added at a time, or the blue colour may be bleached. In alkaline urine the indican is usually destroyed, so that the test may be negative.

Herter gives the following as the conditions under which indicanuria is likely to be present: It is found in chronic intestinal indigestion; in very many cases of chronic constipation; in many cases of epilepsy, just about the time of the seizures; in some cases of masturbation; frequently in children who are the subjects of night terrors, and in whom there are usually disturbances of digestion. According to other observers, it is found with great constancy in acute putrefactive diarrhœas. With the exceptions above noted, the source of the indican is always the same, viz., the excessive putrefaction of the proteid substances in the intestine.

Indicanuria is most frequently a symptom either of acute or chronic intestinal disease. It is important as being a guide by which we may estimate the other symptoms in these conditions, and the effects of treatment. While a trace of indican is frequently present in health, a strong indican reaction is always to be considered abnormal in a child. The indications for treatment are to diminish intestinal putrefaction. This is mainly dietetic. Indicanuria is usually increased by a meat diet and diminished by a milk diet. Other measures are referred to in the treatment of chronic intestinal indigestion.

ACETONURIA—DIACETONURIA.

Acetone exists in small quantities in the urine of healthy children. According to Baginsky and Schrach, it is found in large quantities in many febrile diseases. It increases with the height of the fever and subsides with it. Acetone is probably formed from the destruction of the nitrogenous material of the body, as it is increased by a nitrogenous diet, and may disappear by a diet of carbohydrates. Baginsky found it also in children with epilepsy, sometimes during the attacks. It is not, however, believed to be the cause of the convulsive seizures, as it is absent in convulsions occurring under other conditions. There is no connection between acetonuria and the nervous symptoms accompanying fever.

Acetone and diacetic acid are regularly found in the urine of patients suffering from cyclic vomiting; they are probably a result, not the cause of the attacks. In progressing cases of diabetes and in diabetic coma both these substances are present.

Binet found *diacetic acid* in sixty-nine out of one hundred and fifty examinations in febrile diseases, chiefly in scarlet fever, measles, and pneumonia. Schrach found diacetonuria exceedingly common in cases of continuous high fever. It is more frequently present than acetonuria, and ceases with the fever.*

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* For literature, see Baginsky, Archiv für Kinderheilkunde, Bd. xi, p. 1.

ANURIA.

By this term is meant an arrest of the urinary secretion. To that form which occurs in the course of renal disease the term " suppression " is generally applied. Anuria is to be carefully distinguished from retention, from the scanty secretion which occurs whenever food is refused or withheld on account of illness, and also from that which accompanies acute diarrhœa, with large, watery discharges. Anuria is sometimes seen in the newly born, where it depends upon some malformation of the genital tract; or, more frequently, upon uric-acid infarctions in the kidneys. The first urine passed after such an attack is very often highly acid, and may contain an abundance of uric-acid crystals and larger masses visible to the naked eye. Other cases admit of no such explanation, and the condition must be regarded as of nervous origin. For the time, the secretion appears to be completely arrested, as the bladder, both by palpation and catheterization, is found to be empty. This condition is not a very uncommon one in infancy, and it may continue for from twelve to thirty-six hours. So long as infants appear to be perfectly normal in every other respect, the suspension of the urinary secretion even for twenty-four hours need excite no anxiety.

The treatment is very simple and effectual, and consists in the administration of sweet spirits of nitre, either alone or in combination with the acetate or citrate of potash, and plenty of water. To an infant of three months one minim of the nitre and one grain of the citrate of potash may be given every hour in half an ounce of water until the urinary secretion is established, which will usually be in six or eight hours. If the urine is very highly acid, and stains the napkins, the potash should be continued for several days. Hot fomentations over the kidneys may be used with advantage.

DIABETES INSIPIDUS (POLYURIA).

This is a chronic disease characterized by the excretion of a very large amount of pale urine of low specific gravity. It is invariably accompanied by polydipsia. The disease is an exceedingly rare one in children.

The exact pathology of diabetes insipidus is not known; but from the conditions under which it occurs it is believed to be a neurosis. The irritation which gives rise to it may be in or near the floor of the fourth ventricle, or it may affect the renal nerves.

Etiology.—Of eighty-five cases collected by Strauss, twenty-one were under ten years of age and nine under five years. In Roberts' collection of seventy cases, the disease began in twenty-two before ten years, and in seven during infancy. In some cases it begins soon after birth. Males are more frequently affected than females, and in certain cases heredity is an important factor. Weil has published a remarkable example of the disease existing in many members of a single family. Falls or blows upon the head, concussion of the brain, tumours of the brain, especially of the occipital region, tuberculous or cerebro-spinal meningitis or chronic hydrocephalus, all have been found associated with diabetes insipidus. It sometimes has followed the acute infectious diseases; but in many cases no cause whatever can be found.

Symptoms.—The quantity of urine is enormous, usually exceeding even that in diabetes mellitus. From five to twenty pints daily may be passed. The urine is pale, the specific gravity from 1·001 to 1·006, and it contains neither albumin nor grape sugar. In a few cases the presence of inosite (muscle sugar) has been found. Restricting the amount of fluid taken causes a very marked diminution in the amount of urine. The intense thirst leads patients to drink enormously of water and other fluids. Various contradictory statements are made by different writers regarding the quantity of uric acid and urea eliminated in these cases. The following are the results obtained in a case recently under observation in the Babies' Hospital.* The child was three years old, quite anæmic, and losing in weight. On January 20th the fluids were unrestricted, on the other days they were restricted:

DATE.	Daily quantity of urine.		Specific gravity.	Total urea.	Total uric acid.	Indican reaction.	Inosite.
	Grammes.	Ounces.		Grammes.	Grammes.		
January 20	3,300	101½	1·006	22·276	0·173	None.	None.
" 25	750	25	1·010	9·049	0·072	Strong.	None.
" 26	775	25½	1·010	6·478	......	......	None.
February 8	1,320	49	1·007	12·113	0·110	None.	None.

The elimination of urea in this case is excessive, but the uric acid is not far from the normal.

Nervous symptoms are usually present. There may be disturbed sleep from the frequent micturition, palpitation, flushing of the face and other vaso-motor disturbances, headache, restlessness, and neuralgia. There may be incontinence of urine. The skin is pale and dry, and perspiration is scanty. The general health may not be disturbed. In most cases, however, it is somewhat affected, and there may be the usual symptoms of malnutrition, and even neurasthenia. If it affects young children, their growth may be considerably retarded. The appetite usually remains quite good. The temperature is at times slightly subnormal. The course of the disease is indefinite. It is very chronic, and may last for many years, death taking place only from intercurrent affections.

Prognosis.—A few of the cases recover spontaneously. Those of short duration are often cured by treatment. Of the chronic cases in which the disease is well established very few are controlled. The prognosis is worse if there are marked disturbances of the digestive tract or organic brain disease.

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* The analyses were made by Dr. C. A. Herter.

Diagnosis.—This is easily made from the two marked symptoms, excessive thirst and polyuria. From diabetes mellitus it is easily distinguished by the lower specific gravity and the absence of sugar from the urine. In older children, chronic nephritis with contracted kidney may be confounded with it.

Treatment.—Fluids should be moderately restricted. It is a serious mistake to reduce the quantity of fluids too much, since the drinking is not the cause of the diuresis. The diet should be simple and nutritious, consisting largely of meat, with a moderate amount of carbohydrates. The general treatment should be directed to the condition of malnutrition. The clothing should be warm, and a moderate amount of exercise should be allowed. Drugs are of little use; those which have sometimes been beneficial are arsenic, belladonna, ergotine, the bromides, and antipyrine. Treatment must be continued for many months to be of any value.

CHAPTER II.

DISEASES OF THE KIDNEYS.

MALFORMATIONS AND MALPOSITIONS.

MALFORMATIONS of the kidney are not infrequent. In seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum malformations of the kidney or ureters were met with in seventeen cases. This does not represent the actual frequency with which they occur, for in about half that number of autopsies in two other institutions only a single example was seen. Adding to the cases mentioned two others seen elsewhere, there are twenty cases of renal malformation of which I have notes, classed as follows:

Fusion of the kidneys, or horseshoe kidney	4 cases.
Supernumerary ureters	4 "
Hydronephrosis (alone)	8 "
Cystic degeneration of the kidney (alone)	2 "
Hydronephrosis and cystic kidney	1 case.
Single kidney	1 "

In all malformations the left kidney is much more frequently affected than the right, the proportion being nearly two to one. Malformations are more often seen in males than in females.

Fusion of the Kidneys.—In one case, in a child who died of pneumonia at the age of three years, the kidneys were fused into one irregular ovoid mass, lying upon the lumbar vertebræ; in another case the mass lay upon the promontory of the sacrum; in both there were two renal arteries and two ureters. In the two other cases the organs were united at their lower extremities, and in both of these there were two ureters passing in front of the kidney. In one there was also hydronephrosis and chronic diffuse nephritis. The children died at the ages of four and five months respectively.

Cystic Degeneration of the Kidneys.—In two of these three cases the right kidney was affected, and in one the left. The ages at which the children died were from seven to ten months. No renal symptoms were present. In all the cases the cystic kidney was very small, about an inch and a half in length and one inch in width. The organ was entirely made up of smaller and larger cysts containing a clear fluid, held together by loose connective tissue. The ureter was small and rarely pervious throughout. In one case there was hydronephrosis of the opposite side; in the others the opposite kidney was considerably enlarged, being about one half larger than normal. In addition to these small cystic kidneys there has been described a cystic degeneration in which very large cysts have formed even *in utero*, sometimes filling the abdominal cavity of the child and seriously interfering with delivery.

Single Kidney, the other being rudimentary or absent.—Of this I have seen but one example, which was found in a young man twenty-two years of age, who died of typhus fever in Bellevue Hospital. The right kidney weighed seven and a half ounces; the left was represented by a nodular mass about the size of an ovary, showing no trace of renal tissue. The ureter was pervious to within four inches of the kidney; the suprarenal capsule was normal. Macdonald has reported a case in which there was no trace whatever of the right kidney; the left was greatly enlarged, and weighed nine ounces. There were two suprarenal capsules but only one ureter. Schaeffer has reported absence of both kidneys in a seven-months' fœtus, associated with many other malformations.

Hydronephrosis.—Of the ten cases of which I have notes, this existed as the principal deformity in eight. In two cases it was associated respectively with cystic degeneration of the opposite kidney and horseshoe kidney. In seven cases only the left side was affected; in three there was double hydronephrosis. Seven patients were males and three females. Six died before they were six months old, and only two lived to be two years old. This condition is undoubtedly the result of some obstruction to the outflow of urine in the ureter, bladder, urethra, or prepuce, but in only three of my cases could there be found an obstruction sufficient to explain the deformity. In two there was marked hypertrophy of the bladder. In no case was a calculus found as the cause of the obstruction. In most of the cases the ureter was dilated to a diameter of from one is sufficient water to dissolve it, so that the crystals are deposited in the tubes. Uric-acid infarctions are found chiefly in children dying before the end of the second week, although it is not uncommon to see them as late as the third or fourth or even the sixth month. In most of the cases, as the urinary secretion becomes more abundant, the deposits are washed out in the urine and appear as brownish red or pink stains upon the napkins. Infarctions may give rise to a slight inflammation of the renal tubules, but very rarely to any serious lesion; sometimes they remain as deposits in the calices or the pelvis of the kidney or in the bladder, forming the nucleus of a calculus. The symptoms to which they give rise are mainly scanty urination during the first week of life, and occasionally anuria for the first day or two. Sometimes there is evidence of severe pain; priapism may be present, and there is the stain upon the napkin already referred to. The treatment is to give water freely and some alkaline diuretic such as citrate of potash. One grain should be given every two hours until the secretion is fully established; this in most cases will be within twenty-four hours.

ACUTE CONGESTION OF THE KIDNEY.

In acute congestion of the kidney all its blood-vessels contain much more blood than normal, and from them there may be an escape of serum and even of the red blood-cells by diapedesis. This congestion may result from traumatism, the ingestion of certain poisons, from any of the infectious diseases, or from cold.

The urine is usually scanty, of high specific gravity, and contains albumin and red blood-cells, sometimes blood casts. This may be only a temporary condition passing off in a few days without further symptoms, or it may exist as the first stage of acute nephritis. It is most serious when it occurs in kidneys already the seat of serious disease. There are sometimes no symptoms except those of the urine; or there may be headache, pain in the back, and some general indisposition.

The treatment consists in free catharsis, the use of hot vapour baths, and counter-irritation over the kidneys by means of hot poultices or dry cups.

CHRONIC CONGESTION OF THE KIDNEY.

This results from interference with the return circulation of the kidney, and may be caused by congenital malformation or valvular disease of the heart, chronic broncho-pneumonia or chronic pleurisy; also by the pressure of any abdominal tumour upon the inferior vena cava or the renal veins.

The kidneys are generally enlarged, firmer than normal, and dark-coloured. All the capillary vessels are swollen and distended with blood, and their walls are thickened. In addition to the symptoms of the prifourth to one half of an inch, and in two it was so large as to be easily mistaken for the small intestine. Usually the ureters appeared much elongated and sacculated; the pelvis of the kidney was dilated to the capacity of half an ounce or more, the calices forming pockets about half an inch in diameter. Less frequently the greater part of the kidney was destroyed, leaving only a series of communicating pockets surrounded by a thin cortex of renal tissue from one fourth to one eighth of an inch in thickness. In five cases there was chronic diffuse nephritis of the affected side, and sometimes both kidneys were involved, even though the hydronephrosis was unilateral. The nephritis was usually of a very advanced type. In two cases, typical examples of the atrophic form (contracted kidney) were seen, one of these children dying at the age of one month.* The organs are shown in Fig. 118.

Urinary symptoms were noted in but one case, and in that they were due to pyelo-nephritis dependent upon the presence of calculi in the kidney not the seat of hydronephrosis. In no other case was the malformation suspected during life. Four patients died of marasmus, two of acute broncho-pneumonia, and one of ileo-colitis. In only one was there any malformation outside the urinary tract, this being a case of congenital heart disease.

Double hydronephrosis is generally associated with, or results in, such changes in the kidneys that the patients die during infancy. It may give rise to one or more tumours, which sometimes attain a large size. Changes in the urine may not be present until the disease is very far advanced. There may be the general and local symptoms of chronic diffuse nephritis, or, when infection of the genital tract occurs, there are added the symptoms of pyelitis. In the great majority of cases the condition is unrecognised, the patient dying of some disease not perhaps in itself fatal, but rendered so by the condition of the kidneys.

If hydronephrosis is unilateral there may be no symptoms until the dilatation of the pelvis of the kidney has reached a sufficient size to form an abdominal tumour. In most of the cases in children this condition has been noted between the third and the eleventh years. This tumour may be situated in the lumbar region, or it may fill the abdomen. It is cystic, and may be confounded with a dermoid cyst of the ovary. On aspiration a fluid is withdrawn which may be clear, or of a brownish colour, and recognised as urine by the fact that it contains urates and urea. After aspiration the urine passed *per urethram* may be bloody. Aspiration affords only temporary relief, as the tumour quickly refills. If an incision is made and the kidney drained, a cure may result with the formation of a fistula. This may continue indefinitely, or infection of the fistulous tract may occur and suppurative nephritis be set up, which

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* This was in every way a remarkable case. The child died apparently of marasmus. There was double hydronephrosis, the ureters being three fourths of an inch in diameter. The right kidney was nodular upon the surface, and had a very adherent capsule. Just beneath the capsule there were small cysts containing pus. The left kidney was the seat of hydronephrosis, only its cortex remaining, this being about one sixth of an inch in thickness. Microscopical examination showed great thickening of the capsule of the right kidney, and several small abscesses situated in the cortex just beneath the capsule. The rest of the kidney was converted into a mass of dense fibrous tissue in which were scattered many uriniferous tubules, the epithelium of which was clear, nucleated, and of the embryonic type. The left kidney was the seat of chronic diffuse nephritis of the atrophic variety, with well-marked changes in the medullary portions. The cortex showed much exudation and less atrophy, being nearly normal in thickness. The small size of the organ was due chiefly to atrophy of the pyramids. The walls of the bladder were greatly hypertrophied, being in places one fourth of an inch thick. The urethra and prepuce were normal.

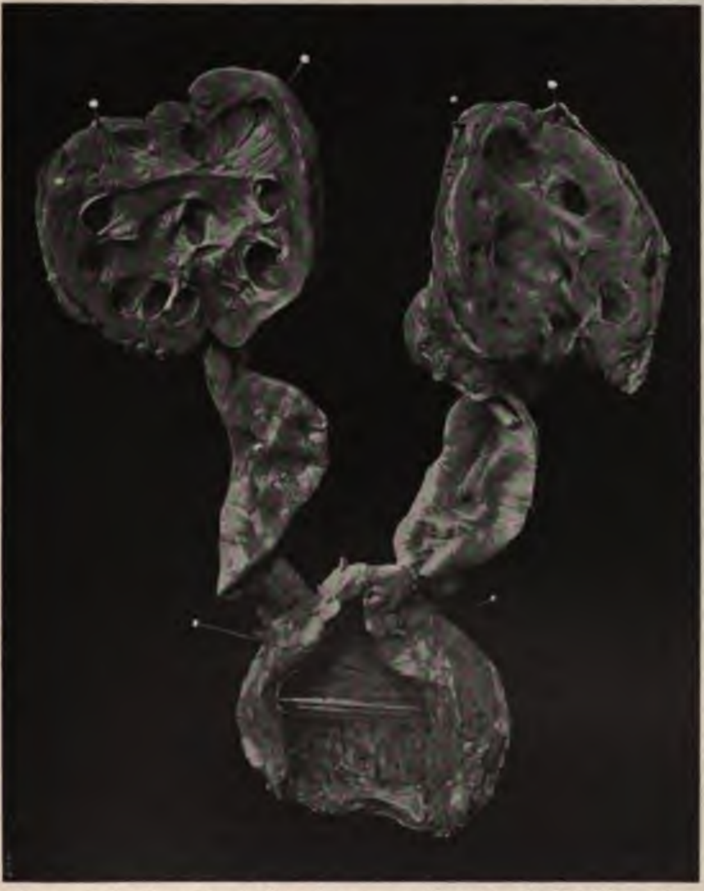

FIG. 118.—Congenital hydronephrosis, dilated ureters, and hypertrophied bladder. (From a child one month old.)

speedily carries off the patient. A better operation is nephrectomy, which may result in a permanent cure if the opposite kidney is healthy, which is usually the case if the child is over three years of age for the reason above stated, viz., that a child with malformation of both kidneys usually dies in infancy.

Supernumerary Ureters.—These were noted in four cases, more frequently on the left side. The usual deformity was for two ureters to be given off, one from the upper and one from the lower part of the kidney, each ureter having a separate pelvis. The ureters either joined just above the bladder, or entered this organ by separate openings. This condition is of no practical importance, and was not found associated with other renal changes.

Malposition of the Kidney.—This was noted in my series of autopsies only once, in the case of fusion of the kidneys already mentioned. Of 21 cases collected by Roberts, the displacement was always of one kidney only; the left being displaced 15 times, the right 6 times. Northrup has reported two cases, both displacements of the left kidney; in one, the organ lay in the hollow of the sacrum; in the other, in the median line, partly above and partly below the promontory of the sacrum. Malpositions of the kidney are compatible with perfect health and development. In most of the cases there is no other deformity present.

Movable Kidney.—This is a very rare condition in early life. Comby (Paris) has collected 18 cases, of which 16 were in girls and 2 in boys. Movable kidney was recognised before the tenth year in 8 cases, and in 2 of these before the fourth month. It has been ascribed to too long a pedicle, which may be congenital; also to pressure from abdominal tumours, and to injury. The most important symptoms are paroxysmal pain which may follow exertion, and a movable tumour. A twist in the ureter may produce hydronephrosis.

URIC-ACID INFARCTIONS.

These consist in a deposit in the straight tubes of the kidneys of uric acid or of amorphous or crystalline urates; usually both kidneys are affected, and all the pyramids of each kidney. The infarctions appear to the naked eye as fine, brownish, fan-shaped striæ. Associated with them there may be granular deposits of uric-acid salts in the pelvis of the kidney, and sometimes evidences of catarrhal inflammation of the pelvis, including even the presence of blood. This condition probably occurs, to some degree at least, in nearly all infants during the first ten days of life. It was formerly supposed that the discovery of these appearances was proof that an infant had breathed, and a certain medico-legal importance was therefore attached to them. This is now known not to be the case, as they are sometimes found in still-born infants.

The cause of this condition is the excretion of uric acid before there is sufficient water to dissolve it, so that the crystals are deposited in the tubes. Uric-acid infarctions are found chiefly in children dying before the end of the second week, although it is not uncommon to see them as late as the third or fourth or even the sixth month. In most of the cases, as the urinary secretion becomes more abundant, the deposits are washed out in the urine and appear as brownish red or pink stains upon the napkins. Infarctions may give rise to a slight inflammation of the renal tubules, but very rarely to any serious lesion; sometimes they remain as deposits in the calices or the pelvis of the kidney or in the bladder, forming the nucleus of a calculus. The symptoms to which they give rise are mainly scanty urination during the first week of life, and occasionally anuria for the first day or two. Sometimes there is evidence of severe pain; priapism may be present, and there is the stain upon the napkin already referred to. The treatment is to give water freely and some alkaline diuretic such as citrate of potash. One grain should be given every two hours until the secretion is fully established; this in most cases will be within twenty-four hours.

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The kidneys are generally enlarged, firmer than normal, and dark-coloured. All the capillary vessels are swollen and distended with blood, and their walls are thickened. In addition to the symptoms of the primary disease, the amount of urine passed is usually scanty and of high specific gravity. Albumin and casts are generally present, but are not constant. The treatment should be directed toward the primary condition, and, in addition, an effort should be made to increase the urine by alkaline diuretics, caffein, digitalis, and the sweet spirits of nitre.

ACUTE DEGENERATION OF THE KIDNEYS.

In the succeeding pages devoted to the kidney I have followed in the main Prudden's classification.

In acute degeneration of the kidney the principal or only change is in the epithelium of the tubules. It is exceedingly common both in infancy and in childhood, being found to a greater or less degree in all autopsies upon patients dying of acute infectious diseases, but it is most marked in cases of scarlet fever, diphtheria, and acute pleuro-pneumonia. It may be found in any disease characterized by prolonged high temperature; and it is the explanation of the cases of so-called febrile albuminuria. The cause is in all probability direct irritation of the epithelium of the tubules by the toxins eliminated by the kidneys. It may also be induced by irritating drugs, such as cantharides or turpentine. By some writers these cases have been classed as examples of acute nephritis; hence the great discrepancy which exists in statements made as to the frequency of nephritis in the different infectious diseases.

The kidneys are usually slightly enlarged, softer, and paler than normal. On section the cortex may be somewhat thickened, and the straight tubules marked by yellowish-gray lines. It is the appearance commonly spoken of as cloudy swelling. The kidneys are seldom much congested. The microscope shows a granular degeneration and death of the epithelium of the tubules, and when severe this may be accompanied by congestion and the exudation of serum.

Acute degeneration of the kidneys gives rise to no symptoms in addition to those of the original disease, except the appearance of a moderate amount of albumin in the urine, with a few hyaline, epithelial, or granular casts. It can not be said that such a condition adds much to the danger from the original disease. In cases that recover, the condition of the kidney entirely clears up. The development of the symptoms of degeneration of the kidneys in infectious diseases calls for no special treatment beyond a continuance of the fluid diet.

ACUTE DIFFUSE NEPHRITIS.

Synonyms: Acute interstitial nephritis, acute exudative nephritis, glomerulo-nephritis, acute Bright's disease.

Etiology.—This variety of nephritis occurs apparently as a primary disease both in infants and in older children. Most such cases are undoubtedly of infectious origin, although the point of entrance of the infection may be difficult or impossible to determine. Acute diffuse nephritis is very frequently secondary to the acute infectious diseases, especially to scarlet fever and diphtheria. It occasionally follows measles, varicella, empyema, typhoid fever, acute diarrhœal diseases, pneumonia, meningitis, influenza, and malaria. It is the characteristic variety of secondary nephritis occurring in severe septic conditions. The exciting cause of the inflammation is in some cases the irritation from toxins; but usually there is in addition the entrance of pathogenic organisms carried by the circulation. Thus in post-scarlatinal nephritis, of which the one under consideration is the characteristic form, the cause is now generally admitted to be the toxins of the primary disease, to which in many cases is added infection by the streptococcus. While nephritis is more frequent after severe attacks of scarlet fever, it may occur after those which are very mild, even when patients have been kept in bed throughout the disease. I have seen two cases of acute nephritis in infants, the apparent cause of which was the irritation of a highly concentrated urine. This was the result of the infants taking for a long time very little food, and almost no water. The frequency of nephritis as a sequel of scarlet fever varies much in different epidemics; the average is from six to ten per cent.

Lesions.—In severe cases the kidneys are usually enlarged, soft, and œdematous. The capsule is non-adherent. The cortex is thickened, either reddened or pale; frequently it is mottled with red, owing to the presence of small hæmorrhages. There may be congestion of the entire organ; or the pyramids may seem unusually red by contrast with the pale and thickened cortex.

All the structures of the kidney—glomeruli, tubular epithelium, and interstitial tissue—are involved in the inflammatory process. The cells covering the glomerular tufts of capillaries are swollen and proliferated. They have frequently undergone fatty degeneration and separated. The epithelial cells lining Bowman's capsule may undergo the same changes, but usually to a lesser degree. The space between the capsule and the tuft may contain exfoliated epithelium in considerable quantity, also cell-detritus, albuminous (granular) exudate, leucocytes, and red blood-cells. The tubular epithelium undergoes albuminous and fatty degeneration and may desquamate. Thus the tubules may contain epithelial fragments, serum, red blood-cells and leucocytes, and some form of casts. The interstitial connective tissue is infiltrated with serous or fibrinous exudate and in places with small round cells. In cases of longer duration a general increase of the connective tissue may take place, which is permanent.

When the glomerular changes are especially marked, as in acute nephritis following scarlet fever, the process is often spoken of as *glomerulo-nephritis*. If the degeneration of the tubular epithelium is extreme, as in severe cases of diphtheria dying shortly after the onset, the nephritis may be described as the *parenchymatous* or *degenerative* type. In the *hæmorrhagic* form there are hæmorrhages into the tubules, glomeruli, or interstitial tissue. In infants and young children the *exudative* type of acute diffuse nephritis is especially frequent. In this there is an exudative inflammation with large accumulations of leucocytes, serum, and red blood-cells in the glomeruli and tubules, the parenchyma and interstitial tissue sometimes being markedly and sometimes but slightly changed. Should the interstitial tissue suffer early and severely, the nephritis becomes of the *productive* or *interstitial* type. This form is most frequently seen with severe, protracted cases of scarlet fever and diphtheria,* especially in older children. It sometimes occurs as an apparently independent process.

Symptoms.—1. *Primary form in infants.*—These cases are not common, and the symptoms are so obscure that they are usually overlooked. In 1887 † I published five cases of my own, and collected from literature fourteen other examples of nephritis, apparently primary, in children under two years of age. Since that time five additional cases have come under my observation. The inflammation in most of them was of the exudative type.

In the exudative type the onset in nearly every instance was abrupt, usually with high fever and vomiting, the temperature being in several cases over 104° F. Dropsy was very exceptional, being noted in but six cases; in most of these it was slight, and seen only toward the close of the disease. Fever was present in all cases. In those observed by myself it was high and irregular in type, ranging from 101° to 105° F. The duration of the disease was from eight days to four weeks, the average being about two and a half weeks. Vomiting and diarrhœa were noted in half the cases, but were rarely prominent, and marked either the onset of the attack, or were traceable to indigestion accompanying the fever; very rarely did they exist as symptoms of uræmia. Anæmia was a prominent symptom in nearly every case, and it was this which enabled me in several instances to make a correct diagnosis. Nervous symptoms were usually prominent. In several patients there was dyspnœa without pulmonary disease, partly due, no doubt, to the anæmia. In nearly all cases there was marked restlessness or muscular twitchings, and in three there were convulsions. Dulness and apathy were present in the majority of the fatal cases, but deep coma was never seen. Several patients presented the typical symptoms of the typhoid condition. The urine was rarely scanty until near the close of the disease, and sometimes not even then. Suppression of urine occurred in but a few cases. Albumin was frequently absent early in the attack, but was invariably present at a late period, although rarely in large amount. Casts were found in all cases that were carefully examined microscopically. They were not usually numerous, and were chiefly of the hyaline, granular, and epithelial varieties. No blood casts were seen. There were usually many pus cells and renal epithelial cells, together with red blood-cells in moderate numbers.

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* Councilman, Mallory, and Pearce, Diphtheria: A Study of the Bacteriology and Pathology of Two Hundred and Twenty Fatal Cases, 1901.

† Archives of Pædiatrics, vol. iv, pp. 1, 103; and ix, p. 263.

Of the twenty-four cases, sixteen died and eight recovered. Of my own ten cases, nine were fatal, the diagnosis being confirmed by autopsy in every case but two. Whether these figures represent the actual mortality of the disease it is difficult to say. No doubt there are many mild cases which are unrecognised. The severe ones, however, are quite uniformly fatal, chiefly on account of the tender age of the patients.

2. *Primary form in older children.*—This also is a rare form of renal disease. As compared with the same condition in infants, the onset is usually less abrupt, the febrile symptoms are less marked, and the termination is less frequently fatal. Dropsy is rarely marked, and often there is none at all. The urine is only slightly diminished in quantity; the amount of albumin is small; casts are not numerous, and usually hyaline, epithelial, or granular; very rarely is there much blood present. Uræmia is infrequent, and the prognosis is better than in infancy.

The interstitial type may begin abruptly with febrile symptoms, dropsy, headache, lumbar pains, scanty urine, and often with vomiting; or it may come on somewhat insidiously with few constitutional symptoms, but with dropsy and changes in the urine.

3. *Secondary form.*—The secondary nephritis of acute infectious diseases usually occurs at the height of the febrile process, and its severity is generally proportionate to the intensity of the infection. The general symptoms of nephritis are often not marked, and dropsy is rare; so that unless the urine is examined the condition may be overlooked. The urinary changes are essentially the same as those already mentioned in the primary cases. Suppression of urine and the development of the symptoms of acute uræmia are infrequent. While nephritis adds considerably to the danger from the primary disease, it is seldom itself the cause of death, although this is sometimes the case in scarlet fever or diphtheria.

The characteristic type of nephritis which follows scarlet fever most frequently develops during the third or fourth week of the disease. The onset may be gradual, dropsy being first noticed. Or it may begin abruptly without dropsy, but with headache, vomiting, scanty urine, fever, and even convulsions. The temperature generally ranges from 100° to 101.5° F., but in very severe attacks it may be 104° or 105° F. While dropsy is usually present, it may be slight or absent in severe and even in fatal cases. It is first seen in the face, next in the feet, legs, and scrotum;

there may be general anasarca, with dropsy of the serous cavities of the body, the pleura, or the peritonæum, rarely the pericardium. As the disease progresses there is always a very marked degree of anæmia.

The urine is, as a rule, greatly diminished in quantity, and may be suppressed. Albumin is invariably present, and usually in large amount, often enough to render the urine solid upon boiling. The urine is of a dark, reddish brown or smoky colour, owing to the presence of red blood-cells or hæmoglobin. The total amount of urea eliminated is far below the normal. The specific gravity may be low, even though the quantity is very small. Casts are present in great numbers, chiefly hyaline, granular, and epithelial casts from the straight tubes; not infrequently there are blood casts. Occasionally twisted or cork-screw casts are seen. Red blood-cells are present in great numbers; also many leucocytes, and always a large amount of renal epithelium.

The duration of the active symptoms in cases terminating in recovery is from one to three weeks. The temperature and dropsy gradually subside. Improvement in the urine is shown by an increase in quantity, by increased elimination of urea, and by a diminution in the amount of blood, albumin, and the number of casts. A few casts may persist for several weeks, and a small amount of albumin for two or three months.

In the graver cases, where the onset is accompanied by high temperature, pain in the back and loins, and a rapid, full pulse of high tension, the urine is very scanty and is often suppressed. Then follow the symptoms of uræmia. In children this is usually manifested by vomiting, great restlessness or apathy, and often by diarrhœa. Less frequently there is headache, dimness of vision, stupor developing into coma, or convulsions. If the secretion of urine is re-established, the nervous symptoms abate and the patient may recover. This has been known to occur after complete suppression has lasted thirty-six hours. Care should be taken not to mistake retention for suppression. If doubt exists, percussion of the bladder and the use of the catheter will quickly settle the question.

There are several complications for which the physician must constantly be on the lookout during attacks of acute nephritis; the most frequent are pneumonia, pleurisy, pericarditis, and endocarditis; more rarely there may be meningitis and œdema of the glottis. It is from complications or acute uræmia that death usually occurs.

Prognosis.—This is to be considered from two points of view: first, the danger to life during the acute stage of the disease, and, secondly, the danger of the development of chronic nephritis. The great majority of patients survive the acute stage, and not infrequently even those recover who have presented grave symptoms of uræmic poisoning. The quantity and specific gravity of the urine, and the number and variety of the casts, are a much better guide in prognosis than the amount of albumin. The existence of severe nervous symptoms, such as stupor, intense headache, dimness of vision, and persistent vomiting, add much to the gravity of the case, as does also the presence of any serious complication. In general it may be said that if there is no suppression of urine, or if there are no symptoms of uræmia and no complications, recovery is almost certain if the child is over three years old; in younger children the outlook is less favourable. The general opinion prevails that acute diffuse nephritis in childhood, whether it is primary or occurs as a complication of scarlet fever, is rarely followed by the chronic form of the disease; and such was the view I formerly held. Larger experience, however, has convinced me that this sequel is not very uncommon. The interval of apparent health may sometimes cover a period of several years, and the later nephritis may be attributed to other causes; but all cases of scarlatinal nephritis should be carefully watched for a long time, and after a severe attack a guarded prognosis should always be given as regards the ultimate result.*

Treatment.—Prophylaxis is important, and relates principally to the secondary form which occurs in the course of infectious diseases, especially post-scarlatinal nephritis; but the measures here outlined apply equally to all varieties. The inflammation of the kidney being in most of these cases the result of direct irritation by the toxins which are eliminated by them, it follows that elimination through the skin and intestines should be increased, and that the urine should be rendered as little irritating as possible by largely increasing its quantity. The first indication is met by frequent sponging, warm baths, and keeping the bowels freely opened by saline cathartics, sufficient being given to produce one or two loose movements daily. To meet the second indication, the patient should be kept upon a fluid diet, preferably milk, at least for the three weeks of the disease, and, if possible, for a full month. At the same time he should drink very freely of alkaline mineral waters, or of plain water to which a small dose (two or three grains) of some alkaline diuretic like the citrate of potassium has been added. If milk is not well borne, kumyss, whey, buttermilk, or junket may be used, or thin gruels mixed with milk. When the first trace of albumin appears in the urine this plan of treatment should invariably be followed. In addition to these measures, after an attack of scarlet fever the patient should be kept in bed for at least a week after the temperature has become normal.

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* The following case may be cited as an illustration of this point: A girl at the age of seven years had scarlet fever, followed by nephritis; the dropsy having lasted, it was reported, for three months. She was believed to have recovered perfectly, and remained in apparent health until she was sixteen, when, as a supposed result of a severe chilling, she developed dropsy and all the symptoms of acute nephritis. From that time, although she lived for three years, and was often for months at a time seemingly in the best of health, her urine was never free from casts and albumin, and she finally died in uræmic convulsions.

The mild cases of acute nephritis tend to spontaneous recovery under the hygienic and dietetic treatment mentioned—i. e., rest in bed, fluid diet, the drinking of large quantities of water, and attention to the action of the skin and bowels. These measures should be continued so long as the urine contains any considerable amount of albumin, or so long as the patient's general condition will permit. Should he become very anæmic, or lose much in weight, it may be necessary to enlarge the diet by the addition of solid food. This should at first be of the carbohydrates only, usually in the form of some farinaceous food. An increase in the diet and exercise should be made very gradually, and the effect upon the urine carefully watched.

The severe cases, with scanty urine, fever, and marked dropsy, require more active treatment. Free diaphoresis should be maintained by the hot pack or vapour bath (page 56). Active counter-irritation should be maintained over the kidneys by dry cups followed by poultices, or the mustard paste. Two or three loose movements from the bowels should be secured by the administration of calomel, or, better, by Rochelle, or Epsom salts. Harm is sometimes done by carrying this depletion too far, and its effect upon the patient's general condition must be closely watched. If suppression of urine occurs with the development of uræmic symptoms—delirium, high temperature, flushed face, vomiting, and a pulse of high tension—nitroglycerin is indicated; a child of five years may take gr. $\frac{1}{200}$ every hour for five or six doses, or until an effect is produced.

In addition to these measures rectal injections of a normal salt solution should be given high in the colon, at a temperature of from 104° to 108° F. At least a pint should be given several times a day, to be continued until a free flow of urine is established. This is one of the most valuable means we possess of increasing elimination by the kidneys and skin.

The nervous symptoms of uræmia are best relieved by chloral or chloralamid, which should be given per rectum. When such symptoms are marked, from six to ten grains are required for a child of five years, to be repeated in two hours if no improvement is seen. Uræmic convulsions may sometimes be averted by the use of morphine hypodermically. In extreme conditions not relieved by the measures mentioned, venesection should by all means be practised; from three to six ounces of blood may be drawn from a child of five years, according to his general condition and the urgency of the symptoms. The depressing effect may largely be overcome by immediately following this with an intravenous injection of a normal salt solution. Twice as much as the fluid drawn should be introduced. This will almost invariably give at least temporary relief, which may afford time for the operation of other measures such as catharsis and diaphoresis. Pulmonary œdema is no contra-indication to bleeding; the best of all guides as to its use is a pulse of very high tension.

One should always be on the lookout for complications, especially dropsy of the serous cavities, pericarditis or endocarditis, and œdema of the lungs. Convalescence is nearly always slow, and a patient who has suffered from nephritis needs careful attention for a long time. Anæmia is always present, and iron is required. The diet must consist largely of fluids for several months. If the disease tends to pass into a subacute form, the child should, if possible, be sent to a warm climate, and kept there during the succeeding winter, and every means taken to improve the general nutrition. Flannels should be worn next to the skin, and special precautions taken against any exposure which might cause an exacerbation of the disease.

CHRONIC NEPHRITIS.

Chronic inflammation of the kidney is an infrequent condition in childhood. In infancy it is almost unknown, except in connection with congenital hydronephrosis or other malformations of the kidney. Two pathological varieties are met with: (1) Chronic diffuse nephritis of the parenchymatous or degenerative type. (2) Chronic diffuse nephritis of the interstitial or productive type. As the disease progresses the former may assume the characteristics of the latter variety.

Etiology.—Chronic nephritis is most frequently seen as a sequel of the acute nephritis of scarlet fever. It also occurs with the prolonged suppuration of chronic bone or joint disease, where it may be chronic from the beginning. The only other important causes in early life are hereditary syphilis, alcoholism, chronic tuberculosis, and valvular disease of the heart. Nearly all the cases occur in children over five years of age.

Lesions.—The lesions of chronic nephritis in childhood do not differ essentially from those seen in later life. In the chronic parenchymatous type the kidneys are usually enlarged, the surface is smooth or slightly nodular, and the thickened cortex yellowish white on section. These are often called " large white kidneys." On the other hand, the kidneys may be nearly normal in appearance, or smaller and with a thinner cortex than is usual. In the so-called " large red kidneys " the cortex is red or mottled red and yellow, owing to hæmorrhages into the tubules or interstitial tissue. The microscope shows that the renal epithelium is swollen, granular, fatty, and degenerated. The tubes contain leucocytes, red cells, cast matter, and the detritus of broken-down epithelial cells. In some places they are dilated, in others atrophied. In the glomeruli there is a growth of capsule cells, compression and atrophy of the tufts, with the formation of new connective tissue. When there is waxy degeneration, the kidneys are usually considerably enlarged, and of a glistening gray colour. Amyloid degeneration is seen especially in the small arteries of the kidney and the capillary vessels of the tufts. With iodine the mahogany-brown reaction is obtained. Amyloid changes in the kidney are nearly always associated with similar lesions in the liver and spleen, and sometimes also in the intestinal villi.

In the chronic diffuse nephritis of the interstitial type (granular kidney) the organs are smaller than normal, with a nodular surface and adherent capsule. The cortex is thinned, and the colour is gray or red. In addition to the lesions found in the preceding variety, there is an extensive production of new connective tissue, which is irregularly distributed throughout the kidneys. The tubules in some places are dilated to form cysts of considerable size, while in others they have completely disappeared. The glomeruli may be atrophied to little fibrous balls; or if chronic congestion has preceded the inflammation, some may be large and the capillaries dilated.

Symptoms.—1. *Chronic nephritis of the parenchymatous type.*—This form of the disease may be chronic from the outset, or follow an acute attack from which the patient is often believed to have recovered completely. The symptoms sometimes immediately follow the acute attack; at others there is an interval of apparent recovery, extending over a few months or even years. Very rarely no such history of an antecedent acute attack can be obtained, and the symptoms come on gradually and insidiously. Such cases occur chiefly in older children, and their clinical features do not differ essentially from those of adult life.

As a rule dropsy is present, although it is variable in amount, and fluctuates considerably from time to time. There may be not only œdema of the cellular tissue, but effusion into the pleura, peritonæum, and even the pericardium. As the case progresses, anæmia is always a marked symptom. There are various disturbances of digestion—loss of appetite, occasional vomiting, and attacks of diarrhœa. From time to time nervous symptoms may be quite prominent, such as headaches, sleeplessness, neuralgia, fatigue upon slight exertion, and dyspnœa. Attacks of epistaxis are not infrequent.

For the greater part of the time the urine contains albumin and casts. They vary much in amount at different periods in the disease, according to the rapidity of its progress. During periods of exacerbation, both albumin and casts are very abundant, while in the intervals the amount of albumin may be small and the casts few. The casts are hyaline, granular, epithelial, and fatty. The daily quantity of urine is much reduced during the periods of exacerbation, while at other times it may be nearly normal. The specific gravity is usually normal or high.

If waxy degeneration is present, there are generally associated with the renal symptoms, others dependent upon waxy changes in other organs. The spleen and liver are enlarged; there may be ascites and diarrhœa, and there is usually present the peculiar "alabaster cachexia."

The duration of this form of chronic nephritis depends much upon the surroundings of the patient and the treatment. It is rarely shorter than two years, and it may last for many years. The progress is always irregular, and marked by periods of exacerbation and remission. The patients die from acute uræmia, or from complicating pneumonia, pleurisy, pericarditis, endocarditis, or from pulmonary œdema.

2. *Chronic nephritis of the interstitial type.*—This is a very rare disease in early life, being much less frequent even than the preceding variety of nephritis. In some cases there is a history of hereditary syphilis; in others, of chronic alcoholism. The early symptoms are few, and the disease usually develops insidiously. The urine is pale, excessive in amount, and of low specific gravity—1·001 to 1·008. Albumin is often absent, and, when found, the quantity is small. Dropsy likewise is rare, and never marked. Nervous symptoms are often prominent, such as headache, attacks of spasmodic dyspnœa resembling asthma, neuralgias, and disturbances of vision. High arterial tension and hypertrophy of the left ventricle are regular symptoms; and even atheromatous degeneration of the arteries may be present. Dickinson reports an instance of this in a patient only six years of age. Late in the disease, hæmorrhages may occur, and these may be the cause of death. Filatoff has reported a cerebral hæmorrhage in a child of eleven. Acute uræmia is, however, the usual termination of this form of nephritis. The course is slow, and the disease may be overlooked until the final uræmic symptoms occur.

Prognosis.—The prognosis of chronic nephritis as to complete recovery is always unfavourable; and although cases are seen in which symptoms are absent for several years, they almost invariably return. Cases have been reported of recovery from waxy degeneration of the kidney after removal of the bone disease upon which the condition depended. An extended period of observation is necessary before the patient can be pronounced cured. As to the duration of the disease, no exact prognosis can be given, because, from the symptoms, it is difficult or impossible to determine exactly the extent of the disease in the kidney and the rapidity of its progress. The continued passage of a large amount of urine of low specific gravity is invariably to be interpreted as evidence of fibroid changes in the Malpighian tufts, and is a bad symptom. A large amount of dropsy, the coexistence of valvular disease of the heart, and marked renal insufficiency, as shown by the quantitative examination of the urine, are all very unfavourable symptoms.

Diagnosis.—Chronic nephritis, like the acute forms, is likely to be overlooked because of the failure to examine the urine in children. Regular and frequent examinations should be made in all cases of convulsions, of persistent or frequent headaches, severe anæmia, hypertrophy of the heart, high arterial tension and of general malnutrition, as well as when the more obvious symptoms of renal disease, such as dropsy and scanty urine, are present. Nor should one be too ready to make the diagnosis of functional albuminuria because he finds albumin only occasionally and in small quantity. All such cases demand most careful observation and the closest attention for a long period before excluding organic renal disease.

Treatment.—Children with chronic nephritis are to be treated on the same general plan as adults. The purpose of treatment is to retard as much as possible the progress of the disease and to relieve the symptoms as they arise. It is of the greatest importance to remove the patient from conditions in which exacerbations are liable to occur. If it is possible, he should be sent to a warm, dry climate in winter, and all exposure to cold avoided; an out-door life is desirable. Most patients require general tonic treatment with very moderate but regular exercise, never carried to the point of fatigue, as much rest as possible in a recumbent position, a fluid diet, consisting largely of milk as long as this can be borne, and the administration of iron, particularly the tincture of the chloride. Excessive dropsy calls for diuretics, saline cathartics, and heart stimulants. If uræmia develops, with high arterial tension and stupor, headache, and convulsions, venesection should be resorted to, or nitroglycerin used. Morphine may be given hypodermically if the pupils are dilated and nervous symptoms are very marked.

TUBERCULOSIS OF THE KIDNEY.

In general tuberculosis, miliary tubercles are frequently seen both upon the surface of the kidney and in its substance. These give rise to no symptoms and are of no clinical importance. Larger tuberculous deposits are extremely rare in early life. They usually occur in patients who are the subjects of general tuberculosis, and are associated with tuberculosis of other parts of the genito-urinary tract, or they may exist as the primary, or even the only, tuberculous lesion in the body. Hamill* (Philadelphia) observed one case of primary renal tuberculosis in an infant seven months old, and collected 54 others in children under fourteen years. A number of these, however, are very doubtful. Boys were more often attacked than girls. Only 2 cases were under one year of age; 13 were between one and five years; 11 were between five and ten years old.

A study of these cases shows that ascending infection occurs occasionally but that it is rare; and that nearly all cases are of the descending type—i. e., primary in the kidney. Infection of the kidney therefore generally takes place through the circulation and not from the bladder.

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* S. M. Hamill, Primary Tuberculosis of the Kidney in Children. From the Pepper Laboratory for Clinical Medicine, Philadelphia, 1896. International Medical Magazine, 1896, v, No. 2.

Aldibert's figures show that in children the bladder usually escapes even when the kidneys are tuberculous, for of 13 cases of renal tuberculosis the bladder was involved in but 2. The disease when primary begins in the cortex, but soon extends to the mucous membrane of the pelvis and the calices of the kidney, and also to the pyramids. As a rule, but one kidney is affected. The process may be confined to the pyramids, where are found cheesy nodules which may be single or multiple. These ultimately break down and form abscesses. The process may result in almost complete destruction of the pyramids, and even of portions of the cortex, so that the kidney may consist of a mere shell of renal tissue. Suppuration in the neighbourhood of the kidney (perinephritic abscess) often coexists.

The symptoms are quite indefinite. There may be localized pain and tenderness in the region of the kidney, and a tumour if there is perinephritis. The symptoms of irritability of the bladder may be almost as severe as in cases of calculus. Pus usually appears in the urine as a constant symptom, and blood is often present. But the only thing that is diagnostic is the discovery of tubercle bacilli in the urine.

The treatment of renal tuberculosis is purely surgical. Of the 17 cases collected by Hamill in which operation was done for this condition, there were 11 recoveries and 6 deaths, 2 of the deaths, however, not being traceable to the operation or to the original disease. Nephrotomy was done 4 times, with 2 recoveries, 1 improvement, and 1 death. Nephrectomy was done 9 times, with 5 recoveries, 1 improvement (died later from perforation of the duodenum), and 3 deaths. Nephrectomy followed nephrotomy in 4 cases, of which 2 recovered, 1 died, and 1 improved. No recurrence had taken place in one case at the end of eight years, and none in another after three years.

MALIGNANT TUMOURS OF THE KIDNEY.

In the great majority of cases tumours of the kidney are malignant. Of 51 cases collected by Aldibert which were operated upon, 48 were malignant and 3 benign.

Malignant growths are almost invariably primary. In children under five years, although not common, they are yet more frequent than any other variety of malignant tumour of the abdomen. The earlier cases reported were classed as carcinoma. It is now well established that carcinoma is very infrequent, and that nearly all the cases are varieties of sarcoma. Fischer reports 19 of sarcoma and 2 of carcinoma; Aldibert, 38 of sarcoma and 5 of carcinoma. The sarcoma may be round- or spindle-celled, or myo-sarcoma. In some of the cases there are both sarcomatous and carcinomatous features, so that they might be classed as sarcomatous carcinoma. The tumour grows from the cortex of the kidney, or from the pelvis, sometimes from the adrenals.

It may infiltrate the whole kidney, so that there is no trace of renal structure remaining, or it may form an immense tumour on one side of the kidney, which is only partially invaded. These tumours are very rarely cystic, but they are quite soft, and hæmorrhages often occur into their substance. There may be secondary growths in the liver, the lungs, the retroperitoneal glands, in the opposite kidney, in the intestines, or in the pancreas. Pressure of the tumour upon the ureter may lead to hydronephrosis; and upon the inferior vena cava, to thrombosis of that vessel. As it grows, the tumour sometimes becomes adherent to nearly all the abdominal organs by localized peritonitis. It may lead to ascites, but it very rarely causes general peritonitis. The growth may reach a great size, usually from 5 to 15 pounds, but in 1 case reported by Jacobi it weighed 36 pounds. In Seibert's collection of 48 cases the right kidney was involved in 24, the left in 22, and both kidneys in 2 cases.

Etiology.—These tumours of the kidney may be congenital. This was true of 5 cases in a series of 55 collected by Jacobi. The majority occur in early childhood. In the collection of 130 cases by Longstreet Taylor in which the ages are given, 106 were in the first five years, and 57 of these in the first two years of life. The sexes were about equally affected. In a small number of cases the history of a fall was given.

Symptoms.—The principal symptoms are tumour, hæmaturia, and cachexia. The tumour is usually first noticed. It is in most cases discovered in the loin, but grows forward toward the median line. Its surface may be lobulated and irregular or quite smooth; and although solid, it is sometimes so soft as to give an obscure sensation of fluctuation. It may grow to an enormous size, causing displacement of the liver, spleen, intestines, and lungs. The progress of the growth is usually rapid, so that from the size of a fist, the tumour may grow in the course of five or six months so as to fill the abdomen. By careful palpation it will be found—certainly when the tumour is small—that although it may be quite freely movable, its attachment is near the lumbar spine. Aspiration may show blood, but more frequently the result is negative.

Hæmaturia was observed before the tumour in 19 of 50 cases (Seibert), it being then the first symptom noticed. The amount of blood passed is sometimes quite large, but is usually small, and may be discovered only by the microscope. Pain is rare, and is due to localized peritonitis. Constitutional symptoms are absent until the tumour has attained a large size, when a cachexia develops and the patient wastes steadily while the tumour continues to grow. The pressure effects are dyspnœa, from compression of the lungs; œdema of the lower extremities, from pressure upon or thrombosis of the vena cava; vomiting and indigestion, from pressure upon the stomach and intestines. Secondary deposits very rarely cause any symptoms except in the lungs, where they may give rise to cough, and even to hæmoptysis.

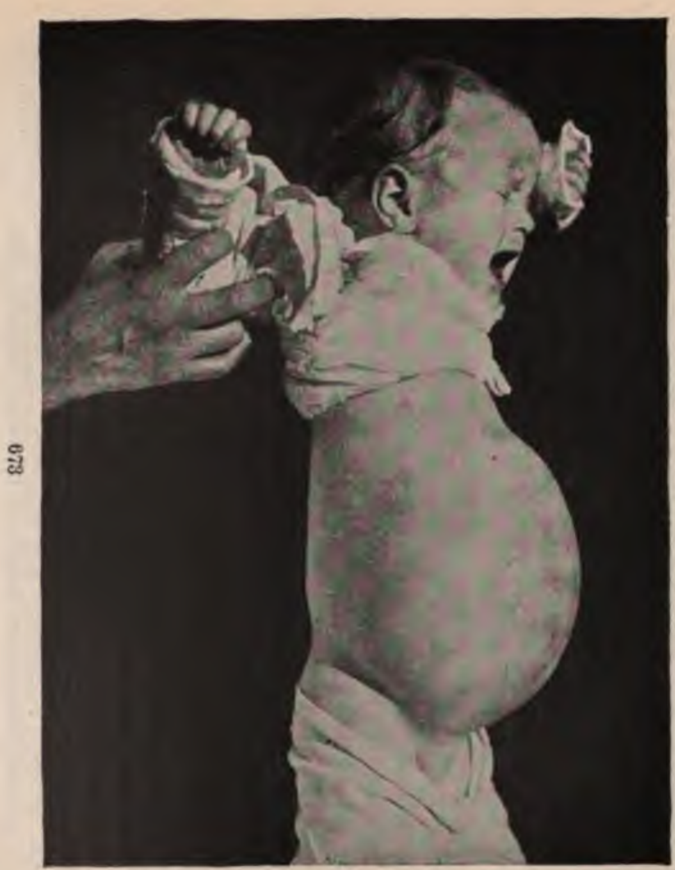

FIG. 119.—Sarcoma of the kidney, child thirteen months old.

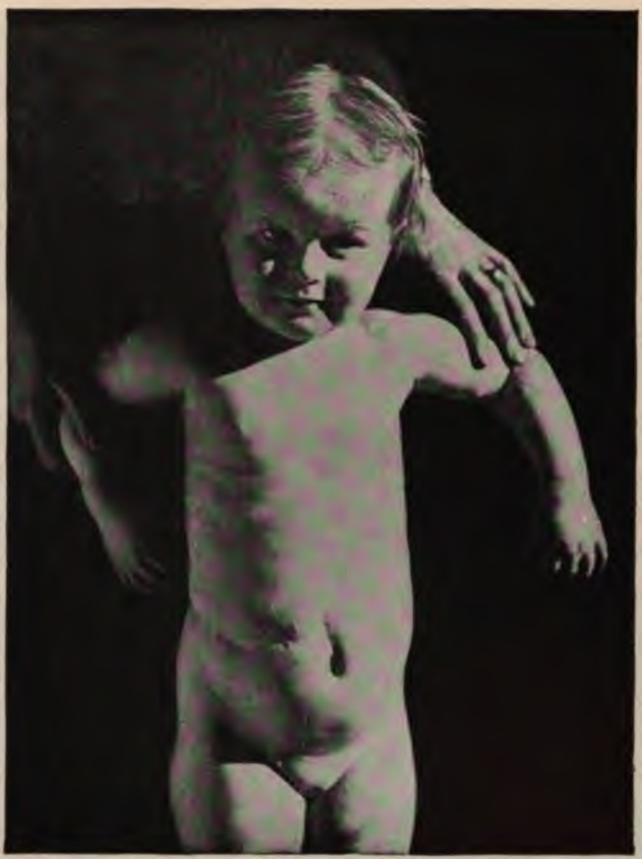

FIG. 120.—The same child one year after operation. Ten years after operation in perfect health.

The course of the disease is steadily from bad to worse. The usual duration of life in patients not operated upon, is from three to ten months after the tumour is large enough to be easily discovered.

Diagnosis.—The important points are, the position and attachment of the tumour, its steady growth and solid character, hæmaturia, and the age of the patient (under five years). It may be confounded with hydronephrosis, dermoid cyst of the ovary, enlargement of the spleen, retroperitoneal sarcoma, tumours of the liver, or even of the abdominal wall.

Treatment.—Nothing is to be said regarding the medical treatment of these cases. Unless operated upon, I believe they invariably terminate fatally. Some of the results of operation during recent years have been so encouraging that no case should be abandoned, no matter how young the patient. Lewi * has collected the results of 60 cases operated upon: 20 deaths occurred soon after operation, from causes connected with it; in 20 cases the cause of death was recurrence of the growth; this raises the total mortality to 67 per cent. In the Babies' Hospital, my colleague, Dr. Robert Abbe, operated upon a nursing child, thirteen months old, where the tumour weighed 7 pounds, and the child after the operation only 15 pounds. This case made an uninterrupted recovery, and ten years after the operation was still in perfect health. The accompanying illustrations (Figs. 119 and 120) are from photographs of this patient. A second child operated on at two years remained well for three and a half years and died from a recurrence in the opposite kidney.

For a discussion of the surgical aspects of this question, and details of the operation, see the papers of Abbe † and Aldibert.‡

Benign Tumours.—These are distinguished by their slow growth, and by the fact that the constitutional symptoms are mild or wanting. Of the three cases mentioned by Aldibert, one was adenoma, one fibroma, and one was fibro-cystic.

PYELITIS—PYELO-CYSTITIS.

Pyelitis is an inflammation of the mucous membrane lining the pelvis of the kidney; cystitis is an inflammation of the mucous membrane of the bladder. They may exist separately or together. With pyelitis there may be inflammation of the ureter or of the kidney itself (pyelo-nephritis), and it may be acute or chronic. It may result in the accumulation of pus in considerable amount in the pelvis of the kidney (pyelo-nephrosis).

Etiology.—The most frequent local cause of pyelitis is irritation from renal calculi. It is also associated with congenital malformations of the kidneys or ureters, with renal tuberculosis and renal tumours. It may result from an extension of inflammation from the tissues surrounding the kidney (perinephritis), or from an abscess opening into the pelvis of the kidney. An infectious form of acute pyelitis sometimes occurs as a complication of scarlet or typhoid fever, diphtheria, malaria, or pyæmia; but it is also seen apart from these diseases, when it occurs apparently as a primary affection. In most of the severe cases of pyelitis there is also present a certain amount of nephritis.

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* Archives of Pædiatrics, February, 1896. † Annals of Surgery, January, 1894.

‡ Revue Mensuelle des Maladies de l'Enfance, November, 1893.

Acute pyelitis may also be secondary to acute cystitis even in infants. In such cases the inflammatory process travels upward along the ureter, which may or may not be involved. These cases of cystitis occur chiefly in female infants and have been especially studied by Escherich, Trumpp, and Finkelstein, who found the characteristic features of the disease to be the presence of the colon bacillus in pure culture in freshly voided urine; the term "coli-cystitis" has been applied to them. Of ten cases observed by Escherich and seven by Finkelstein, all were girls. I have myself seen six severe cases, all in female infants from six to twelve months of age, which corresponded closely with the type described by these writers. The infection probably occurs through the urethra, and originates from the stools through the napkins or the passage of the stools over the vulva. This more frequently occurs in diarrhœal diseases, with which the cystitis has often been found associated. It is surprising that vulvo-vaginitis is seldom present. It seems quite possible that infection may also occur, especially in male infants, by a direct extension from the intestine to the bladder, or through the blood. Trumpp examined the urine in sixteen patients with gastro-enteritis and found the colon bacillus in thirteen, of whom nine were females. The association of cystitis and gastro-enteritis deserves further study.

Lesions.—When pyelitis develops from a local cause it is usually unilateral; otherwise both sides are involved. In the cases of acute cystitis or pyelo-cystitis there are the usual appearances of an acute catarrhal inflammation of the mucous membrane, with congestion, swelling, and sometimes minute hæmorrhages. In chronic cases there is thickening and sometimes a granular condition of the lining membrane. There may be an accumulation of pus of considerable size, distending the pelvis and calices (pyonephrosis). If the condition is one depending upon a calculus or congenital deformity, and in all protracted and severe cases, the kidney itself is involved to a greater or less degree; the extent of the nephritis will depend upon the nature of the exciting cause and the duration of the process.

Symptoms.—The history of the following case illustrates the main clinical features of acute infectious pyelitis, in this instance occurring apparently as a primary disease:

A previously healthy female infant of eight months was taken suddenly with a chill, followed by a very high fever. The child was ill for ten days before the nature of the disease was suspected. During this time the temperature ranged between 101° and 106° F., touching 105° nearly every day; but the chill was not repeated. The other constitutional symptoms were not severe. At the first examination of the urine there was found a large amount of pus, which on standing was equal to one twelfth of the volume of the urine passed; the reaction was strongly acid. There were no signs of vaginitis or vulvitis, no *ardor urinæ*, no evidence of local pain either in the bladder or kidney, no abnormal frequency of micturition, no localized tenderness, and no vomiting. At later examinations there were found in moderate numbers epithelial cells from the bladder, and the tubules and pelvis of the kidney, also a few hyaline casts, but not more albumin than would be explained by the amount of pus. Under no treatment except alkaline diuretics, the temperature gradually fell to normal, and the pus steadily diminished in quantity, and at the end of five weeks had practically disappeared from the urine. A report sixteen months later stated that the child had remained well and entirely free from urinary symptoms.

In some cases there are recurring chills, with wide fluctuations in temperature; in others there may be only pyuria, with moderate fever and few other constitutional symptoms. If the disease complicates one of the acute infectious diseases, pyuria may be the only symptom. If cystitis is also present micturition is frequent and may be painful. The urine in acute pyelo-cystitis is turbid from the presence of pus, the amount of which may be from one to fifty per cent of the volume of the urine. The quantity of urine is generally somewhat diminished, and it may be quite scanty. The reaction is usually acid, even though the amount of pus is large. Albumin is present in proportion to the amount of pus or the degree of nephritis. Red blood-cells are found under the microscope in most of the very acute cases, and may be in sufficient numbers to colour the urine. The pus cells in recent cases are usually well preserved, but in old cases they may be degenerated. There are many epithelial cells—conical, fusiform, and irregular cells with long tails. There may be renal epithelium and hyaline, granular, or epithelial casts, varying in number with the severity of the nephritis. The colon bacillus may be present in pure culture.

In chronic pyelitis only pyuria may be present, or there may be a tumour owing to the pyonephrosis. From time to time in the chronic form there may be intermittent attacks of acute pyelitis resembling those above described. In pyelitis depending upon congenital malformations, pyuria is usually the only symptom, unless pyonephrosis is present. With calculi we may have acute or chronic pyelitis; there may be localized pain, tenderness, sometimes a tumour, occasionally hæmaturia, and perhaps a history of renal colic or the passage of gravel. With tuberculosis we have chronic pyuria and the presence of tubercle bacilli in the urine. There are commonly associated the symptoms of general tuberculosis. If associated with perinephritis, the inflammation is usually acute, and there are present the local symptoms of the original disease. If an abscess opens into the pelvis of the kidney we may have a sudden discharge of pus in large quantity with a subsidence of previous local symptoms, including the tumour. With neoplasms we have congestion and hæmorrhage more frequently than pus, but both may be present.

Diagnosis.—The characteristic symptoms of acute pyelitis are chills, which may be repeated, high and fluctuating temperature, scanty urine, frequently pain and tenderness over the kidneys, and pyuria. The diagnosis of pyelitis is made only by an examination of the urine, which should never be omitted in cases of obscure high temperature, even in infancy, particularly if chills are present. When cystitis is associated, the only additional symptoms may be pain and other signs of vesical irritation. These symptoms, with an acid urine containing a large amount of pus and epithelial cells like those described, are sufficient to establish the diagnosis of pyelo-cystitis. If the pus comes from the opening of an abscess into the bladder, ureter, or pelvis of the kidney, the local signs of such abscess will usually be present.

Prognosis.—In cases apparently primary, and in those complicating infectious and other diseases, the prognosis is good. The danger is chiefly from the nephritis which follows or complicates the process. In cases depending upon local conditions, the prognosis will depend upon the nature of the exciting cause. Here, also, the principal danger is from nephritis. If calculi are present and if pyonephrosis occurs, the patient may die from exhaustion before a serious degree of nephritis has developed.

Treatment.—Water should be given freely, and alkalies up to the point of neutralizing the excessive acidity of the urine. In infants, from twelve to twenty-four grains of the citrate of potash are required daily for this purpose. If the urine is alkaline, benzoic acid may be used in the same doses. The most important remedy is urotropin, which should be given in doses of one or two grains every three hours to an infant of a year, and proportionate doses to older children. In acute cases, counter-irritation over the kidney by means of poultices or dry cups may be employed. If calculi are present the same treatment is indicated. Surgical interference is called for if pyonephrosis develops, or if the disease is evidently unilateral and the kidney is seriously involved. The advisability of surgical interference will depend upon the clearness of diagnosis and the severity of the symptoms.

RENAL CALCULI.

Small renal calculi are very common in infancy. In the autopsy-room we frequently see, on opening the kidneys of young infants, fine brown granules in the pelvis and calices, and occasionally a calculus as large as a small pea is found. They are usually composed of uric acid. Only once in over one thousand autopsies of which I have records, was a stone of any considerable size seen in an infant. In this case it was an inch in length and half an inch wide. It is surprising that these are so rare, when we consider how very frequently the minute calculi are met with. The probable explanation is, that the majority of them are dissolved or washed down into the bladder and passed *per urethram* because of the fluid diet of the first two years. The granular deposits are usually lodged in the pelvis of the kidney, and are generally seen upon both sides. With the larger collections there is often a slight catarrhal pyelitis.

Symptoms.—The small deposits give no symptoms, and even quite large calculi may be found at autopsy where no indication of their presence had existed during life, as in the case above mentioned. In some cases symptoms are produced which resemble those of renal calculi in the adult. In infants less definite symptoms are often passed over as merely intestinal colic.

In well-marked cases in older children there is tenderness, pain localized over the affected kidney, or radiating to the bladder, the perinæum, and even the opposite kidney, and there may be irritation and retraction of the testicle. The urine may show, especially after exercise, a trace of blood; there may be the added symptoms of pyelitis, with some fever, localized tenderness, and the appearance in the urine of pus and epithelial cells from the pelvis of the kidney.

Renal colic is produced when a stone of any considerable size passes from the kidney to the bladder. It is characterized by symptoms similar to those seen in the adult. There are sudden attacks of severe sickening pain in the loins, shooting down the thigh or to the testicle. There may be vomiting and even collapse. The urine is passed frequently, in small quantities, and contains blood. The symptoms quickly subside when the stone reaches the bladder. The calculus may sometimes become impacted in the ureter and give rise to hydronephrosis or pyonephrosis, which soon becomes pyelo-nephritis.

The existence of small calculi may be suspected from the symptoms above mentioned; the diagnosis is made positive by the appearance of gravel in the urine. The use of the Röntgen rays is of service in recognising even small calculi.*

Treatment.—The only medical treatment consists in a fluid diet, the free use of alkaline mineral waters, and a sufficient quantity of some drug to render the urine alkaline. Such measures will relieve only the milder conditions. With larger calculi and more marked symptoms, a surgical operation should be considered and should be urged in proportion to the severity of the symptoms and the clearness of the diagnosis. If calculous pyelitis exists, it is certain sooner or later to lead to serious nephritis, and it is only a question of time when the kidney will be disabled. The same is true of hydronephrosis from the impaction of a calculus in the ureter. Aldibert has collected four cases of nephrectomy in children for renal calculi in which the kidney was healthy, with three recoveries and one death from shock. In nine cases of operation for calculous pyonephrosis, there were six recoveries and three deaths. This is certainly an encouraging showing, and should lead one to consider operation seriously in many cases for which formerly nothing was done. The earlier the operation the greater the chances of success, because of the better condition of the other kidney. Although the continued use of water and the so-called solvents may relieve some of the symptoms, it is very questionable whether they do more.

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* Abbe, Annals of Surgery, August, 1899.

TRAUMATIC HYDRONEPHROSIS.

In addition to the hydronephrosis which results from congenital malformations and from the impaction of calculi, a form is occasionally seen following severe injury to the kidney. The pathology of hydronephrosis in these cases is not well understood. After the early symptoms of traumatism have subsided, there develops in from two weeks to two months a tumour in the region of the kidney, which may reach a considerable size and present all the ordinary characteristics of hydronephrosis arising from other causes. This tumour may disappear spontaneously, or it may increase in size and demand surgical intervention for its cure. In seventeen cases which Aldibert has collected there was only one of spontaneous recovery; aspiration was done in seven cases, with six cures and one death; incision with or without nephrectomy was practised in nine cases, with seven recoveries and two deaths.

PERINEPHRITIS.

This consists in an inflammation in the cellular tissue surrounding the kidney, which may terminate in resolution or in suppuration. It is not of very uncommon occurrence, and is of importance chiefly from the frequency with which it is confounded with disease of the hip or spine. Perinephritis may be secondary to suppurative processes in the kidney itself, whether from calculi or tuberculous deposits, or it may be primary. In children the latter is the common form. Primary perinephritis is attributed to traumatism, cold, or exposure, or it may develop without assignable cause. It usually runs an acute or subacute course; very rarely it may be chronic.

For the clinical picture of this disease I am chiefly indebted to a paper by Gibney, who published in 1880 a report of twenty-eight cases of primary perinephritis in children. I was at that time an interne in the Hospital for the Ruptured and Crippled, New York, where these cases were under observation, and had an opportunity to see many of those reported in Dr. Gibney's paper.*

The ages of these patients were between one and a half and fifteen years, the majority being between three and six years. The two sides and the two sexes were about equally affected. About one third of the cases were clearly traceable to traumatism; in the others no adequate exciting cause could be discovered. The majority of the cases were referred to the hospital with the diagnosis of hip-joint disease or caries of the spine. Resolution followed in twelve of these cases, and sixteen terminated in suppuration.

When abscess forms, it usually burrows between the lumbar muscles and comes to the surface posteriorly near the middle of the ilio-costal space; it may burrow forward between the abdominal muscles and point just above Poupart's ligament; very rarely it may follow the psoas muscle and appear at the upper and inner aspect of the thigh, like an ordinary psoas abscess; or it may open into the peritoneal cavity.

Symptoms.—The onset of acute perinephritis may be quite abrupt, with chill, fever, and localized pain; or it may be gradual, with stiffness of the spine, lameness referred to the hip, and deformity due to contraction of the flexors of the thigh. The pain is usually felt in the loin, but may be referred to the groin, to the inner side of the thigh, or to the knee. It is often severe, and increased by using the limb. It is in most cases accompanied by localized tenderness in the neighbourhood of the kidney. There is lameness upon the affected side which may come on gradually, being sometimes referred to the hip and sometimes to the spine. These symptoms often develop slowly in the course of two or three weeks. They are usually accompanied by a slight elevation of temperature. In the most acute cases the temperature is high (102° to 104° F.), and prostration severe.

As the disease progresses fever is a constant symptom, the temperature usually varying between 101° and 103° F. There is in most cases increasing deformity, and finally the patient may be unable to walk at all. On examination at the height of the disease there is found in a typical case a deviation of the spine with the concavity toward the affected side; the thigh may be held flexed to a right angle; passive extension is resisted and causes pain, although all the other movements at the hip joint are normal. In the lumbar region there is tenderness, and there may be an area of infiltration filling the ilio-costal space. At first this is only appreciable by percussion, but later a distinct tumour is present. In addition to the tumour in the usual region, there is sometimes one at the upper and inner aspect of the thigh, owing to a burrowing of pus, and the sacs may communicate.

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* Chicago Medical Journal and Examiner, 1880, where will be found a very full bibliography.

Lameness, pain, deformity, and fever sometimes exist for two or three weeks before any tumour can be made out. The constitutional symptoms are often severe, and symptoms of the typhoid condition may even be present. The bowels are usually constipated. The size of the abscess is sometimes very great. In one case I have seen it extend from the spine to the median line in front, and from the crest of the ilium nearly to the free border of the ribs. The amount of pus varies from a few ounces to two or three pints. Urinary symptoms are sometimes wanting; at other times there is increased frequency of micturition, accompanied by pain from an irritation referred to the bladder. The urine may contain pus from a complicating pyelitis. In only one of Gibney's cases was this present. It developed in the fourth week, and the case recovered.

The duration of the disease in the acute cases varies from three to eight weeks; in the subacute it may be five or six months. When suppuration occurs the symptoms subside quite rapidly after the pus has been evacuated, and recovery is complete. Where resolution takes place, there is a gradual subsidence of the symptoms, and often some stiffness of the thigh, with slight lameness for several months. In the series of cases above referred to, 65 per cent recovered completely in three months.

Diagnosis.—In many cases a diagnosis of hip-joint disease is made, and they are reported as "hip-joint disease cured without deformity," etc. The points of differential diagnosis are quite distinct, and if a careful examination is made there is no excuse for confounding the two conditions. Hip-joint disease develops more insidiously, is very much more chronic, and rarely produces so great deformity in a year as is often seen in perinephritis in two or three weeks; abscess is infrequent during the first year of the disease; on examination, there is found limitation of all the movements of the joint, and not of extension alone; atrophy of the thigh and joint tenderness are present. In perinephritis, on the other hand, we have a tolerably acute onset, sometimes with chill, fever, marked lameness, and deformity, developing in two or three weeks; abscess often forms in a month, and complete and permanent recovery usually follows after a few months at most; the deformity is due solely to flexion of the thigh; all other movements at the hip may be free, and joint tenderness is absent. Psoas abscess from Pott's disease may cause deformity, tumour, and lameness similar to that seen in perinephritis, but on examination there is found the angular prominence and other signs of disease of the lumbar vertebræ.

Prognosis.—Primary perinephritis in children almost invariably terminates in complete recovery. Of the twenty-eight cases referred to, and eight subsequently observed by Gibney, all recovered perfectly. The only condition liable to prove fatal is rupture of the abscess into the peritoneal cavity.

Treatment.—The patient should be put to bed and kept as quiet as possible throughout the attack. In the early stage, a blister, hot fomentations, or an icebag, should be applied over the affected side; heat is generally to be preferred. When suppuration is inevitable and pain severe, a poultice may be used. Abscesses should be opened early, to prevent burrowing, and danger of a possible rupture into the peritoneal cavity.

GENERAL ŒDEMA NOT DEPENDENT ON RENAL DISEASE.

This is a frequent occurrence in infants and young children. In the Babies' Hospital, at least a score such cases are seen every year. Nearly all are in infants under six months of age, and the majority under three months. This general dropsy is invariably associated with extreme malnutrition and anæmia. It comes on gradually in the course of four or five days, often the first thing noticed being that a wasting child has unexpectedly increased half a pound or a pound in weight. On closer inspection there will be found œdema of the feet, ankles, thighs, face, hands, and sometimes of the abdominal walls, and the back. This may be quite marked, so that it may be almost impossible to open the eyes, and the extremities may be nearly double their normal size. I have occasionally seen dropsy in the serous cavities. No explanation of this œdema is found in the urine. It is not albuminous; it is frequently very scanty, but is sometimes apparently normal in amount. Opportunities for the examination of the kidneys have been afforded in several instances, and these organs have been in all cases normal, even upon microscopical examination.

The cause of this œdema was ascribed by Tarnier, who had observed it in connection with premature infants fed by gavage, to the giving of too much fluid food. He states that it disappeared when the amount of food was reduced. This has not been my experience. Many children who were fed by gavage showed no signs of it, and others who took a comparatively small quantity of food became œdematous. The best explanation seems to me to be that it depends upon a condition of hydræmia, associated with feeble resistance in the walls of the small blood-vessels, through which a transudation of serum readily takes place. The degree of anæmia noted in these patients is sometimes extreme.

The prognosis in this condition is extremely bad, as it rarely occurs except in hopeless cases of marasmus. This is not, however, invariably the case. The dropsy may disappear to return again, or it may disappear permanently and the case go on to recovery.

If the urine is scanty, such diuretics as the citrate of potash and the sweet spirits of nitre often cause a diminution and sometimes even a disappearance of the dropsy in a short time. The best of all remedies, however, is digitalis. To an infant of two months, ♏ $\frac{1}{10}$ of the fluid extract may be given every two hours for two or three days; and for a short period somewhat larger doses may be employed.

CHAPTER III.

DISEASES OF THE GENITAL ORGANS.

MALFORMATIONS.

Adherent Prepuce.—This condition is sometimes called false phimosis. It is so constantly present that it can hardly be regarded as a malformation. It is, however, a condition needing attention in every male infant. The prepuce should be forcibly retracted so as to expose the glans completely. The smegma should then be washed away, the glans covered with a drop of oil, and the skin drawn forward. This should be repeated daily until there is no disposition to a recurrence of the adhesions.

Phimosis.—This is such a narrowing of the prepuce that it can not be retracted over the glans. The degree of phimosis varies greatly. In very rare cases there is no preputial opening. In other cases the orifice is so small that no part of the glans can be exposed, and there is obstruction to the outflow of urine; but usually a small part of the glans can be seen. Phimosis may be complicated by an elongated prepuce (hypertrophic phimosis), and the elongation may exist without any narrowing of the orifice, although this is usually present to some degree.

The presence of phimosis makes cleanliness impossible in many cases, and want of cleanliness leads to infection and to balanitis. This is quite frequent even in infants. It may be complicated by urethritis, and even by cystitis. Another consequence of the straining induced by phimosis is hernia, which may be either inguinal or umbilical. To cure the hernia is often impossible, unless the phimosis is relieved. Straining also leads to prolapsus ani, and, from pressure on the spermatic vessels, to hydrocele. More important even than these mechanical results of phimosis are the reflex conditions resulting from the irritation. Such symptoms may come from preputial adhesions as well as from phimosis. The hyperæsthetic condition and the resulting pruritus cause frequent priapism, and are among the most common causes of masturbation. It may produce other nervous symptoms, such as insomnia, night terrors, etc. Phimosis often causes frequent micturition, dysuria, and, in fact, most of the symptoms of stone in the bladder. It sometimes leads to vesical spasm and retention of urine, but more frequently to nocturnal incontinence.

The list of reflex phenomena which have been attributed to phimosis is a long one, and includes most of the functional nervous diseases of childhood. There is abundant evidence that phimosis may be a cause, although a rare one, of chorea, convulsions, epilepsy, hysterical manifestations, pseudo-paralysis, spasm of the muscles about the hip causing symptoms resembling the early stage of hip-joint disease, strabismus, amaurosis, diarrhœa, and many other nervous conditions. There is, however, no evidence that cases of spastic diplegia or paraplegia are ever caused by phimosis or improved by circumcision. There has been in the past a disposition on the part of some writers to attribute nearly all the nervous disturbances of boyhood to phimosis, and an exaggerated importance has certainly been attached to this condition. Still, in a delicate, anæmic child with unstable nervous centres, phimosis is capable of giving rise to nervous symptoms of a most serious and alarming character. It is an important etiological factor in many neuroses, and one which should not be overlooked. On the other hand, a very marked degree of phimosis often exists in robust children without producing any symptoms whatever.

Treatment.—Every case of phimosis should receive attention in infancy. Often very little treatment is needed; but trouble is likely to come sooner or later if it is neglected. When there is a very long prepuce with phimosis, the operation of circumcision should invariably be done, even when the degree of phimosis is slight. Many cases of phimosis in which the prepuce is not long can be relieved by stretching. If no part of the glans can be exposed, the simplest plan is to slit up the dorsum of the prepuce with a pair of scissors and forcibly break up the adhesions. The corners of the flaps thus made can then be snipped off and one stitch inserted on either side. This is very easily done, and gives most excellent results. In the case of obscure nervous symptoms in older boys, the condition of the prepuce should be examined and the same rules of treatment applied. In all cases of hernia, hydrocele, or prolapsus ani, when phimosis is present it should be relieved as the first step in the treatment.

Hypospadias.—In this condition the urethra is not continued to the tip of the penis, but opens on the inferior surface some distance back, being represented in front of this only by a shallow furrow. In more severe cases there is a deep fissure which divides the scrotum, and sometimes even the perinæum. Into this fissure the urethra opens. This is a condition likely to be mistaken for that of hermaphrodism, especially as the testicles are frequently in the abdominal cavity. It may be impossible to decide the sex of the child until puberty. Surgical operations for the relief of these deformities are not very successful.

Epispadias.—This is a condition in which the urethra opens on the dorsal surface of the penis. It is much less frequent than hypospadias.

There may be simply a division of the glans, or the fissure may extend the whole length of the organ and be complicated by exstrophy of the bladder.

Exstrophy of the Bladder.—In the complete form there is a median fissure from the umbilicus to the tip of the penis. It includes the anterior abdominal wall, the pelvic bones, and the urethra. The bones are entirely separated at the symphysis, or connected behind the bladder by a fibrous band. The hypogastric region is occupied by a red, mucous surface, slightly corrugated, which is all there is of the bladder. In the lower lateral portions of the red mucous membrane two slightly rounded elevations are seen, from which urine oozes. These are the openings of the ureters. The penis is short, and presents a shallow furrow on its dorsal surface. The testes are often in the abdominal cavity.

An analogous deformity is sometimes seen in girls. There is a division of the clitoris and the labia minora and majora. The fissure may be so deep as to reach nearly to the anus. The vagina is usually absent. The rectum may open into the prolapsed bladder.

All these deformities are compatible with long life. In most of them the individual is incapable of procreation. In exstrophy of the bladder, whether complete or partial, patients are a nuisance to themselves and to all about them. It is almost impossible to prevent the clothing from being soaked with urine, which gives everything connected with the patient a strong ammoniacal odour. The skin is often excoriated. Operation for the relief of these cases should, I think, always be undertaken. Brilliant results have been obtained even in some of the most severe cases.

Undescended Testicle—Cryptorchidism.—In fœtal life the testes are situated in the abdominal cavity below the kidneys. They usually descend into the scrotum during the ninth month, but in children born at term the testicles may be in the inguinal canal, or even in the abdomen. The former condition is quite frequent, being present in fully ten per cent of all male children. In most of these the descent takes place without difficulty during the first weeks of life, and causes no symptoms. In others the condition may persist. Spontaneous descent may take place at any time before puberty, the chances, however, steadily lessening as age advances. When in the inguinal canal, an account of its exposed situation, the testicle may be injured, or become painful and tender as puberty approaches. In any abnormal position it probably will not develop properly, and may remain without function, but interference with the development of the body is rare. Hernia is a frequent complication.

When in the inguinal canal, descent of the testicle may sometimes be facilitated by manipulation. If the condition is unilateral, operation is unnecessary except for relief of pain. If it is double, operation should be performed before puberty, preferably in the eleventh or twelfth year. Transplantation into the scrotum is at this time simple, and usually successful. Should pain be persistent, and transplantation impossible, the testicle may be replaced in the abdominal cavity. Removal is indicated only when degeneration has taken place.

With the exceptions already mentioned, deformities of the female genitals belong rather to gynæcology than to pædiatrics, since they are chiefly of the internal organs, and do not usually give symptoms before puberty.

DISEASES OF THE MALE GENITALS.

Balanitis.—Balanitis, or inflammation of the prepuce, is one of the results of phimosis. It may follow decomposition of the smegma, infection of the mucous membrane, injury, or masturbation. The parts are swollen, œdematous, red, painful, and sometimes bathed in pus. Retraction of the prepuce is impossible. Under proper treatment the inflammation usually subsides in two or three days, but there may be some discharge for a considerable time. Abscess may follow, and even gangrene of the prepuce. The most severe cases are likely to be complicated by anterior urethritis. I have frequently seen erysipelas start from balanitis, and occasionally diphtheria occurs here.

The object of treatment is to remove the irritating and infectious material lodged beneath the foreskin. This may be quite difficult. It is best accomplished by syringing with a 1-to-5,000 bichloride solution, and the constant application of a wet antiseptic dressing. Ice is often useful where the œdema is great. It is sometimes necessary to slit up the prepuce before the parts can be thoroughly cleansed, and in severe cases this is often the quickest method of cure. Circumcision should not be done during an attack.

Urethritis.—This, like the same disease in females, may be simple or specific. Both forms are less frequent in little boys than in the other sex. In simple urethritis the inflammation usually affects only the anterior part of the canal, the fossa navicularis. There is a slight discharge of pus, and sometimes pain on micturition. The most frequent cause is want of cleanliness.

Gonorrhœal inflammation is more common. This occurs even in boys as young as eighteen months, but most of the cases are in those over seven years old. The usual cause is direct contagion. The symptoms are more severe than in the simple form, and resemble the same disease in the adult, with the exception that constitutional symptoms are usually absent. A microscopical examination of the discharge is the only positive means of diagnosis between the two varieties. In these cases it reveals the gonococcus in great numbers. Conjunctivitis and arthritis are seen as complications, just as in the female. Orchitis is very rare, but balanitis and bubo are not infrequent. Poynter has reported a case in a boy of three years, who, when five years old, required treatment for a urethral stricture. He was infected by a nurse.

The first thing in the treatment is always to keep the parts covered, otherwise the infection is almost certain to be carried by the hands to other mucous membranes, usually the conjunctiva. In other respects the treatment is the same as in the adult.

Hydrocele.—Hydrocele consists in an accumulation of serum in some part of the serous pouch brought down by the testicle in its descent. In infants it is usually due to the imperfect closure of this pouch at some point, where a fluid accumulation occurs. Four varieties of hydrocele are met with in young children:

1. *Congenital hydrocele.*—In this the condition is a congenital one, although the tumour is not necessarily present at birth. The tunica vaginalis communicates with the general peritoneal cavity. There is present an elongated tumour, extending from the bottom of the scrotum throughout the whole length of the cord. The tumour is reducible, sometimes spontaneously by position, sometimes, when the opening is smaller, only by pressure. It reduces slowly, without gurgling, never going back *en masse* like a hernia. The tumour is translucent, and is flat on percussion. The testicle is above and posterior, and usually indistinctly felt. Congenital hydrocele may be complicated by hernia.

2. *Hydrocele of the tunica vaginalis with the canal closed.*—In this form the accumulation of fluid is in the scrotum, communication with the peritoneal cavity having been entirely cut off by the complete obliteration of this pouch in the canal in the normal way. This is one of the most frequent forms. It gives rise to an oval or pear-shaped tumour, quite tense and firm, usually about two inches in length. The cord is distinctly felt above it, the testicle is behind and somewhat above it, and not always felt very distinctly. This variety gives translucency and the usual elastic feeling of a hydrocele.

3. *Hydrocele of the cord.*—This is one of the rare forms. The serous pouch which accompanies the spermatic cord is open above, and communicates with the peritoneal cavity; but below it is closed. The scrotum is normal, and the testicle is in its usual position. The tumour is small, elongated, and reducible, and entirely above the scrotum. Usually it stops at some point in the inguinal canal. This hydrocele also may be complicated by hernia. The diagnostic points are the same as in the form first mentioned.

4. *Encysted hydrocele of the cord.*—The peritoneal pouch of the cord in this variety is closed for some distance above, and again below, but somewhere in its course it is open, and here the fluid accumulates in the form of a cyst. When small it resembles an undescended testicle; but on examination the testicle is found below and in its normal position. When in the canal it is often mistaken for a lymph gland, sometimes for a small hernia. The tumour is usually about the size of an almond. It is elastic and irreducible, and translucent like the other varieties. In cases of doubt it may be punctured by a hypodermic needle.

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per cent of his cases that the mother had a leucorrhœal discharge. The mode of contagion may be difficult to trace, but this fact should cast no doubt upon the diagnosis.

Symptoms.—In infants and young children, in the mild cases the disease is limited to the mucous membrane of the vagina. There is a moderate yellow discharge which, by microscopical examination, contains pus cells and gonococci. There is little redness and no symptoms of discomfort. In more severe cases the discharge is copious, often thick and of a yellow or yellowish-green colour. It may be tinged with blood from slight erosions. It often causes excoriation of the labia or thighs. In many cases, but by no means in the majority, the urethra is involved, causing frequent, painful micturition. Less frequently the inflammation extends to the bladder, but seldom or never at this age to the mucous membrane of the uterus. The symptoms are chiefly local, but there may be a slight rise of temperature to 100° or 101° F. during the period of most acute inflammation.

In girls past the age of six or seven years, the symptoms resemble those of the adult: copious secretion, the formation of crusts on the labia, frequent, painful micturition from involvement of the bladder and urethra, and difficulty in locomotion. There may be slight fever and general malaise. The inflammation may extend to the lining membrane of the uterus and, through the Fallopian tubes, to the pelvic peritonæum. Sanger has reported such a case in a child of three years. The endometritis may be demonstrated by the use of a small speculum, by which the discharge may be seen coming from the cervix. Swelling, and very rarely suppuration, of the inguinal glands may take place.

A positive diagnosis between simple and gonococcus vaginitis can be made with certainty only by a microscopical examination of the discharge, though in default of such examination an abundant purulent catarrh should be assumed to be due to the gonococcus until the opposite is proved. In simple catarrh the discharge is made up of epithelial and pus cells, with quite a wide variety of bacterial forms, chiefly cocci and bacilli, occasionally a few diplococci. In gonococcus vaginitis the gonococci are found in large numbers, and are usually the only bacteria present. To be diagnostic, they should be demonstrated within the pus cells as well as outside them. The gonococcus decolourizes when stained by Gram's method, which fact distinguishes it from the other organisms likely to be present in the vagina. The staining is quite as diagnostic as the cultural characteristics of this organism. Cases of vaginitis are to be regarded as suspicious if pus is found and few organisms are detected; in such conditions subsequent examination usually reveals the gonococcus. In my hospital experience the gonococcus cases have outnumbered the simple purulent forms, fully ten to one.

In infants, where the amount of discharge is small and likely to be overlooked, it is an advantage to apply between the labia a fold of gauze upon which the yellow stain of a purulent discharge is readily noticed, which might otherwise escape observation.

Gonococcus vaginitis may be complicated by conjunctivitis, arthritis, endo- or pericarditis, peritonitis, and proctitis. Conjunctivitis is the most frequent, the infection usually being carried by the hands. Gonococcus arthritis is not uncommon even in young infants. It is usually a multiple arthritis, with the constitutional symptoms of pyæmia. The wrist, ankle, knee and elbow, and small joints of the fingers and toes are most frequently involved. These cases are considered more fully in the chapter on Acute Arthritis in Infants.

The diagnosis in all the complicating conditions is based upon the presence of the gonococcus.

Prophylaxis.—The highly contagious character of gonococcus vaginitis makes it imperative that such cases should not be received into the same ward or dormitory with other children. Only in this way can house epidemics be prevented. Cases which are mild should be excluded, as well as those which are severe. The only effective measure is to make the microscopical examination of vaginal discharges of children admitted to an institution as much a matter of routine as the taking of throat cultures if there is a tonsillar exudate. Cases showing the gonococcus should be quarantined or excluded. When there are a great many admissions every month, a case occasionally escapes detection. The rule which we have followed in the Babies' Hospital has been to make not only an examination on admission, but routine examinations of all patients at stated intervals. Only by this means has it at times been possible to eradicate the disease.

The attendants, both day and night nurses, as well as the children, should be quarantined. Napkins, underclothing, and sheets from the beds of infected children, also towels and wash-cloths, should not go into the common laundry, but should be first soaked in a strong solution of carbolic acid, and afterward boiled. All articles connected with the children's toilet, also syringes, thermometers, etc., should be carefully disinfected. The organism is one that is fairly easy to kill, and if proper precautions are taken epidemics may be prevented. The essential measure is a prompt recognition and isolation of the first case in the hospital. Quarantine should continue not only until the catarrhal inflammation has subsided and the organism has disappeared, as shown by a single negative microscopical examination, but for a considerable time longer, since a slight discharge containing a few organisms may remain for weeks after the case is considered cured. Relapses are very frequent.

Treatment.—Cases of simple vaginal catarrh should be irrigated twice daily with a warm saturated solution of boric acid or 1 to 5,000 bichloride. Cleanliness should be secured by frequent bathing and the skin protected by ointments. In more severe cases, astringent injections, such as sulphate of zinc or tannic acid, should be used, or protargol applied in solutions of from one to five per cent strength. The general health should be built up by iron, cod-liver oil, and other tonics.

In gonococcus vaginitis more energetic treatment is necessary. Every child should wear a napkin, to prevent carrying the infection to the eyes by means of the hands. Irrigations should be used at least twice a day, and stronger antiseptics employed than in the simple cases. The best are protargol, in solutions from one to ten per cent strength, and argyrol, in solutions from five to twenty-five per cent strength. Applications should be made with a cotton swab; the same substances may be used in the form of suppositories, or the vagina may be packed with gauze wet in these solutions. The closest attention to cleanliness is required in all cases. This disease is very tedious; many weeks, and often months, may be required for a cure. On the whole, treatment is very unsatisfactory on account of the difficulties in the way of making thorough local applications. When the disease involves the bladder and urethra, the same general measures as in adults are indicated.

GANGRENOUS VULVITIS (NOMA).

This is the same process as that seen in the mouth and known as cancrum oris. It usually follows one of the infectious diseases, most frequently measles, occurring in patients whose general vitality has been greatly reduced. There is first noticed a tense, brawny induration, the skin being shiny and swollen over a circumscribed area. In the centre of this there soon appears, usually upon one of the labia majora, a dark, circumscribed spot. Day by day the gangrenous area advances, preceded by the induration. It may involve the whole labium, extending even to the mons veneris and the perinæum. These cases are generally fatal. If recovery takes place, it is with considerable deformity of the parts in consequence of the extensive sloughing and cicatrization. As sequelæ, there may be fistulæ, stenosis, or atresia of the vagina. The prognosis is very bad. The only radical treatment is early excision, and the application of the actual cautery, carbolic or nitric acid.

CHAPTER IV.

ENURESIS.

Synonyms: Incontinence of urine; bed-wetting.

Enuresis may be due to some malformation of the genital tract, such as an abnormal opening of the bladder into the vagina, to extroversion of the bladder, or to the persistence of the urachus; in the latter case the urine is discharged from the umbilicus. It also occurs in organic diseases of the central nervous system, such as idiocy, cerebral palsy, acute meningitis, tumours of the brain, certain forms of myelitis, and in injuries of the cord. In many of these conditions there is associated incontinence of fæces. Both of the groups of cases mentioned are quite distinct from the ordinary form of incontinence of urine which is seen in childhood. The latter is to be regarded as a neurosis, and is the only variety which will be considered here.

It is in many cases possible to teach infants to control the evacuation of the bladder before the end of the first year; usually, however, control is not acquired even during waking hours until some time during the second year, and in some healthy infants not before the end of the second year. The time depends very much upon the training. If a child during its third year can not control the evacuation of the bladder during its waking hours, incontinence may be said to exist.

Etiology.—Incontinence of urine may be due to a continuance of the infantile condition, to anything which increases the irritability of the spinal centre, or which interferes with the cerebral control over this centre, or to anything which increases the irritability of the terminal filaments of the vesical nerves or of those in the neighbourhood. The causes of incontinence thus may be in the central nervous system, in the urine, in the bladder, or in any of the adjacent organs.

The causes relating to the central nervous system are in the main those of the other neuroses of childhood; these are anæmia, malnutrition, an inherited nervous constitution, or a condition of extreme nervousness or neurasthenia, the result of the child's surroundings. In such cases incontinence is often associated with chorea, epilepsy, hysteria, headaches, neuralgia, and other nervous symptoms. In these conditions there may be not only an increased irritability of the nerve centres, but also of the peripheral nerves, accompanied by loss of tone of the vesical sphincter. A similar condition may exist with almost any form of acute illness, usually, however, being only temporary.

Incontinence may be caused either by a highly acid, concentrated urine where an insufficient amount of fluid is taken, or to the opposite condition, where, owing to the drinking of a large quantity of water, often only a matter of habit, the amount of urine is very greatly increased and passed at frequent intervals.

In the bladder itself, cystitis and vesical calculus, although infrequent, should not be overlooked as possible causes. In a few cases, where incontinence has existed a long time, the bladder becomes so contracted that it will hold only an ounce or two of urine. This condition, although not the primary cause of enuresis, may be enough to continue it.

Local irritation in the neighbouring organs may be due to adherent prepuce, balanitis, phimosis, or to a narrow meatus. All of these conditions are frequently associated with incontinence. Rectal irritation may be due to pinworms, anal fissure, or rectal polypus; and vaginal irritation to vulvo-vaginitis or adherent clitoris; but these are rarely the only cause. Often we have incontinence as the result of a combination of several causes, no one of which alone would have been sufficient to produce it. Thus, in a healthy child phimosis may give rise to no symptoms, while in one who is anæmic or neurasthenic it may produce enough local irritation to cause incontinence. In many cases heredity seems to be a factor of some importance, parents often having suffered in their childhood from the same condition; quite frequently two and sometimes even three children in the same family are affected. In many cases the condition seems to be mainly the result of habit, and in all cases habit is a potent factor in continuing the incontinence, sometimes after the original exciting cause has been removed. Frequently no adequate cause can be found. Both sexes are about equally liable to enuresis, and it may be seen in all ages up to puberty.

Symptoms.—Enuresis may be nocturnal or diurnal, or both. Of 184 cases, 73 were nocturnal, 9 diurnal, and 102 were both nocturnal and diurnal. Cases differ greatly in severity. Incontinence may be habitual, occurring every night, often several times during the night, and frequently during the day; or it may be only occasional under the influence of some special exciting cause, where it continues a few days or weeks until the cause is removed. In a considerable number of cases, the condition lasts from infancy until the sixth or seventh year. It may even continue until puberty; but it generally ceases at that period, unless its cause is mechanical, or depends upon some organic disease of the brain or cord. In ordinary enuresis there is never dribbling of the urine, but usually a contraction of the walls of the bladder follows almost immediately upon the desire before the patient can make his wants known or reach a convenient place for micturition. At night the same thing may occur without wakening the child, the contraction being of purely reflex origin.

Prognosis.—The condition is usually hopeless when it depends upon organic disease of the brain and cord; also in cases due to malformation, unless these are amenable to surgical treatment. In the ordinary cases seen, the prognosis depends upon the age of the child, the duration of the symptom, and the nature of the exciting cause. In children of from three to five years a cure can usually be accomplished with proper management. Those who are older are much less amenable to treatment, especially if the condition has persisted since infancy; but if the incontinence has lasted only for a year or so, the outlook is much more encouraging. When some cause can be discovered which can be removed, the prognosis is better than if none can be found. There are, however, some cases in which no other cause than habit can be discovered which resist all treatment, the condition finally ceasing spontaneously at or a little before puberty; in very few does it continue beyond this period.

Treatment.—The first indication is to remove the cause, where one can be found. If there are preputial adhesions, they should be broken up and irritating smegma removed. If phimosis is present, it should be relieved by stretching or circumcision. A narrow meatus should be cut to proper dimensions. If stone in the bladder is suspected, as it should be when the incontinence is worse by day and accompanied by straining and painful spasm of the bladder, the patient should be sounded for stone. Pinworms in the rectum should receive the appropriate treatment by injections. While the local conditions mentioned should always be attended to, the fact remains that few cases are cured simply by relieving them, except those due to vesical calculi. The explanation of this is that habit is so important a factor in keeping up incontinence where it has existed a long time.

A concentrated urine of high acidity with deposits of uric acid, is an indication for alkalies and the free use of all fluids, especially water. On the other hand, when there is passed a large quantity of urine with low specific gravity, the amount of water and other fluids should be restricted. During the night, water should be forbidden and the amount given in the latter part of the day greatly reduced. In these cases the incontinence is often simply the result of the polyuria, which in turn depends upon polydipsia.

To institute a proper general régime should be the next step in the treatment. Care should be taken to secure for the child a simple, natural life, preferably in the country. There should be no overtaxing of the nervous system at home or in school. Every cause of unnatural excitement should be avoided. Early hours and plenty of sleep must be insisted upon. Certain articles of diet are to be avoided, and coffee, tea, and beer should be absolutely prohibited. Sweets and all highly seasoned food should be very sparingly allowed, or not at all. Although it is believed by many that a diet into which meat enters largely is injurious, from personal experience I have not found the exclusion of meat to be of any advantage. The diet which succeeds best is a simple one composed of milk, vegetables, fruits, meats, and cereals. With most patients who have nocturnal incontinence, it is well to allow fluids freely during the early part of the day, but little or none after 3 or 4 P. M., a dry supper being given just before retiring. The child should be taught to hold his water as long as possible during the day, to accustom the bladder to full distention.

Measures directed toward improving the general muscular and nervous tone are of the greatest importance, and they are required in most cases, excepting the very young children. It should be remembered that incontinence of urine is a neurosis, depending like most neuroses of childhood upon disturbed nutrition. Anæmia, chlorosis, malnutrition, indigestion, and constipation should each receive careful attention. Any local condition, such as adenoid growths of the pharynx, which might serve to increase the general nervous irritability, should be removed.

The moral treatment of the case is important. One should work upon the child's pride and use every possible means to strengthen his will. Punishments whether corporal or otherwise do little good, and with most children they are absolutely harmful. With children in whom incontinence is chiefly a matter of habit, I have often found rewards more efficacious than any other means of treatment. One should first find out what it is that the child desires most—a new doll, a bicycle, a pony—and allow him to have it if his bed is dry, taking it away if it is wet. A reward of five cents for every dry night sometimes works marvels.

The measures described—removal of local causes, building up of the general health, institution of a proper régime, and mental and moral means—in a very considerable number of cases suffice for a cure. They generally constitute the most important part of the treatment, and their value is not sufficiently appreciated. Personally I have found these means more effective than the use of specific drugs. Drugs are useful as accessories, but alone seldom accomplish a cure. Of those employed belladonna is certainly the most effective, but its administration should be continued for a long time. Atropine either in solution or in tablet form is the most convenient method of administration. For nocturnal incontinence, $\frac{1}{1000}$ of a grain for each year of the child's age up to seven years, is a suitable initial dose. A child of five would thus be taking $\frac{1}{200}$ of a grain. At first, a single dose should be given at bedtime; after a few days a second dose may be given three or four hours earlier. To push the drug much further than this, causes much discomfort and is of doubtful advantage. After the condition is under control, the same dose should be continued for some time and then reduced, the atropine being given for at least two months in gradually diminishing doses after the incontinence has ceased. This is very important if the cure is to be permanent, as there is so strong a tendency in these cases to relapse.

Strychnine may be added in cases not yielding to the atropine alone. It is particularly advantageous when there is diurnal as well as nocturnal incontinence, for under these conditions there is usually a lack of tone in the sphincter, as well as increased irritability in the mucous membrane of the bladder. The initial dose for a child of five years should be $\frac{1}{100}$ of a grain twice daily; this may be gradually increased to $\frac{1}{50}$ of a grain three times a day; but there is rarely any advantage in pushing it further. Ergot is sometimes useful in conjunction with other drugs, but rarely gives relief when both strychnine and atropine have failed. Some obstinate cases are reported to have been relieved by faradism; the positive pole is attached to a small electrode passed into the rectum and the negative pole applied over the bladder. The sitting should last for ten minutes and be repeated three times a week. My own experience with this method of treatment has been disappointing. If there is reason to suspect a contracted bladder, as when the incontinence has lasted for years and the bladder will never hold more than an ounce or two of urine, cure is sometimes accomplished by daily distending the organ up to its normal capacity with warm water.

Careful, intelligent, systematic training is a most valuable adjunct to all measures employed for the relief of this very annoying condition.

VESICAL SPASM.

This is quite a common condition, and often passes under the name of *genital irritation*. It is characterized by frequent, sometimes by difficult and painful, micturition. It occurs in children of all ages, even in infants, but is especially frequent between the ages of two and five years. This symptom has already been referred to in connection with uric-acid infarctions in very young infants.

The usual cause is the irritation of the bladder by a concentrated, highly acid urine. It often results from cold; it may accompany acute febrile processes, and is sometimes merely a symptom of nervous irritability. The cause may thus be in the bladder or in the urine. It may be accompanied by enuresis, but usually occurs without it. It is sometimes symptomatic of disease in adjacent parts, as in the rectum or the pelvic peritonæum, or it may be associated with inflammation of the vulva or urethra. It is also one of the symptoms of vesical calculus.

The *symptoms* of vesical spasm are local only. The child passes water very frequently, often several times an hour. The accompanying pain may be intense, not infrequently sufficient to cause the child to cry out. Often there is pain and severe vesical tenesmus with the passage of only a few drops of urine at a time, but blood is not present. If the condition depends upon the character of the urine, or is only an expression of an extreme vesical irritability, the symptoms are generally of short duration, possibly a day or two. If it depends upon vesical calculus, it may be intermittent. If it is associated with disease of the adjacent pelvic viscera, it is inconstant, and may continue for a considerable period, depending upon the nature of the cause.

The *treatment*, in the ordinary cases, consists in the administration of an abundance of water, with alkaline diuretics, and either belladonna or hyoscyamus. The following formula is one that I have usually found efficient:

℞	Tincturæ hyoscyami	ʒ ss.
	Potassii citratis..	ʒ j
	Aquæ destillat	℥ ij

M. Sig.: Half a teaspoonful in water every hour to a child of two years.

If the cause is outside the bladder, it should receive appropriate treatment.

VESICAL CALCULI.

The nucleus of a vesical calculus is usually a renal calculus which has passed the ureter, but has been prevented by its size from going farther. Stone in the bladder is extremely rare in infancy, probably owing to the fluid diet, but it is not infrequent in children from two to ten years of age. The most common variety of calculus at this time is the uric acid. The other forms, although occasionally seen, are all quite rare.

The symptoms in children are somewhat different from those in adults, and the condition is often overlooked. There is frequently pain upon micturition, especially at the close of the act, which may be felt at the end of the penis or in the perinæum. There may be a sudden stoppage in the flow of urine. The straining often leads to rectal tenesmus and even to prolapse. This complication is so frequent that, in a case of persistent prolapse, stone should always be suspected. Incontinence of urine is a prominent, and often the principal symptom; in many cases it is noticed only during the day. The urinary changes are not generally marked; hæmaturia is rare, and mucus and pus are infrequent and in small quantity. The genital irritation may lead to the habit of masturbation. A stone of any considerable size may often be felt by a bimanual examination, one finger being placed in the rectum and the other hand above the pubes. This is easier in males than in females, but it is not very trustworthy, and not conclusive when it gives a negative result. A positive diagnosis is made only by exploring the bladder with a sound.

The treatment of calculus is purely surgical. In young children the suprapubic is now generally preferred by surgeons to the perineal operation, if the calculus is too small to be easily removed by crushing.

SECTION VII.

DISEASES OF THE NERVOUS SYSTEM.

CHAPTER I.

INTRODUCTORY.

The Weight of the Brain.—From ninety-eight observations made in the post-mortem room of the New York Infant Asylum, the following were the average weights noted:

At three months	21 oz. (602 grammes).
At six months	25½ " (712 ").
At twelve months	32½ " (916 ").
At two years	35 " (990 ").

The following are the figures given by Boyd and Schäfer:*

Age.	Males.		Females.	
	Ounces.	Grammes.	Ounces.	Grammes.
At birth (full term)	11½	330	10	283
Under three months	17½	493	16	451
From three to six months	21	602	20	560
From six to twelve months	27	776	26	727
From one to two years	33	941	30	843
From two to four years	39	1,095	35	990
From four to seven years	40	1,138	40	1,135
From seven to fourteen years	46	1,301	40½	1,154
From fourteen to twenty years	48½	1,374	44	1,244

At birth the weight of the brain to that of the body is nearly 1 : 8. During infancy and childhood the following is the ratio, according to Bischoff: during the first year, 1 : 6; the second year, 1 : 14; the third year, 1 : 18; at the fourteenth year, 1 : 15 to 1 : 25; in adults, 1 : 43.

The Spinal Cord.—The weight of the cord to the weight of the body at birth is 1 : 500; in adult life it is 1 : 1500. According to Kölliker, the spinal cord and the vertebral column are the same length until the end of the third month of fœtal life, there being at this time no cauda equina. At the ninth month the lower end of the cord is opposite the third lumbar vertebra; in the adult it is opposite the first.

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* Quoted by Sachs.

Some Peculiarities in the Diseases of the Nervous System in Infancy and Childhood.*—The relatively large size, the rapid growth, and the immaturity of the brain and cord during early life, explain much that is peculiar in the nervous diseases of this period.

At this time, apparently trivial causes are enough to produce quite profound nervous impressions, because of the instability of the nervous centres and the greater irritability of the motor, sensory, and vaso-motor nerves. These are conditions which are very much increased by all disturbances of nutrition. These disturbances may be manifold in character, but they lie at the root of very many of the neuroses of early life,—e. g., extreme nervousness, disorders of sleep, stuttering, chorea, incontinence of urine, tetany, and convulsions. The great liability to convulsions depends not only upon the greater irritability of the peripheral nerves, but on the instability of the nervous centres and the lack of inhibition over the motor ganglion cells of the spinal cord. The nervous centres are more easily exhausted than later in life. Prolonged or continuous overstrain from any cause whatsoever, frequently leads to headache and chorea, and sometimes even to epilepsy and insanity.

Another peculiarity is the serious consequences which often follow reflex irritation, although this is rarely the only factor in the case. Conditions which in adult life produce almost no effect may in infancy be the cause of most alarming symptoms. As a few examples may be cited, reflex symptoms due to phimosis or intestinal worms, convulsions from disturbances of digestion, nervous symptoms due to eye-strain, or to adenoid growths of the pharynx. In the production of some of these, especially attacks of convulsions, there are several factors, such as the great irritability of the peripheral nerves, the instability of the nervous centres—often a result of disturbed nutrition, as in rickets—and the lack of inhibitory action of the cortex of the brain.

As a third point of importance may be mentioned the grave permanent results which often follow relatively small organic lesions. A good illustration is seen in the lesions which produce cerebral birth-palsy. Here the damage is only in small part the immediate effect of the hæmorrhage, for this often is not great, but it is the interference with the development of certain parts of the cortex that makes this condition so serious.

From what has been said, it follows that the hygiene of the nervous system is of the utmost importance in infancy and childhood. It is essential for the healthy development of the nervous system that all stimulants should be avoided,—not only tea, coffee, and alcohol, but undue and unnatural excitement, the effect of which in infancy is almost as serious. A normal development can take place only in the midst of quiet and peaceful surroundings, with plenty of time for rest and sleep. The conditions of modern life, especially in cities, are such that these laws are almost invariably violated, and the consequences of this are seen in the marked and steady increase in nervous diseases among children.

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* See Rachford; Some Physiological Factors in the Neuroses of Childhood. Cincinnati, 1895.

CHAPTER II.

GENERAL AND FUNCTIONAL NERVOUS DISEASES.

CONVULSIONS.

UNDER this head are included attacks of acute transient nervous disturbance, characterized by involuntary rhythmical spasm of the muscles, either of the face, trunk, or extremities, or all of them, usually accompanied by loss of consciousness. They may be regarded as "motor discharges" from the cortex of the brain.

Etiology.—The principal predisposing causes are infancy, conditions affecting the nutrition of the brain, and hereditary influences. Of all these factors, the most important one is the instability of the nerve centres which is characteristic of infancy and is associated with the non-development of the voluntary centres of the cortex. The brain grows more during the first year than in all later life, and this rapidity of growth is in itself an important predisposing cause of functional derangement. After infancy, attacks of convulsions are much less frequent, and after seven years they are relatively rare. While convulsions occasionally occur in children previously healthy, the majority of attacks are in those in whom there is at least some disturbance of the nutrition of the brain,—the cerebral instability of infancy being greatly exaggerated by such nutritive disorders. The most frequent one is rickets, which may be regarded as altogether the most important predisposing cause of infantile convulsions. They are often one of the earliest symptoms of that disease, and where convulsions occur in infancy without evident cause, rickets should always be looked for. Any disturbance of nutrition may predispose to convulsions, such as exhaustion, anæmia, malnutrition, syphilis, and debility resulting from any acute disease, especially those of the digestive tract. Children who inherit from their parents a peculiarly nervous temperament are more liable to convulsions than are others. This predisposition is often seen in several members of the same family. Females are rather more frequently affected than males.

The exciting causes include a wide variety of pathological conditions, among which disturbances of digestion take the first place. Where the susceptibility is very great, the exciting cause may be a trivial one. These causes may be grouped under three general heads: (1) direct irritation of the cortex of the brain; (2) reflex irritation; (3) toxic influences.

Under the head of direct irritation may be included all convulsions occurring with the various forms of cerebral disease; the most frequent are meningitis, meningeal or cerebral hæmorrhage, tumour, abscess, hydrocephalus, embolism, and thrombosis. As examples of reflex irritation may be classed the convulsions following severe injuries, like compound fractures or burns, renal or intestinal colic, retention of urine, phimosis, a foreign body in the ear, or intestinal strangulation. A case has been related to me in which the application of cold to the skin repeatedly induced convulsions. Other conditions classed under this head are dentition and worms, but both must be regarded as exceedingly rare causes of convulsions. The exciting cause is very frequently the presence in the stomach or intestines of undigested food; such attacks are sometimes ascribed to reflex irritation, but the majority are better regarded as toxic. Acute and chronic indigestion are to be ranked among the most frequent causes of convulsions, both in infants and older children. In either there may be but one attack, or attacks may recur at intervals of a few months with a repetition of the cause. Of toxic origin may be considered not only the convulsions resulting from conditions like uræmia and asphyxia, but also those which occur at the onset or in the course of various infectious diseases, sometimes classed as febrile convulsions. They are very frequent at the onset of certain diseases, particularly pneumonia, scarlet fever, malaria, acute indigestion, and gastro-enteric intoxication; less frequently measles, typhoid fever, ileo-colitis, and diphtheria. In these cases the convulsions seem due partly to the intensity of the poison and partly to the suddenness with which it affects the nervous system. Convulsions occurring late in the course of many diseases may be due to toxic influences, especially when associated with exhaustion of the nerve centres, from the prolonged disturbances of nutrition accompanying the febrile condition.

In pertussis, which of all infectious diseases is the one in which convulsions are most frequent, several factors may be present: asphyxia due to a severe paroxysm, cerebral congestion or hæmorrhage resulting from such a paroxysm, or simply from the peculiar susceptibility of the patient brought about by the disease itself.

Convulsions may be associated with enlargement of the thymus gland. I have seen several fatal cases of convulsions where there was found at autopsy great enlargement of the thymus, which weighed from one to one and a half ounces. Some of these infants were previously healthy; some were rachitic. The similarity of all these cases indicated that the convulsions were in some way due to the enlarged thymus, possibly from pressure either upon the lungs, the large vessels, or the pneumogastric nerves, or in some other way, not yet understood.

There are some cases of convulsions for which no cause can be discovered even at autopsy, and for the present we must be content to class them as idiopathic. One attack of convulsions renders the patient more liable to a second, and where there have been several, they occur from causes which are less and less marked.

Pathology.—The "nervous discharge" which occurs in an attack of convulsions differs in no essential particulars from that of ordinary epilepsy. In the latter disease there is seen a tendency to recurrence with greater or less frequency, until the discharge may take place from very slight causes.

The part of the brain most intimately concerned in the production of convulsions is the cortex. Such attacks may be regarded as involuntary discharges of nerve force from the cortical motor centres, which result from direct irritation of these parts by disease; or from an irritation arising in some other part of the brain, as from the vaso-motor centres of the medulla; or from a reflex irritation in a distant part of the body. Convulsions may depend upon the fact that while nerve cells may be able to generate nerve force they can not control its discharge, as in the convulsions of rickets. An important element in the convulsions of infancy, according to Hughlings Jackson, is the lack of development of the higher cerebral functions, in consequence of which they do not exert the controlling influence over the discharge of nerve force which they do in later life.

The condition of the brain in the beginning of an attack of convulsions is one of anæmia; this is shortly followed by venous hyperæmia which may be very intense. In infants who die during convulsions the brain and its meninges are usually found intensely congested. They may be the seat of punctate hæmorrhages, and sometimes of more extensive ones. The lungs are also deeply congested, and the right heart is generally distended with dark clots. The other lesions found are accidental.

Symptoms.—In some cases prodromal symptoms are present, such as extreme restlessness, irritability, slight twitchings of the muscles of the face, hands, feet, or eyelids. More frequently, however, the attack comes quite suddenly with but momentary warning. Usually the first thing noticed is that the face is pale, the eyes fixed, sometimes rolled up in their orbits; in a moment or two convulsive twitchings begin in the muscles of the eye or face, or in one of the extremities, which usually rapidly extend until all parts of the body participate. In most cases the convulsions become general, but they may, however, remain unilateral even when not due to a local cause,—a point which is often forgotten. The contraction of the facial muscles causes a succession of grimaces; the neck is thrown back; the hands are clenched; the thumbs buried in the palms; and a quick spasmodic contraction of the extremities occurs. There may be some frothing at the mouth, and in all true convulsions there is loss of consciousness. Respiration is feeble, shallow, and may be spasmodic. The pulse is weak; it may be slow or rapid; often it is irregular. The forehead is covered with cold perspiration. The face is first pale, then becomes slightly livid, especially about the lips. Unnatural rattling sounds may be produced in the larynx. The bladder and rectum may be evacuated. The convulsive movements consist in an alternation of flexion and extension recurring rhythmically. All varieties of tonic and clonic spasm may be seen, and in all degrees of severity. The contractions of the two sides of the body are usually synchronous. After a variable time, from a few moments to half an hour, the convulsive movements are gradually less frequent and finally cease altogether, usually leaving the patient in a condition of stupor. They may recur after a short time or there may be but one attack. A period of general relaxation usually follows the convulsive seizures, frequently accompanied by marked evidences of prostration. Transient paralysis, apparently due to exhaustion of the nerve centres, is not an uncommon sequel.

Death may take place from a single attack; this, however, is rare except in very young infants, especially those who are rachitic. There may be no sequel to the convulsions if the cause is a temporary one, or they may produce some serious brain lesion, particularly meningeal hæmorrhage. Death from convulsions is generally due to asphyxia, or to exhaustion from the rapidly recurring attacks. Many cases recover in which the children for several minutes had the appearance of being moribund.

One attack of convulsions is very apt to be followed by others; for the occurrence of the first one usually reveals a peculiar susceptibility of the nervous system, and each succeeding attack comes from a less powerful exciting cause than the previous one. The longer the interval which has passed, the less likely is there to be a repetition, especially if the child has passed its third year. The number of attacks may be very great. In a case under the care of Dr. A. M. Thomas and myself in 1896, an infant during the latter part of its second year had during six months over thirty-five hundred distinct attacks of convulsions. For a considerable period they reached the almost incredible number of eighty a day, and yet the mental condition of the child in the interval was apparently normal.*

Diagnosis.—There can rarely be any difficulty in recognising an attack of convulsions. The difficulty consists in determining with which of the many possible exciting causes we have to do in the case before us. Is it epilepsy? Does it depend upon cerebral disease? Does it mark the onset of some other acute disease? Is it reflex, and if so to what is it due? To answer these questions a careful history must be obtained, and all the circumstances surrounding the patient, the character of the convulsions, and all the other symptoms present must be taken into consideration.

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* The microscopical examination of the brain showed only degenerative changes in the nerve cells of the cortex in the motor area and an increase in the neuroglia. These changes existed over quite an extensive area, and were more marked upon one side.

In infancy, epilepsy is certainly the least probable diagnosis. In older children the most important points indicating that disease are: the presence of some of the stigmata of degeneration, a history of previous attacks, a distinct aura preceding the seizure, or a sudden onset with a cry or fall, biting of the tongue, a tonic spasm preceding the clonic, and, finally, perfect recovery in the course of a few hours after the attack. Convulsions which come on with high fever, even though a patient may have repeated attacks, are seldom epileptic. However, in some cases only prolonged observation can enable one to decide positively whether or not epilepsy is present.

Convulsions occurring in brain disease, except acute meningitis, are not as a rule accompanied by any marked rise in temperature. Focal symptoms are often present, such as localized paralysis or rigidity, changes in the pupils, and strabismus. The convulsive movements are frequently limited to one side of the body. It should, however, be borne in mind that unilateral convulsions, even when repeated, do not always mean a local lesion, as I have seen proved by autopsy more than once. In hæmorrhage or meningitis, convulsions are likely soon to recur. In tumour they may recur after a longer interval.

Convulsions may be thought to indicate the onset of some acute disease when they occur in a child over two years old, and when they come on suddenly or with only slight premonition in a child previously well; but the most important point is that they are accompanied by a high temperature,—104° to 106° F. Acute meningitis is the only other condition likely to produce these symptoms. Whether the convulsions mark the onset of lobar pneumonia, scarlet fever, malaria, or some other disease, can be determined only by carefully watching the patient's symptoms for twenty-four or thirty-six hours.

In convulsions depending upon some disorder of the alimentary tract, one may get a history of chronic constipation or improper feeding, and in nursing infants sometimes of passion or intoxication in the wet-nurse. Convulsions are so frequently due to digestive derangements that the condition of these organs should be one of the first things to be looked into.

Examination of the urine should never be omitted in any case of convulsions of doubtful origin, even where no dropsy is present. This, both in infants and older children, is too often overlooked. Asphyxia may be suspected in the case of convulsions occurring in the newly born, late in pneumonia, in some cases of pertussis, in spasmodic or membranous laryngitis, or in laryngismus stridulus. Dentition and worms should be considered among the least probable, never as the most probable, causes of spasmodic. The pulse is weak; it may be slow or rapid; often it is irregular. The forehead is covered with cold perspiration. The face is first pale, then becomes slightly blue, especially about the lips. Unnatural rattling sounds may be produced in the larynx. The bladder and rectum may be evacuated. The convulsive movements consist in an alternation of flexion and extension occurring rhythmically. All varieties of tonic and clonic spasm may be seen, and in all degrees of severity. The contractions of the two sides of the body are usually synchronous. After a variable time, from a few moments to half an hour, the convulsive movements are gradually less frequent, and finally cease altogether, usually leaving the patient in a condition of stupor. They may recur after a short time or there may be but one attack. A period of general relaxation usually follows the convulsive seizures, frequently accompanied by marked evidences of prostration. Transient paralysis, apparently due to exhaustion of the nerve centres, is not an uncommon sequel.

Death may take place from a single attack; this, however, is rare except in very young infants, especially those who are rachitic. There may be no sequel to the convulsions if the cause is a temporary one, or they may produce some serious brain lesion, particularly meningeal hæmorrhage. Death from convulsions is generally due to asphyxia, or to exhaustion from the rapidly recurring attacks. Many cases recover in which the children for several minutes had the appearance of being moribund.

One attack of convulsions is very apt to be followed by others; for the occurrence of the first one usually reveals a peculiar susceptibility of the nervous system, and each succeeding attack comes from a less powerful exciting cause than the previous one. The longer the interval which has passed, the less likely is there to be a repetition, especially if the child has passed its third year. The number of attacks may be very great. In a case under the care of Dr. A. M. Thomas and myself in 1896, an infant during the latter part of its second year had during six months over thirty-five hundred distinct attacks of convulsions. For a considerable period they reached the almost incredible number of eighty a day, and yet the mental condition of the child in the interval was apparently normal.*

Diagnosis.—There can rarely be any difficulty in recognising an attack of convulsions. The difficulty consists in determining with which of the many possible exciting causes we have to do in the case before us. Is it epilepsy? Does it depend upon cerebral disease? Does it mark the onset of some other acute disease? Is it reflex, and if so to what is it due? To answer these questions a careful history must be obtained, and all the circumstances surrounding the patient, the character of the convulsions, and all the other symptoms present must be taken into consideration.

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* The microscopical examination of the brain showed only degenerative changes in the nerve cells of the cortex in the motor area and an increase in the neuroglia. These changes existed over quite an extensive area, and were more marked upon one side.

In infancy, epilepsy is certainly the least probable diagnosis. In older children the most important points indicating that disease are: the presence of some of the stigmata of degeneration, a history of previous attacks, a distinct aura preceding the seizure, or a sudden onset with a cry or fall, biting of the tongue, a tonic spasm preceding the clonic, and, finally, perfect recovery in the course of a few hours after the attack. Convulsions which come on with high fever, even though a patient may have repeated attacks, are seldom epileptic. However, in some cases only prolonged observation can enable one to decide positively whether or not epilepsy is present.

Convulsions occurring in brain disease, except acute meningitis, are not as a rule accompanied by any marked rise in temperature. Focal symptoms are often present, such as localized paralysis or rigidity, changes in the pupils, and strabismus. The convulsive movements are frequently limited to one side of the body. It should, however, be borne in mind that unilateral convulsions, even when repeated, do not always mean a local lesion, as I have seen proved by autopsy more than once. In hæmorrhage or meningitis, convulsions are likely soon to recur. In tumour they may recur after a longer interval.

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Prognosis.—This depends upon the age of the patient and the cause of the convulsions. Idiopathic or reflex convulsions are rarely dangerous to life except in very young or in rachitic infants. Convulsions associated with enlarged thymus are often fatal. Convulsions occurring at the onset of acute febrile diseases are seldom fatal, and not often serious; they may not even indicate an unusually severe type of the disease. Especially fatal are the convulsions of pertussis and of asphyxia when they occur late in any form of laryngeal or pulmonary disease. In nephritis, while always serious, convulsions are by no means invariably fatal. The conditions during an attack which should lead one to make a bad prognosis are when the convulsions are prolonged or recur frequently; also the presence of very great prostration, a feeble pulse with cyanosis, or deep stupor.

In the prognosis one must take into account not only the immediate result of the attack, but its possible outcome. Except where convulsions mark the beginning of epilepsy, they are much less serious than they are generally supposed to be by the laity. In a highly nervous or susceptible child a convulsion may often mean no more than does an attack of severe migraine in an older person. Such are undoubtedly most of the attacks seen in practice. Permanent injury to the brain, simply as a result of an attack, although possible, is still rare. But when convulsions are repeated the development of epilepsy is to be feared. There is little doubt that some cases of epilepsy have their origin in attacks of convulsions, which in the beginning were the result simply of digestive derangements; by a constant repetition of the exciting cause the convulsive habit finally becomes established. This possibility is therefore to be borne in mind in all cases where children have had several convulsions, although it is unusual that this result is seen. The farther apart the attacks are and the more definite the exciting cause, the less likely is this to be the case.

Treatment.—Summoned to a child in convulsions, a physician should go at once and remain until the attack has subsided. He should take with him chloroform, a hypodermic syringe with morphine, a soft catheter or rectal tube, and a solution of chloral. In order to treat convulsions intelligently one must have in mind the prominent pathological conditions. These are acute cerebral hyperæmia, a more or less severe asphyxia with pulmonary congestion, an overtaxed right heart, and in fact a tendency to congestion of all the internal organs. The nervous centres are in a condition of such unnatural excitability that the slightest irritation may bring on convulsive movements when they have temporarily subsided. The patient should therefore be kept perfectly quiet, and every unnecessary disturbance avoided. Cold should be applied to the head—best by means of an ice cap or cold cloths—and dry heat and counter-irritation to the surface of the body and extremities. The time-honoured mustard bath causes so much disturbance of the patient that it can usually be dispensed with and the mustard pack (page 54) substituted. The feet may be placed in mustard water while the child lies in its crib. The mustard pack and footbath should be continued until the skin is well reddened. The degree to which counter-irritation of the skin should be carried will depend upon the condition of the pulse and the cyanosis.

In controlling convulsions the three remedies which may be depended upon are the inhalation of chloroform, morphine hypodermically, and chloral. Chloroform is undoubtedly the most reliable remedy for an immediate effect, and should be used even in the youngest infant. At the same time that it is being administered, chloral should be given *per rectum*. The initial dose should be, at six months, four grains; at one year, six grains; at two years, eight grains, dissolved in one ounce of warm milk. It should be injected high into the bowel through a catheter, and prevented from escaping by pressing the buttocks together. It may be repeated in an hour if necessary. The effect of the drug is generally obtained in twenty minutes. If, in spite of the chloral, the convulsions show a marked tendency to continue as soon as the chloroform is withdrawn, or if the enema of chloral has been expelled, morphine should be given hypodermically. Where the heart's action is weak, this is probably the best of all remedies. Objections are urged against it only by those who have had no experience with its use. To a well-grown child two years old, $\frac{1}{16}$ of a grain may be given; one year old, $\frac{1}{24}$ of a grain; six months old, $\frac{1}{48}$ of a grain. This dose may be repeated in half an hour if no effect is seen. The tolerance of opium in cases of convulsions is very marked, and sometimes double the doses mentioned may be required. The only other agent of much value is oxygen. I have seen convulsions which continued in spite of all other means, yield immediately to oxygen. This is most likely to be valuable in cases of convulsions due to asphyxia.

When once under control, the recurrence of the convulsions may be prevented by keeping the patient for two or three days under the influence of chloral with bromide of sodium, the amount of chloral being gradually reduced. If it is badly borne by the stomach and not easily retained by the rectum, either antipyrine or phenacetine may be used with the bromide. Where there is a strong tendency to recurrence of the convulsions, urethan is sometimes even more efficient than chloral. It may be given in the same or in slightly larger doses.

As soon as the convulsions have ceased, the cause should be sought and treated. In infancy it is wise in every case to irrigate the colon thoroughly with warm water, to remove any possible source of irritation. If there is reason to suspect the presence of undigested food in the stomach, this may be washed out. Much more frequently it is in the intestines, and free purgation by calomel is advisable. If there is high temperature, this should be reduced by the cold bath or pack. Secondary attacks are to be prevented by careful feeding, by improving the general nutrition by means of fresh air, iron, cod-liver oil, and phosphorus. The last two are especially valuable in cases due to rickets.

EPILEPSY.

Epilepsy may be defined as a disease in which there is an established disposition to convulsions of a certain type, with loss of consciousness, which have recurred until a habit of convulsions has become fixed.

A distinction must be made between cases of so-called "idiopathic" epilepsy and those which are secondary to a definite lesion of the brain, such as tumour, sclerosis, or abscess. Convulsions of the latter character are designated as "symptomatic" epilepsy, and are discussed in connection with the various diseases in which they occur. The nature of the attack may, however, be identical in both varieties, and may not differ from an ordinary attack of convulsions or eclampsia.

The proportion of idiopathic cases in children is not so large as was formerly supposed; many of these have been shown to depend upon lesions once overlooked, particularly mild infantile cerebral paralyses.

Etiology.—From a consideration of 1,450 cases of epilepsy, Gowers states that 12 per cent begin in the first three years of life, and 46 per cent between ten and twenty years. The greatest tendency to the development of the disease is shown about the time of puberty. Females are rather more liable to be affected than males, although the difference in sex is slight. Heredity plays an important rôle in the production of the disease. In one-third of the cases, according to Gowers, there is a family history either of epilepsy or insanity. All hereditary nervous diseases predispose to epilepsy, but it is a question whether other hereditary diseases have any special influence.

Not very infrequently epilepsy may be traced to convulsions occurring during infancy. In what proportion of the cases this is true it is impossible to state with accuracy. Infantile convulsions are very common, and usually the cause which produces them is a transient one. The proportion of such cases which develop epilepsy later in life is certainly small. One frequently meets with children from two to five years old who have occasional attacks of convulsions, often from apparently trivial causes. In my experience, the great majority of these also recover completely with proper treatment; a very few become epileptic. Given a strong predisposition to epilepsy, it is easy to see how convulsions early in life so often associated with rickets may have been the first of the epileptic series. The first seizure is sometimes traceable to fright, great excitement, heat-stroke, or blows or falls upon the head even without any gross lesion. It may follow any of the acute diseases of childhood, particularly scarlet fever, rarely measles or typhoid. In none of these, however, is it often seen. As reflex causes may be mentioned intestinal worms, phimosis, adenoid vegetations of the pharynx, delayed or difficult menstruation, and masturbation. Most of these are rare causes, but they may be sufficient to produce the disease where a strong predisposition exists. Syphilis may be the cause of epilepsy even when there is no local disease of the brain.

Among the most important factors in producing a paroxysm, is intestinal putrefaction associated with chronic constipation and chronic intestinal indigestion. This subject has been investigated with great care by Herter and Smith,* who studied 238 specimens of urine from 31 epileptics. In 72 per cent of their observations there was unmistakable evidence of excessive intestinal putrefaction, as shown by the presence of ethereal sulphates in the urine in large amount, just before the occurrence of the paroxysm. The inference seems warranted that this intestinal condition was closely connected with the epileptic seizures. The statement of Haig, that there is an excessive elimination of uric acid preceding the paroxysm, was not borne out by the observations of Herter and Smith. The association of intestinal putrefaction with seizures of epilepsy is very important as furnishing a clew to the management of many of these cases. I believe it to be one of the most important etiological factors in cases occurring in children, particularly as an exciting cause of the first attacks.

Pathology.—It is not within the scope of this work to discuss the various theories which have been advanced. The following are the conclusions reached by Gowers: †

"The muscular spasm is to be regarded as the result of the sudden overaction (discharge) of nerve cells, the violent liberation of nerve force, and the sensations which the patient experiences before losing consciousness must be due directly or indirectly to the same cause. The disease which excites convulsions is most frequently at the cortex, and when organic disease causes convulsions that begin locally, the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this way, and this suggests very strongly that in such cases the change occurs in the cortex. Epilepsy must then be regarded as a disease of the gray matter, most frequently of the gray matter of the cortex."

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* New York Medical Journal, August and September, 1892.

† Diseases of the Nervous System, American ed. 1888, p. 1098.

While there is pretty general agreement that the seat of the morbid changes in true epilepsy are in the cortex, but little is yet definitely known as to the nature of these changes. Van Gieson has published * some very careful observations made upon portions of the cortex removed at surgical operations from two epileptic patients. In one of these the disease was primarily due to a foreign body; in the other, to an old cicatrix. The conditions found represent the earlier changes of the disease, and were essentially the same in both cases. There were degenerative changes in certain of the ganglion cells, which in places had resulted in almost complete dissolution of these cells. In addition there was a distinct hyperplasia of the neuroglia tissue. Diffuse neuroglia sclerosis starting from the focus of disease has been reported by certain French writers—Marie, Féré, and Chaslin.

Symptoms.—Two distinct types of epileptic seizures are met with: the major attacks, or *grand mal*, in which there are severe convulsions lasting from two to ten minutes, with loss of consciousness, etc.; and minor attacks, or *petit mal*, in which the convulsive movements are slight and may be absent, and in which the loss of consciousness is often but momentary. Between these two extremes all gradations are seen.

Grand mal.—The onset may be sudden, without premonition, or it may be preceded by certain prodromal symptoms known as the aura. The aura may be motor, such as a local spasm of the hand, face, or leg; or sensory, such as numbness and tingling in any part of the body, or some abnormal sensation rising gradually to the head, at which time loss of consciousness occurs. The variety of sensations described by patients as indicating an attack is endless. There may be a sensation in one finger, in the face, tongue, eye, or in any part of the body; or the warning may be of a general character, like a tremor or a shivering sensation, or a feeling of faintness. There has also been described a visceral or pneumogastric aura, in which there is epigastric pain, sometimes nausea, and a sensation of a ball in the throat; or there may be palpitation, or cardiac distress. There may be general giddiness or vertigo, or a sensation of fulness in the head; or feelings of strangeness, or a dreamy, dazed condition; and, finally, the aura may have reference to any of the special senses, most frequently to sight. Sparks may appear before the eyes, or flashes of light or colour, or strange objects may be seen; or there may be a momentary loss of hearing; or strange sounds may be heard. In most cases the aura is peculiar to the individual, whose attacks are likely to be preceded by the same symptoms.

At the beginning of the seizure the face becomes pale, the pupils widely dilated, the eyes rolled up in their orbits and fixed. Speedily there is loss of consciousness. Simultaneously with these symptoms, or immediately following them, there occurs a violent tonic muscular spasm to which are due the characteristic symptoms of the early part of the seizure—viz., the fall, cry, biting of the tongue, cyanosis, and evacuation of the bladder or rectum. The fall is forcible, violent; in fact, the patient is precipitated usually forward, and frequently suffers injury, never sinking down as in a faint. The head is often strongly rotated to one side. The position of the hands is often that assumed in tetany. The cry is a hoarse, inarticulate sound, not very loud, and is due to forcible expiration, owing to spasm of the muscles of respiration with the glottis partially closed. The cyanosis is the result of tonic spasm of the muscles of respiration; it may be quite intense, so that the face is livid, bloated, and the features distorted. The spasm of the muscles of mastication causes the biting of the tongue. Evacuation of the bladder and rectum may result from contraction of their walls, or from spasm of the abdominal muscles. The violence of the muscular spasm in this stage may be very great; it has caused fracture of bones, rupture of muscles, and even dislocation of joints.

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* New York Medical Record, April 24, 1893.

The stage of tonic spasm may be only momentary, the patient passing almost at once into the stage of clonic convulsions. The usual duration is from ten seconds to half a minute. In the stage of clonic spasm which follows, the symptoms are those of an ordinary attack of convulsions. The muscular contractions are violent, and there is often frothing at the mouth. Gradually the muscles of respiration relax, air enters the lungs, and the cyanosis passes off. After the clonic spasm has continued for a variable time—from two or three minutes to half an hour—the muscular contractions become less and less frequent, and finally cease altogether. In a few minutes the patient may regain consciousness, look vacantly around, and in a dazed way perhaps ask what has happened, he being completely oblivious to all that has occurred. More frequently, however, he passes at once into a deep sleep, which continues for an hour or more, but from which he can be aroused. From this he usually wakens with a severe headache, which may continue for several hours. After this he often feels better than for several days preceding the attack. During the seizure the temperature may be elevated one or two degrees, but rarely more. The attack may be followed by a slight temporary paresis, or aphasia, hysterical phenomena, vomiting, and intense hunger. In very rare cases the urine may contain a trace of sugar.

Petit mal.—The minor attacks of epilepsy may present a very great variety of symptoms, and at times it is almost impossible to decide that these are epileptic, except from their periodical occurrence. They pass under the names of "spells," "attacks of dizziness," "fainting turns," etc. The most striking thing which stamps them as epileptic is the loss of consciousness, and this may be of short duration, sometimes only momentary, and so pass unnoticed. In some cases it is absent altogether. There is no fall, but there may be a slight dropping of the head, a fixed stare for a moment or two, and that is all. This may or may not be preceded by an aura. After such a mild attack the patient's mind may be somewhat confused, and he may do or say strange things. All sorts of curious acts have been performed in an automatic way by patients in the condition which follows an attack of epilepsy, which may perhaps be regarded as part of the attack. In rare instances even acts of violence may be done.

The mental condition of epileptics.—In this connection a careful distinction must be made between cases in which epilepsy is secondary to some organic brain disease, such as infantile cerebral palsy, which may itself be a cause of mental impairment, and the mental disturbances seen in cases of idiopathic epilepsy. The children who are the subjects of the latter disease, and who are perfectly normal mentally, are certainly few. All degrees of disturbance may be seen, from those who are simply dull, apathetic, backward in development, and uncontrollable in temper, to those who are melancholic, idiotic, and even maniacal. The earlier in childhood epilepsy develops, the greater is usually the mental disturbance seen, because of the effect of the seizures upon the brain during its period of active growth. Speech and all mental development may be greatly retarded. The more frequent and more severe are the attacks, the more marked are the mental symptoms present.

Symptomatic epilepsy.—This occurs most frequently in children as a sequel of cerebral palsy, usually with hemiplegia, and it may follow either the congenital or acquired form. Epilepsy may come on at any time after the onset of the paralysis—from a few months to five or six years. At first the attacks may be separated by long intervals, but they gradually become more frequent as time passes. The convulsions in post-hemiplegic epilepsy begin, as a rule, on the paralyzed side, and for a long time they may be confined to that side; but later they may become general, in which cases they are indistinguishable from attacks of idiopathic epilepsy. Severe seizures are more likely to be seen than are the mild ones.

Course of the disease.—This is extremely irregular. In most cases seizures at first occur at long intervals, of perhaps a year, but later they become more and more frequent. Either the mild or the severe attacks may be first seen, and may remain throughout as the only type present, or they may be associated in the same case. There are most frequently seen, occasional major attacks with a large number of minor ones. The interval between the epileptic seizures in most cases is from two to four weeks, although they may be of daily occurrence. Sometimes three or four seizures will follow one another closely, and then there will occur a long interval of immunity. The seizures may come on either during sleep or in the waking hours, and in some cases for a long time they may occur only in sleep. Such cases present peculiar difficulties in diagnosis, and are often long unrecognised as epileptic. The general health of patients may be quite normal.

Death rarely, if ever, results from epilepsy, except from some accident at the time of the seizures, or from the condition known as the *status epilepticus*; in this the attacks come on with great frequency and severity, the patient at times passing rapidly from one convulsion into another, the temperature rising to 105° or 106° F., and death occurring either from exhaustion, owing to the severity of the convulsions, or from coma.

Diagnosis.—In most cases there is little difficulty in recognising the major attacks when they occur by day. Nocturnal attacks may be diagnosticated by the cry, the biting of the tongue, blood upon the pillow, sub-conjunctival extravasation, evacuation of the bladder or rectum, and the severe headache. Minor attacks present the greatest difficulties, and a positive diagnosis is often impossible until the patient has been watched for a long time. The most important points to be noted are sudden pallor, dilatation of the pupils, temporary loss of consciousness, or simply mental confusion, and sometimes the evacuation of the bladder. The duration of the attack is shorter than is usual in an ordinary faint. The difficulty of distinguishing epilepsy from hysteria rarely occurs in childhood.

It is not always possible to distinguish between secondary or symptomatic epilepsy and the idiopathic or hereditary form, particularly if the case comes under observation late in the course of the disease. The points which go to establish the first form are: that the convulsive movements are partial, or limited to one side; that when they are general, they always begin in the same part of the body; or that there is a history of partial or unilateral attacks for some time before the occurrence of any general convulsions. It is important in all cases to examine the patient carefully for signs of an old hemiplegia, the symptoms of which may be so slight as to be readily overlooked. A marked increase in the reflexes of one side is, according to Sachs, quite as conclusive evidence as a distinct weakness of the arm or leg. In idiopathic epilepsy some of the stigmata of degeneration are usually present. The sudden development of epileptic seizures in a child previously healthy, and in whom there is no hereditary history of the disease, should always arouse the suspicion of organic brain disease, especially tumour; and if there are besides, severe headache, vomiting, and optic neuritis, the existence of tumour is reasonably certain.

Prognosis.—The danger to life in epilepsy is very slight. Death is generally due to some accident, particularly drowning, at the time of a seizure. The tendency to spontaneous cessation of the attacks is small, while the tendency to recurrence is very great.

The prognosis in any given case depends upon the cause of the disease and the duration of the symptoms. Where the cause can be removed, and where the symptoms have lasted less than a year, the prospects of permanent cure are fairly good. This is particularly true of cases in which the epilepsy clearly depends upon gross errors in diet, with chronic intestinal indigestion. In such cases, if the patient can be placed under proper control and dietetic measures well carried out, the development of chronic epilepsy can be arrested in a considerable number of cases. If, on the contrary, the hereditary tendency to the disease is marked, if the epileptic seizures have developed apart from any adequate exciting cause, and if they have continued untreated or in spite of treatment for two or three years, the symptoms may perhaps be relieved, but there is no prospect whatever of permanent cure. In the cases also which are due to local irritation, like that resulting from an old meningeal hæmorrhage, the prognosis is invariably bad, and only temporary relief is to be expected. A few cases of traumatic epilepsy have been cured and many have been greatly improved by a surgical operation.

Treatment.—The first indication is to remove the cause where one can be found. If in the male phimosis exists, or other evidence of genital irritation, circumcision should be done, or the prepuce retracted and adhesions broken up. Adenoid growths of the pharynx should be removed, and likewise every other cause of reflex irritation. Particular attention should be given to the digestive organs. The most hopeful cases are those associated with acute and chronic disturbances of digestion, especially chronic intestinal indigestion with constipation. These cases are to be managed like others of the same sort in which epileptic attacks are not present (page 418). Meat should be allowed once a day and in moderate quantity. Milk should be given, diluted if necessary, also kumyss and matzoon. Green vegetables, except peas and beans, may be given freely; also all fresh fruits. Tea, coffee, and alcohol in every form must be absolutely prohibited; also potatoes and oatmeal. The most careful attention should be given to the bowels. Under no circumstances should a condition of chronic constipation be neglected. A dose of calomel once a week and intestinal irrigation two or three times a week are of great value in many cases. Where the symptoms of intestinal putrefaction are marked, borax is at times of decided value—two grains three times a day to a child of five years—or salicylate of sodium, salol, or the benzoate of sodium may be given; the dose of each being from two to ten grains, according to the age of the child, after each meal. The general hygiene of the patient must receive careful attention. He should lead a simple, regular life, as much as possible out of doors, away from the excitements of a large city, or from association with many children, and in short the nervous system should be kept as quiet as possible.

All the foregoing means of treatment are of equal importance with the use of special drugs. The most common mistake is to rely only upon drugs, ignoring the other measures mentioned. It not infrequently happens that drugs are without any effect when they are the only means of treatment employed, whereas in conjunction with other measures marked improvement is seen.

The bromides are unquestionably the best means of combating the epileptic habit. Either the sodium salt alone or a combination of the sodium and ammonium bromides is to be preferred. The purpose should be to give the smallest doses which will control the seizures. Children require proportionately larger doses than adults, and in most cases a child of five years will need from twenty-five to fifty grains a day. Seguin's* method of administering the bromides is largely followed in New York, and is of great value. It is to give the larger part of the quantity for twenty-four hours, shortly before the time when the seizures have usually occurred; in the interval to give much smaller doses, and in all cases to give the dose largely diluted,—in from six to eight ounces of water. He gives a full dose early in the morning, and, where the seizures are apt to come at night, one at bedtime.

Cases of *petit mal* are especially difficult to control. For such there is often an advantage in combining belladonna with the bromides. In all cases the treatment must be continued for a long time if anything is accomplished. The bromide should be gradually reduced after the attacks are controlled, but must be given in moderately large doses for at least two years after the seizures have ceased. The addition of borax seems occasionally better than the bromides alone in cases where there is excessive intestinal putrefaction. Sometimes the combination of chloral or antipyrine with bromides is advantageous, particularly if the latter are badly borne or cause an annoying amount of acne. Seguin states that he has been able to control the acne in many cases by giving at the same time moderate doses of arsenic. Other drugs occasionally useful as adjuvants to the bromides are strychnine and digitalis.

The surgical treatment of epilepsy has of late attracted much attention. An operation is to be considered in cases in which the paroxysms are very frequent and severe, and when there is present a definite local cause, such as an old fracture of the skull, or where epilepsy has followed an injury to the head even without fracture. Sachs sums up the present status of this question as follows: "In a case due to a traumatic or organic lesion an early operation may prevent the development of cerebral sclerosis. If early operation is not done, the occurrence of epilepsy is a warning that secondary sclerosis has been established and an operation may prevent it from increasing. Operation must include the removal of the diseased area; here, if all other parts are normal, a cure may result. Under favourable conditions a few cases of epilepsy may be cured by surgery and many more improved."

The education of epileptic children is a subject of great difficulty and is often neglected. There are many reasons why it is impracticable to send them to ordinary schools, and it is very desirable that special schools and colonies for them should be established.

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* New York Medical Journal, March 29, 1890.

The management of the attack.—Abortive measures are sometimes successful in cases with a distinct aura, the most reliable being the inhalation of nitrite of amyl. While the seizure lasts, the patient should be prevented from injuring himself. The clothing should be loosened, a spool or cork should be placed between his teeth to protect the tongue, but no effort made to restrain his movements unless he is liable to do violence to himself. An epileptic child should never be without some companion.

TETANY.

Tetany is a condition characterized by tonic muscular spasm, which may be intermittent or continuous. It usually affects the muscles of the extremities, especially the hands and feet, more rarely the neck, face, and trunk. When limited to the hands and feet it is known as carpo-pedal spasm or arthrogryposis; and although sometimes classed separately, this seems to be really only one manifestation of the same general condition. In infants, tetany is very frequently associated with laryngismus stridulus, this being present in fully two thirds of the cases; but in older children this association is quite rare. General convulsions occur in from twenty to thirty per cent of the cases. Tetany is not a frequent disease in America. In a pretty large hospital service I seldom see more than four or five cases a year, while in some European cities tetany is reported to be very common and at times to occur epidemically. It is probable that more than one pathological condition has been included under this term.

Etiology.—While tetany may occur at any age, it is most frequent in infancy. Of eighty-seven cases reported by Barthez and Sanné, fifty per cent were observed in the first two years, twenty per cent from three to six years, and twenty-five per cent from twelve to fifteen years. Of thirty-eight cases in children collected by Griffith, sixty-six per cent were under two years of age. In infancy males are much more frequently affected; but when the disease occurs in older children, females appear more liable to it. Tetany rarely occurs as a primary disease. It is most frequently associated with rickets; in fact, rickets is almost invariably found in the infantile cases. It sometimes occurs with chronic diarrhœa and with marasmus. It has been known to follow broncho-pneumonia, pertussis, typhoid fever, rheumatism, and measles. Of the exciting causes, the most frequent one is some irritation in the gastro-enteric tract. This may be the products of chronic indigestion, or acute intoxication, worms, and sometimes even intussusception. Attacks in older children are frequently ascribed to cold. In girls, tetany may occur at the time of puberty, especially where menstruation is delayed; it has followed removal of the thyroid gland.

Pathology.—Up to the present time no constant anatomical lesions have been demonstrated in tetany. The circumstances in which it occurs, its symptoms and course, all indicate that it is a neurosis probably depending upon disturbances of nutrition in the nerve cells of the spinal cord and medulla.

Symptoms.—The spasm may develop abruptly, or it may be preceded by sensory disturbances, such as pain, numbness, or tingling. The upper extremities are usually first affected, the spasm gradually becoming more severe and finally involving the lower extremities. Both sides of the body are equally affected. The position assumed by the hands is very characteristic: The fingers are flexed at the metacarpo-phalangeal joint and the phalanges extended; the thumbs are adducted almost to the little finger; the wrist is flexed at an acute angle, and the whole hand drawn somewhat to the ulnar side (Fig. 121). No motion is allowed at the wrist, but movements at the elbow and shoulder are usually normal. The feet are strongly extended, sometimes in the position of typical equino-varus. The first phalanges of the toes are flexed, and the second and third rows extended; the plantar surface is strongly arched, and the dorsum of the foot is very prominent, standing out like a cushion. The typical position of the feet is well shown in Fig. 121. The tendo-Achillis stands out prominently. Motion at the hip and knee is generally free. The spasm in many cases is limited to the hands and feet; more rarely the muscles of the thigh, usually the adductors, may be involved. In very rare cases the muscles of the trunk, the face, or the eye may be involved.

The knee-jerk and the cutaneous reflexes are exaggerated, and there is abnormal excitability both to the galvanic and faradic currents and to mechanical irritation. Light percussion upon the nerve trunk often induces marked contraction of the muscles supplied by the nerve. This is particularly striking in the face. The contraction of the facial muscles following such irritation is known as " Chvostek's symptom " or the facial phenomenon. Spasm may also be excited by pressure upon the large nerve trunks and arteries of the parts affected. This is known as " Trousseau's symptom."

Pain owing to the spasm is frequently present. It is usually sharp and lancinating, and may be so severe as to cause children to cry out. Pain is induced by any attempt to overcome the spasm, and sometimes it is constant. Other disturbances of sensibility are even more common than pain. There is no loss of consciousness and no fever. The spasm is generally continuous, although there may be periods of remission or even of intermission. When associated with laryngismus stridulus, the spasm is much increased during these attacks.

The duration of the disease is from a few days to several weeks. The mild form, which is usually seen in infants, in most cases passes away spontaneously in one or two weeks, although there may be relapses and second attacks at variable intervals. The most important complication is general convulsions. These may come on at any time in the course of the disease. Spasm of the glottis may either precede or follow tetany. When associated they generally cease at the same time. Slight paralysis may follow or alternate with the spasm.

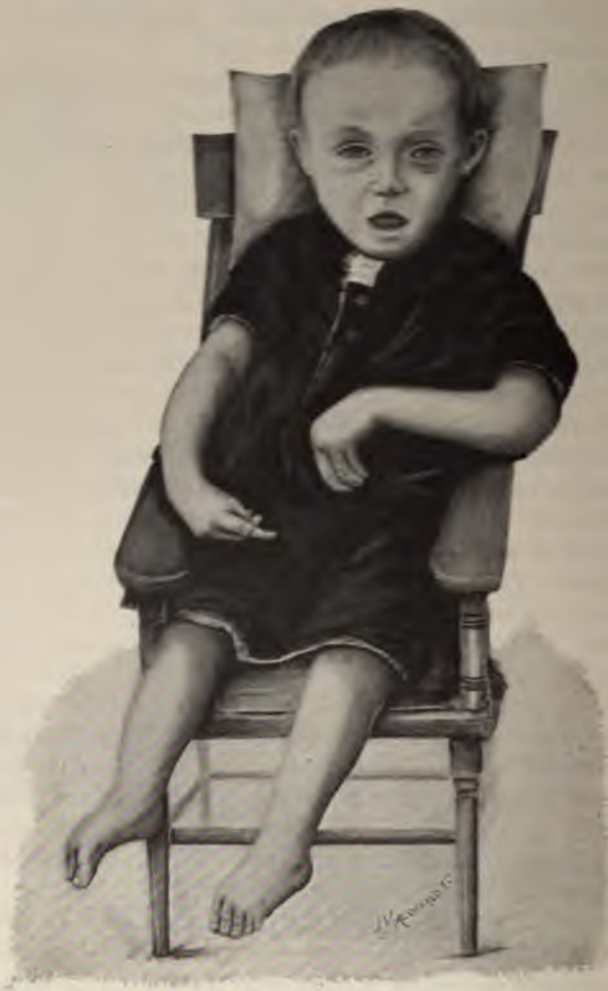

Fig. 121.—Tetany, showing the characteristic position of the hands and feet, in a child two years old.

Diagnosis.—The diagnostic features of the disease are bilateral spasm—in infants usually limited to the hands and feet—without loss of consciousness, the spasm being increased or excited by pressure upon the nerves, exaggerated reflexes, and the presence of some previous disease, especially rickets or some disorder of the intestines. The severe form may be mistaken for tetanus; but this is very rare except in the newly born; and trismus is the rule, and generally it is the first symptom. Trismus is extremely rare in tetany. From meningitis, tetany is distinguished by the absence of cerebral symptoms; from cerebral tumour, by the bilateral character of the spasm, the absence of headache and focal brain symptoms; from hæmorrhage, by the absence of cerebral symptoms; from malarial spasm, by the fact that it is constant, not intermittent.

Prognosis.—Tetany *per se* is not fatal, but death may result from the development of general convulsions or from the original disease which tetany complicates. Recovery is usually perfect, although Gowers states that in rare cases it is followed by muscular atrophy.

Treatment.—The first indication is to remove the cause, and this in most cases is found in the digestive tract. If rickets is present it should receive the usual treatment, both dietetic and medicinal. If worms are suspected a vermifuge should be given. For the relief of the spasm, the hot bath is a most valuable remedy; friction may also be employed. Drugs which have the power of allaying spasm should be given,—chloral, bromides, and antipyrine. In the event of failure by these methods galvanism may be tried. After the attack the child's general nutrition should receive careful attention, to prevent relapses.

LARYNGISMUS STRIDULUS—SPASM OF THE GLOTTIS.

Idiopathic spasm of the glottis, or laryngismus stridulus, is a rather rare disease, and belongs especially to infancy. It is a pure neurosis, not often seen except in children who are rachitic. It is frequently associated with carpo-pedal spasm and with general convulsions. The disease is not to be confounded with ordinary spasmodic croup or catarrhal spasm of the larynx, which is of very frequent occurrence.

Spasm of the larynx may be seen in several conditions quite different from laryngismus stridulus. It forms one of the essential features of pertussis. It occurs both in infants and in older children from pressure upon, or irritation of, the pneumogastric or recurrent laryngeal nerve by a tumour in the mediastinum,—usually a tuberculous lymph node, or retro-œsophageal abscess. Reflex spasm of the larynx is also associated with enlarged tonsils, adenoid growths of the pharynx, and elongated uvula. There is a form of reflex spasm which occurs in the newly-born accompanied by crowing inspiration; this is not frequent, and is rarely serious.

Idiopathic spasm of the larynx is quite different from any of these conditions. It is peculiar to infancy, the great proportion of cases occurring between the sixth and eighteenth months. Males appear to be more susceptible than females. The constitutional condition with which it is usually associated is rickets. In a large number of cases, but not in all, there is cranio-tabes. Many writers believe that laryngismus is invariably of rachitic origin. Of fifty cases observed by Gee, there were found in all but two unmistakable evidences of rickets. The disease occurs in delicate infants who have been closely confined in warm rooms, and it is probably on this account that it is more often seen in the winter and spring than at other seasons. The exciting causes of this spasm may be a breath of cold air, or any form of nervous excitement, such as passion, fright, or crying.

Pathology.—There are no anatomical changes in this disease. It is a pure neurosis, and it is generally believed to be of central origin, depending essentially upon imperfect nutrition of the motor centres of the spinal cord and medulla.

Symptoms.—The disease is often unnoticed by the parents until the attacks have become quite frequent, the first ones being mild, and the later ones more and more severe. Occasionally the very first paroxysms may be severe. The attack comes on suddenly. The child throws back his head, the face becomes pale, then livid, and for the time there is complete arrest of respiration. This continues for a few moments, during which the cyanosis deepens, and the child seems in great distress, making violent efforts to breathe. If the paroxysm is a severe one, the asphyxia may be so great as to lead to loss of consciousness, and it may even be fatal, or the attack may terminate in general convulsions. In milder attacks, after fifteen or twenty seconds the muscular spasm relaxes, the glottis opens, and a long, deep inspiration occurs, with the production of a crowing sound. The so-called "holding-breath spells" and the "crowing attacks" of infants are usually of this nature. Such forms of spasm are often brought on by passion or any excitement, and may occur from two or three to twenty times a day. Between them the condition of the child may be normal, or carpo-pedal spasm may be present. It is important to note that in this disease there is not a stridor due to narrowing of the glottis, as in ordinary croup, but a condition of apnœa from its complete closure. Not all the paroxysms in the same case are equally severe. A child may have in the course of a day a great many mild attacks, but only a few severe ones. General convulsions are seen in over one third of the cases, and carpo-pedal spasm or tetany complicates a still larger proportion. If tetany is present in the interval, it is always increased during the attacks.

The duration of the disease varies from a few days to several weeks, or even months. In cases which terminate in recovery there is a gradual diminution in the frequency and severity of the paroxysms, until they finally cease altogether.

Prognosis.—This is good, except when there are general convulsions. The cases in which fatal asphyxia occurs are very rare. Usually with proper treatment marked improvement begins in the course of a few days.

Diagnosis.—This is to be made from catarrhal spasm of the larynx. The differential points have been mentioned under the latter disease.

Owing to the occurrence of the paroxysms and the crowing sounds, the disease may be mistaken for whooping-cough, and in fact this diagnosis is not infrequently made by parents. A careful examination of the patient during the attacks, the absence of cough, and the frequent association of tetany, are sufficient to differentiate this from pertussis.

Treatment.—During the attack the object is to break the spasm. In mild cases this may be done by sprinkling water in the face. In severe cases inhalations of chloroform may be required, and even intubation. Between the attacks the patient should be given either bromide and chloral, or antipyrine. Sodium bromide, gr. v, and chloral, gr. ij, may be given every three or four hours to a child a year old until the frequency and severity of the attacks are controlled; afterward three times a day. My own experience with antipyrine in this disease leads me to the belief that it is more effective than bromide and chloral. When the symptoms are severe, two grains of antipyrine may be given every four hours to a child a year old, the dose being gradually diminished as the symptoms improve.

The general treatment of the child is quite as important as drugs directed toward relieving the spasm. Cold sponging should be used in every case unless it occasions so much fright as to increase the number of paroxysms. Careful attention should be given to the diet. Children should be kept in the open air as much as possible. Cod-liver oil is needed in most cases, and rachitic cases are sometimes much benefited by phosphorus. Any source of local irritation, such as enlarged tonsils, elongated uvula, or adenoid growths, should be removed; for, if not the actual cause of the attack, they may be the means of aggravating the symptoms. In all cases the treatment should be continued for several weeks after the paroxysms have subsided.

CHOREA—SAINT VITUS'S DANCE.

Chorea is a functional nervous disease characterized by aimless, irregular movements of any or all the voluntary muscles. Choreic movements are of a somewhat spasmodic character, often accompanied by an apparent or real loss of power in the groups of muscles affected, and by a mental condition of extreme irritability.

Etiology.—Chorea is most frequently seen between the ages of seven and fourteen years. Of 146 cases, 6 were under five years, 72 between five and nine years, and 68 between ten and fourteen years. The youngest case of which I have record was that of a child four years old. It is extremely rare before the third year, although it may occur even in infancy, and in a few recorded cases it was undoubtedly congenital. My own observations coincide with those of nearly all writers, that the disease is more than twice as frequent in females as in males. While chorea may be seen at all seasons, it is much more frequent in the spring months. Of 717 attacks studied by Lewis (Philadelphia), the largest number began in March, and the next largest number in May; in my own cases May stood first.

The relation of chorea to rheumatism is of much importance, and has during late years attracted a great deal of attention. Thus far the investigations of different writers have given results which are somewhat contradictory. Some have found evidences of rheumatism in but a small proportion of the cases—in not more than 5 or 10 per cent—while the statistics of others have placed the percentage of rheumatism as high as 50 or even 60 per cent. It is rather striking that the statistics of neurologists, almost without exception, have given a very much smaller percentage of rheumatism in choreic cases than those taken from children's clinics and hospitals. The question hinges largely upon what is to be admitted as evidence of rheumatism in a child; if cases of acute articular inflammation only, then the number will be very small; if subacute cases with joint swellings are included, the proportion will be considerably larger; while if we admit cases of acute endocarditis without articular symptoms, and those of articular pains and joint stiffness but without swelling, the proportion will be very much increased. My own belief is that there is a very close connection between chorea and the rheumatic diathesis as manifested by all the symptoms above noted, and accompanied by a family history of rheumatism. On careful scrutiny, the number of cases of chorea in which unmistakable evidence of this diathesis is found, is very large, including in my own observations over one half the cases. There seems, then, to be a large group of cases which may be classed distinctly as rheumatic chorea. There are, however, many others in which no such element can be found.

My former associate, Dr. F. M. Crandall, has analyzed 146 cases of chorea treated by us at the New York Polyclinic and elsewhere, with the following results: Of 111 cases in which the question of rheumatism was investigated there was a definite history of it in 63. In 41, rheumatism occurred before the chorea; in 13, the first evidence of rheumatism was coincident with the chorea; and in 9 it first occurred subsequently to the chorea, usually within three months. In about one third of the cases, attacks or rheumatism occurred during or subsequent to the chorea as well as before it. It may then be stated that previous rheumatism was evident in 37 per cent, concurrent rheumatism in 24 per cent, and subsequent rheumatism in 15 per cent of the cases. Excluding cases mentioned twice, and also all those in which there was a history only of "growing pains," there was evidence of articular rheumatism in 56.7 per cent of the cases. Many of these patients have now been under observation for several years, and it has been interesting to see, as time has passed, how the evidences of the rheumatic diathesis have multiplied the longer the cases have been followed.

In the above statistics only articular symptoms have been accepted as evidence of rheumatism. If the cases of endocarditis without articular symptoms were included, as I think they might fairly be, it would raise the proportion of rheumatic cases still higher. The great proportion of cardiac murmurs persisting after chorea, if not all of them, should, I believe, be classed as rheumatic, even if no articular symptoms have been present.

Overpressure in school is often an important factor in the production of chorea, as has been shown by Sturges (London). Anæmia, if not an essential factor, is certainly a very important one, and the great proportion of cases present very distinct evidences of it. Chorea may develop as a sequel of any of the infectious diseases, more particularly scarlet and typhoid fevers. It is seen quite often in cases of chronic malarial poisoning. Among the reflex causes may be mentioned phimosis, either lumbricoids or pinworms, delayed menstruation, and ocular defects,—although the latter more frequently cause a local spasm of the muscles of the eyes, which can hardly be considered choreic. It has been claimed that chorea may result from the reflex irritation arising from adenoids of the pharynx and enlarged tonsils. Whether this is directly or only indirectly a cause is not evident. The association of the two conditions is not very infrequent.

Hereditary influence is of considerable importance in the production of chorea. It is much more frequent in children of neurotic families, and very often several successive generations, or several children in the same family, may suffer from the disease.

The exciting cause of chorea in a certain proportion of cases is fright; occasionally it arises from imitation, and the disease has been known to occur epidemically in institutions. Choreiform movements may follow hemiplegia. Chorea and epilepsy may be associated in the same patient, or one disease may follow the other.

The causes which underlie the occurrence of chorea therefore, seem to be a rheumatic diathesis, a neurotic constitution, anæmia, and some severe disturbance of general nutrition. When these predisposing factors are present, an attack may be induced by many things. The greater the predisposition the less important may be the exciting cause. A very large number of the cases of chorea are in children who present distinct evidences of rheumatism, although the explanation of this relationship is not yet understood. In another group the neurotic element predominates, and in these there may be no connection whatever with rheumatism.

Pathology.—The exact pathology of chorea is at the present time not settled. The seat of the morbid process is undoubtedly the central nervous system, probably the motor areas of the cortex. The cases associated with rheumatism are now generally regarded as of infectious origin. In some severe cases which were fatal, owing to association with acute endocarditis, capillary emboli have been found in the brain. However, it is by no means established that this is the condition present in most of the rheumatic cases. The fact that in the great majority of such cases complete recovery occurs in the course of a few weeks or months, speaks strongly against any important structural change in the nervous centres. In cases not rheumatic, the most probable explanation of the symptoms is to be found in vascular changes, having their origin in disturbances of nutrition.

Symptoms.—An attack of chorea generally comes on gradually. At first the child may be considered simply as unusually nervous; if at school, there may be noticed a difficulty in writing, drawing, or in using the hands for other delicate operations. At home, the child is continually dropping things, has difficulty in feeding himself, sometimes in buttoning his clothes, and very frequently he is not brought to the physician until the symptoms have lasted a week or two. Sometimes the legs are first affected, and a history is given of frequent falls, a stumbling gait, difficulty in going upstairs, etc. At other times the spasm is first seen in the facial muscles, with disturbance of articulation, twitchings of the eye muscles, and the child may be punished for making grimaces. In most cases the spasmodic movements soon extend to all parts of the body. According to Starr, they remain limited to one side of the body (hemichorea) in about one-third of the cases. When fully developed, the movements of chorea are quite unmistakable. They are irregular, jerking, spasmodic, never rhythmical, rarely symmetrical, and vary in intensity from an occasional muscular contraction to almost constant motion. The movements are not under the control of the patient's will, and are usually intensified by efforts to repress them. They are increased by excitement, embarrassment, or fatigue, but do not continue during sleep.

Very often there is some weakness of the affected muscles, which may be so great as to lead to the suspicion that actual paralysis exists. Not infrequently I have had patients brought to the clinic for supposed paralysis, either of one extremity or of one side of the body, where the choreic movements have not been severe enough to attract the attention of the mother. This paralysis usually disappears in the course of a few weeks.

In severe forms of chorea the patient may be unable to help himself or even to walk. The symptoms may be so intense as even to endanger life. Such cases, however, are dangerous, not from the choreic movements, but from the acute endocarditis with which they are frequently associated.

The mental condition of choreic patients is one of marked irritability. They are fretful, emotional, easily provoked to tears or laughter, and difficult to control. In extreme cases a mental disturbance bordering upon acute mania has been observed. In other cases the facial expression and manner of speech strongly suggest beginning imbecility. All degrees of speech disturbances are seen from the slight difficulty in articulation due to inability properly to control the movements of the tongue and lips, to a condition in which speech is almost impossible. In rare cases speech has been temporarily lost. Heart murmurs are frequent in chorea. Some of these are of anæmic origin, some possibly are due to chorea of the heart-muscle itself—although this is a matter of some uncertainty—but a large number, probably the majority, are due to concurrent endocarditis, as is shown by the fact that they are permanent, and are followed by all the signs of organic heart disease. During every attack the heart should be closely watched, especially in children in whom there is a strong predisposition to rheumatism.

The urine in chorea has recently been studied with care by Herter and Smith, who have shown that in very many cases there is an excessive elimination of uric acid. This is neither the cause nor the effect of the chorea, but is to be regarded as evidence of a profound disturbance of nutrition, of which the choreic movements are but another manifestation.* The general condition of choreic patients is usually much below normal. They are anæmic; the appetite is poor, often capricious; they sleep very badly; they suffer frequently from headaches; they are easily fatigued by slight muscular exertion; and in short they have all the symptoms of a greatly disturbed nutrition.

Course and Duration.—The ordinary form of chorea tends to spontaneous recovery in from six to ten weeks. Exceptionally it may last for three or four months. In a small number of cases the disease may become chronic and continue indefinitely. Certain forms of local spasm, particularly choreiform movements of the muscles of the face, eyes, or neck, may be permanent. In any case of chorea which lasts longer than the usual time, the patient should be carefully examined for some cause of peripheral irritation. The tendency to relapses and second attacks is very marked. Later attacks are likely to occur in the spring succeeding the first illness, and in a small number of patients attacks may come every year for four or five years.

Diagnosis.—There is little difficulty in recognising chorea from the sudden, irregular, spasmodic contraction of the muscles coming on under the circumstances indicated. No other movements of childhood are likely to be confounded with it. The form of chorea following hemiplegia is usually more athetoid than choreic, yet at times it closely simulates ordinary chorea. The difficulty in distinguishing between the two is often increased by the fact that the weakness of simple chorea may, if unilateral, closely simulate hemiplegia. The existence of rigidity, contractions, and increased reflexes belongs exclusively to hemiplegic cases, and these will usually suffice to clear up all doubt with reference to the diagnosis.

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* Dr. Herter has called my attention to the fact that in many cases of well-marked chorea the urine contains a peculiar reddish colouring matter called hæmato-porphyrin. This is also found in many cases of rheumatism, another evidence of the close relationship existing between these two diseases.

Prognosis.—As a rule this is favourable, and complete recovery can be predicted, the exceptions being few in number. Parents should always be warned of the tendency of the disease to return in succeeding years, and the fact should be stated that in a certain proportion of cases the disease may be permanent. The prognosis of the cardiac murmurs occurring in chorea should always be guarded, although some of these are functional and disappear with recovery from the chorea; but the number of those which do not disappear is sufficiently large to make one always apprehensive as to the ultimate result. Acute chorea accompanied with endocarditis may be fatal; a number of such cases are on record in which there was no other evidence of rheumatism.

Treatment.—The general management of the case is equally important with the administration of drugs. A child with chorea should at once be taken from school, and should never be subjected to punishment or to ridicule on account of the movements. Special attention should be given to the patient's diet and general nutrition. Tonics, especially iron, are indicated in most cases. The food should be simple and nutritious, and all stimulants, particularly tea and coffee, should be absolutely prohibited. While fresh air is desirable, exercise should be prescribed with great caution and its effect should be carefully watched. It should never be carried beyond the point of slight fatigue. A certain amount of moral restraint is absolutely necessary; thus it often happens that choreic patients do very badly at home where they are indulged and receive sympathy, while in a hospital, where they are under restraint and made to control themselves, they begin to improve immediately. Gymnastics, although useful in some of the milder cases, may do positive harm in those which are severe. They should be regularly and systematically practised twice a day, but not continued too long. In all severe cases the " rest treatment " should be employed, which is equally beneficial in the milder ones; the patient is put to bed, and complete mental and physical rest secured. This may be combined with gentle massage for fifteen or twenty minutes a day. The daily use of warm baths, either alone or in conjunction with massage, is decidedly beneficial. In other cases the regular use of cold sponging is of the greatest value.

With reference to the use of drugs, it is advisable to separate from other cases those in which the connection with rheumatism is very close. In the rheumatic cases, salicylate of soda is often efficient, while the drugs usually employed may be absolutely without effect. In a case recently under observation, arsenic had been continued for two weeks without the slightest improvement, when the patient had an intercurrent attack of subacute rheumatism for which salicylate of soda in full doses was given, with the effect of controlling the choreic symptoms promptly and permanently. In the non-rheumatic cases, arsenic is almost universally admitted to be the most valuable remedy we possess. The method of administration is important; failure frequently results from the use of too small doses. Beginning with four drops of Fowler's solution three times a day for a child of eight years, the daily quantity may be increased by one drop each day until a disturbance of the stomach or bowels is produced, with puffiness under the eyes. The drug should now be stopped for two or three days, and then the same doses resumed and gradually increased, usually up to eight drops three times a day, sometimes to ten, and even twelve drops, unless the movements cease before that time; but when this occurs the drug should be stopped. Arsenic should always be given after meals, and largely diluted, the dose being taken in a full glass of water, but not necessarily drunk at one time. The possibility of arsenical poisoning should be remembered, although it is extremely rare. Semple has reported a case in which multiple neuritis and general pigmentation of the skin occurred after four weeks' administration of the drug.

In the event of the failure of arsenic alone, it should be combined with the rest treatment. Drugs which sometimes succeed where arsenic fails are antipyrine and strychnine. From fifteen to twenty grains of antipyrine should be given daily in divided doses to a child of eight years. There are a certain number of cases in which striking improvement follows the use of this drug if given in the full doses mentioned. To a child of eight years strychnine should be given in doses of $\frac{1}{60}$ of a grain three times a day, the dose being gradually increased until double this quantity is given; sometimes even larger doses than these are well borne. Galvanism is of some value in cases not relieved by drugs. Acute chorea of great severity may require opium, bromides and chloral, or even chloroform.

In estimating the value of drugs in the treatment of chorea, the natural course of the disease should be kept in mind, since those drugs which are taken after the third or fourth week are much more likely to be thought beneficial than those used in the early period of the attack.

There is no doubt that chorea may be dependent upon some ocular defect, and a correction of this will then form an essential part of the treatment, although few, if any, cases are cured by attention to the eyes alone.

Chorea has a strong tendency to recur, especially in the spring of the year. Children who have had one attack should be closely watched, particularly with reference to their work in school. They should not be crowded in their studies, they should have long vacations, and the nervous system should not be put upon any severe tension for a long time.

OTHER SPASMODIC AFFECTIONS.

Habit Spasm.—This term is used to describe certain spasmodic muscular movements which at first are only occasionally noticed, but which may persist until they become habitual and almost entirely involuntary. The movements usually affect the muscles of the face, but they may be seen in almost any part of the body. The most frequent varieties consist of blinking or sudden frowning, raising the eyebrows, or some peculiar grimace. At other times there is sudden twisting of the head, shrugging of the shoulders, or jerking of the hands. It is not often seen in the lower extremities, but the muscles of respiration are quite frequently affected. There may be a half-sigh, a sort of sob, or a peculiar dry, laryngeal cough.

These movements are at first infrequent; but as the habit becomes more firmly fixed the spasm recurs every few minutes, and in severe cases it may be almost continuous. The form of spasm is not always the same; one may disappear and another take its place. The condition may last for months or years, and it may even be permanent.

Habit spasm is really little more than exaggerated nervousness continuing in some definite form until by repetition a fixed habit is established. It is different in cause, course, prognosis, and treatment from chorea, with which, however, it is often confounded.

The causes are those of neuroses in general. In the beginning, at least, there is usually a somewhat depreciated general health. The patients are nervous children of neurotic antecedents. There may be a history of some definite exciting cause, such as illness or overwork in school. The spasm of the muscles about the eyes may be associated with pathological conditions of these organs.

Habit spasm is to be differentiated from chorea: this is usually easy, from the limitation of the movements to one part or group of muscles and from the duration of the disease.

Treatment is quite unsatisfactory after the habit has become fixed, hence it is of very great importance that it should be arrested at the earliest possible age. Punishments are of no avail, and usually aggravate the condition. Rewards are much more effectual. The general health should receive attention and nerve tonics should be given, especially strychnine.

Athetosis and Athetoid Movements.—This term, introduced by Hammond, is used to describe a chronic form of spasm usually seen in the hand, but sometimes also in the foot, and even the face. It may affect both sides, but in most cases it is unilateral. The movement is slow, irregular, and inco-ordinate—a sort of "mobile spasm," it has been called—and there may be associated a certain amount of muscular rigidity. Such movements may occur in persons otherwise healthy, but are usually seen as a sequel of cerebral palsies, generally hemiplegia. Recovery from the paralysis may be so nearly complete that the athetoid movements are looked upon as primary. In some cases the movements are more rapid and somewhat resemble those of chorea, the condition being sometimes classed as *post-hemiplegic chorea.* Athetosis is not influenced by treatment.

Rotary and Nodding Spasm of the Head.—These are rare forms of irregular movements usually observed in infancy. The condition was described long ago by Henoch, and since then cases have been reported by Hadden,* Peterson, and others. The most frequent is the rotary spasm, which consists in a side-to-side oscillation of the head, which may be slow or rapid, and in some cases is almost continuous. Some children have at times the nodding spasm also, and in others this is the only movement seen. Nystagmus is frequently associated, and may affect one or both eyes. In a few of the reported cases convergent strabismus was present.

The causes of the condition are extremely obscure. It is usually seen in infancy between the third and eighteenth months, and, like most nervous symptoms of this period, has been ascribed to dentition, but without any special reason. In three of the cases reported by Hadden, it followed an injury to the head, and might perhaps be regarded as a result of cerebral concussion.

As a rule, the condition lasts for several months and improves, recovery generally taking place. The prognosis is therefore usually favourable. In most of the reported cases improvement has followed the use of bromides; from ten to twelve grains daily should be given.

Nystagmus.—This term is applied to rhythmical, involuntary, oscillatory movements usually of both eyes. They are caused by the alternate contraction of opposing muscles. Nystagmus may be either vertical or horizontal. It is most often seen in infants a few months old, and is a symptom of irritation which may be general or local. In some cases the movement is almost continuous, occurring even in sleep; in others, it is only noticed at times of special excitement.

The etiology of nystagmus is obscure, and it may occur in quite a variety of conditions,—sometimes referable to the eye, at other times to the central nervous system. On the part of the eye, nystagmus may be due to blindness from any cause, to congenital cataract, corneal opacity, disease of the choroid or retina, or to errors of refraction. It may be seen in almost any organic disease of the nervous system, both with focal and diffuse lesions, especially in chronic hydrocephalus, insular sclerosis, tuberculous meningitis, and in diseases in which sight is impaired. Nystagmus may be of reflex origin, as in a case recently occurring in the Babies' Hospital, where an infant with a severe diarrhœa had repeated attacks, which disappeared each time after intestinal irrigation. While it is of no importance as a localizing symptom, nystagmus usually indicates something more than functional disturbance. An exception to this may perhaps be made when it follows cerebral concussion. In such cases it is usually temporary, disappearing in a few days or weeks. Under most other conditions it may continue indefinitely.

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* Lancet, June 14, 1890.

The condition of the eyes should be investigated in every case of nystagmus; it is only when the cause is here, and can be removed, that habitual nystagmus is amenable to treatment.

Hiccough (Singultus).—This is a spasm of the diaphragm which is usually seen in young infants. In them it is in most cases due to some irritation in the stomach. It is seen after eating, and may depend upon overfilling of the stomach by food, swallowing of air, etc. In other cases it has no relation to the taking of food, and is to be regarded as a form of reflex spasm, which may occur from a variety of causes, such as cold feet, chilling of the surface during bath, or suddenly taking an infant from a warm bed into a cold room. In cases like the above, hiccough, though sometimes annoying, is of little importance. It may be associated with gastric indigestion, with intestinal flatulence or inflammation, with peritonitis or intestinal obstruction. With the last two conditions it is always an unfavourable symptom. In older children hiccough sometimes occurs as a pure neurosis.

The object of treatment is to remove the cause. In infants this is to aid in the expulsion of the gas from the stomach by manipulation, position, or the other means useful in gastric colic. Where it is a nervous symptom only, it may be arrested by holding the breath, by prolonged forced expiration, as in blowing a trumpet, and sometimes it may be relieved by drugs which control muscular spasm—e. g., antipyrine or chloral.

Thomsen's Disease (Congenital Myotonia).—This rare disease is usually congenital. It may occur in several members of the same family, and is often hereditary. The characteristic symptoms are a peculiar rigidity of the muscles which is observed when they are first brought into action after repose. This rigidity is spasmodic, and usually continues but a few moments. It may recur when voluntary movements are again attempted. If, however, muscular effort is persisted in, it soon passes off. It is increased by apprehension, excitement, or cold, and by observation. The legs are most frequently affected, the condition being often noticed when the patient starts to walk; any of the voluntary muscles, however, may be involved. It may be greater upon one side of the body than upon the other. The muscles are abnormally sensitive to mechanical stimulation, and often to galvanism. They are above normal size, and the fibres themselves are enlarged.

The pathology of this disease is, according to Gowers, an altered functional condition of the muscle fibres, and an abnormal functional state of the nerve cells of the cord and the cortex. It is incurable, although the symptoms may be improved by active muscular exercise.

Cervical Opisthotonus.—This is usually a symptom of disease at the base of the brain, occurring with simple, tuberculous, and chronic basilar meningitis, sometimes with tumours of the posterior fossa of the skull. However, in certain cases it occurs as a form of reflex spasm, particularly in young infants who are suffering from diarrhœal diseases or marasmus. In these cases it may last for days or weeks. The deformity is produced by a contraction of the superior fibres of the trapezius and by the posterior group of cervical muscles.

Torticollis—Wry-Neck.—Torticollis is usually produced by a tonic spasm of one sterno-mastoid muscle, with which may be associated spasm of the posterior cervical muscles, including the trapezius. In recent cases there is simply a condition of muscular spasm; in those of long standing there may be permanent shortening of the affected muscle, atrophy, and partial paralysis. A somewhat similar deformity may be caused by cicatricial contraction of the tissues of the neck following burns.

The deformity varies somewhat according as the sterno-mastoid muscle is alone affected, or the posterior muscles also, and as to which predominates. In simple sterno-mastoid spasm the head is inclined to the affected side and rotated toward the opposite side; the chin is raised, and the ear approaches the clavicle. When other muscles are involved the deformity is modified. If the trapezius is affected (Fig. 122) there is less rotation of the head, but it is drawn to the affected side and somewhat backward, while the shoulder is raised and the spine curved. Both of these symptoms may be seen to a slight degree in almost any marked case of sterno-mastoid spasm. Sometimes the spasm of the posterior muscles affects both sides; the head is then drawn backward and held rigidly but without rotation. In most of the recent cases the deformity can be partially or entirely overcome by passive force; but after a time this is impossible, owing to muscular shortening. In recent cases also localized pain and tenderness are frequently present, and sometimes they are severe.

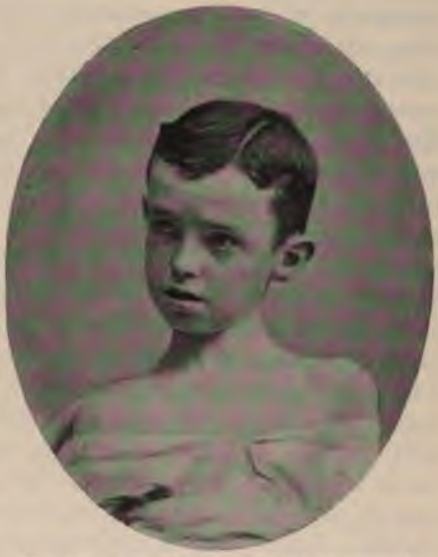

FIG. 122.—Spasmodic torticollis from malaria. Trapezius and sterno-mastoid of the left side are affected.

Etiology.—Spasmodic torticollis may be produced by anything causing irritation of the trunk or the branches of the spinal accessory nerve; the source may be in the spinal canal, in the cranium, along the course of the nerve trunk, or of any of its peripheral fibres.

Cases are usually divided into congenital and acquired. Whitman,* from the records of the Hospital for the Ruptured and Crippled, New York, for nineteen years, gives the following statistics of 264 cases,—torticollis from Pott's disease not being included: Males, 109; females, 155; congenital, 32; under two years, 33; from two to ten years, 153; over ten years, 46; acute (i. e., of less than two months' duration), 77; chronic, 60, of which number 22 had lasted two years or longer.

Regarding the cause of congenital torticollis there is some dispute. Such cases have often been attributed to the contraction resulting from hæmatoma of the sterno-mastoid (page 96). My own experience coincides with Whitman's, that this is rarely if ever the case. While it is possible that the deformity is sometimes the consequence of injury received during delivery, the cause of most of the congenital cases goes back to conditions existing before birth. It may be compared to club-foot, and may be due to a faulty position of the child *in utero*, or it may come from more serious conditions, such as malformations, or unequal development of the two sides of the body.

One of the most frequent causes in the acquired cases, is irritation of the spinal accessory nerve by an enlarged cervical lymph gland; this was the cause assigned in nearly half of Whitman's cases; such is the usual etiology of torticollis following scarlet fever, measles, or diphtheria. I have seen it in the early stage of quinsy, and it may occur in cellulitis of the neck. A cause which the physician should always have in mind is cervical Pott's disease; torticollis may be the earliest, and for several weeks sometimes almost the only, objective symptom of this disease. Torticollis coming on acutely is most frequently due to cold (rheumatism?) or malaria. I have notes of eight cases clearly traceable to malaria, and have seen at least a dozen others. In several of these there was a distinct periodicity in the spasm, it recurring regularly at about the same time each day until quinine was given; in some cases it was accompanied by fever, in others not. In the so-called rheumatic torticollis, muscular pain and soreness are rather more prominent than in the other forms. In fourteen of Whitman's cases the spasm was attributed to injuries other than burns; and in only nine was it associated with some other disease of the nervous system, most frequently with chorea.

Prognosis.—The result in a case of torticollis depends upon the cause, the severity, and the duration of the deformity. Most of the acute cases from malaria, rheumatism, etc., recover, under appropriate treatment, in the course of a few weeks, sometimes in a few days, and not a few recover spontaneously. The congenital cases with slight deformity are usually amenable to mechanical or postural treatment if begun early. There is, however, in most of the other varieties a disposition of the deformity, if untreated, to persist, and even to increase. If it has lasted several months the probabilities of spontaneous recovery or even of improvement are small.

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* Observations upon Torticollis, Medical News, October 24, 1891.

Treatment.—The first indication is to remove or treat the cause where one can be found. Malarial cases require quinine; rheumatic cases are benefited by rest in bed, hot applications, counter-irritation, friction, and sometimes by anti-rheumatic remedies. Cases which have lasted a month usually require some orthopædic head-support, and those which have lasted six months or more are rarely cured without a surgical operation. This may be either a subcutaneous tenotomy or myotomy of the sterno-mastoid, or an open incision. Whitman gives the result of thirty-two cases admitted for treatment to the hospital mentioned, as follows: In 17 in which the deformity had lasted less than six months, 10 were cured, the average duration of treatment being three months; 4 were improved, and 3 not improved, the average duration of treatment in these cases being eleven months. Of 15 cases in which the deformity had lasted over six months, none were cured and only 6 improved, after an average of about eight months' treatment. In the foregoing series of cases the treatment consisted mainly in the use of orthopædic apparatus; later results from incision have been considerably more favourable. But these figures show how serious a matter is an old case of torticollis, and emphasize the importance of resorting to radical measures early in the disease.

HYSTERIA.

This is not a disease of childhood, but one which is occasionally seen in early life. All that will be attempted in this chapter is to point out the most common manifestations of hysteria when it occurs in young children. After puberty it is essentially the same as in adults.*

Etiology.—Hysteria is very rare before the seventh or eighth year, and most of the cases seen in children occur after the tenth year. As to sex, there is no such predominance of females as in later life, although even in childhood they are more frequently affected than males. Hereditary influences play an important part in the production of this disease. It is seen in children who inherit a nervous constitution, or in whose parents nervous diseases, such as insanity, or hysteria, or alcoholism have been present. Of the other etiological factors the most important are a disordered nutrition, frequently with anæmia or chlorosis, and overpressure in schools. Masturbation or phimosis may act as an exciting cause, or, indeed, anything which leads to an exalted nervous irritability and depreciation of the general health. It is occasionally associated with tuberculosis; it may follow any of the acute infectious diseases; or it may be excited by injury, fright, or imitation.

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* For a fuller discussion of this subject, and references to recent literature, see Mills, in Keating's Cyclopædia, vol. iv.

Symptoms.—There is scarcely any disease in which the clinical picture presented is so varied as in hysteria. It may simulate almost any form of organic disease of the brain, lungs, digestive organs, bones, or joints. The most common symptoms may be grouped under four general heads. These are, however, seen in almost every conceivable combination.

1. *Psychical symptoms.*—Where these predominate there may be seen periods of mental depression of longer or shorter duration, a change in disposition, an indifference to surroundings, a capricious humour, or a nervous condition of extreme irritability with irregular paroxysms of laughter or weeping without cause. There may be great excitability of temper, and fits of passion almost maniacal in their severity. There may be various hallucinations. Sleep is frequently disturbed, sometimes by attacks resembling ordinary night-terrors; sometimes somnambulism is present. There is often a disposition to deception about the most trivial matters, which may last for weeks. There is a tendency to imitate the symptoms of various diseases, which the patients may have witnessed in others or about which they have read.

2. *Sensory symptoms.*—These are the most frequent manifestations of hysteria in early life. There is often general or local hyperæsthesia, which may be so great as to simulate inflammation of the various internal organs. Anæsthesia is much less common, although it may be seen in children as young as eight or nine. Headache is an occasional symptom, and is sometimes associated with great tenderness of the scalp. There may be neuralgias in the different parts of the body, or sharp epigastric pain, sometimes accompanied by vomiting. Sometimes the special senses are affected, giving rise to hysterical blindness or deafness, usually of short duration.

3. *Joint symptoms.*—These are really a variety of sensory disturbances. They are not uncommon, and are often most puzzling. The symptoms may be referable to the spine, or to any of the large joints, particularly those of the lower extremity. All forms of organic disease of these joints may be simulated, and these patients are often treated for months with orthopædic apparatus, with the belief that they are suffering from Pott's disease, lateral curvature of the spine, club-foot, or ostitis of the hip, knee, or ankle. Cases of this sort have been very fully described by Gibney,* and by Shaffer, whose articles should be consulted for fuller details. They are usually seen between the ages of ten and fourteen years, and occur in both sexes. There may be lameness referred to one of the large joints, curvature of the spine, or torticollis. The symptoms are most frequently

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* Gibney, Transactions of the American Neurological Association, 1877. **Shaffer**, Archives of Medicine, New York, December, 1879, February and April, 1880.

referred to the hip, and next to the knee, the ankle, or the spine. The pain is often acute. It is increased by motion, and by attempts at overcoming the deformity, if any is present. There is a marked hyperæsthesia of the whole limb, and sometimes of the body. In nearly every case there is marked tenderness of the spine upon pressure, especially in the dorsal region. The deformity may be very slight from spasm of the flexors only, or it may be severe, and followed by contracture, so that the thighs may be flexed tightly against the abdomen with the heels against the buttocks. Such deformities may last for months. There may be considerable muscular atrophy, but only that which comes from disuse. A special difficulty in diagnosis arises from the circumstance that these symptoms occasionally follow an injury.

Organic disease of bones and joints may usually be excluded by attention to the following points: The mode of onset is more abrupt than is seen in bone disease, and the course of the disease is quite irregular. The degree of deformity is greater than is seen in bone disease of the same duration. There are general hyperæsthesia of the limb, acute tenderness of the spine upon pressure, and undue sensitiveness to heat or cold. The deformity varies from time to time, being always more marked when examination is attempted. If the patients are closely watched, other evidences of hysteria may be seen. Under complete anæsthesia the contractures may disappear entirely. There is no enlargement of the articular ends of the bones, no swelling of the soft parts, and no evidence of active inflammation or of suppuration. All the symptoms except the deformity are subjective. Under proper treatment there is in most cases perfect recovery, often in a surprisingly short time.

4. *Motor and convulsive symptoms.*—In the milder forms we may see many varieties of tonic or clonic spasm. There may be seen local spasm of the eyes, face, or mouth, spasm of the muscles of the neck producing torticollis, of the muscles of respiration causing dyspnœa, which may be constant or paroxysmal. There may be hiccough, or spasm of the larynx causing hysterical aphonia. A very common symptom is hysterical cough, which may be so frequent and so severe—even accompanied by hæmoptysis—that grave disease of the lungs is suspected; the chest, however, is free from the physical signs of disease. There may be frequent attacks of vomiting with eructations; these may be continued sometimes even for months, and in rare instances blood has been vomited. There may be dysphagia from spasm of the œsophagus, or regurgitation of food on attempts at swallowing. In more severe cases we may have the symptoms of chorea major and attacks of hystero-epilepsy. The latter are rare in children and do not differ essentially from such attacks in older patients. There are usually prodromal symptoms. The convulsive movements are exceedingly varied in type. There are painful sensations and sensitive areas, by pressure upon which hysterical symptoms may be in-

creased or even convulsions excited. The respiration may be rapid or irregular. All variations in tonic and clonic spasm may be seen. Opisthotonus is frequent. Consciousness is not fully lost, but is disturbed, and hallucinations are present. The temperature is normal.

Hysterical paralysis is not common in children, but it may be seen even in the very young. Gillette has reported the case of a child eighteen months old who exhibited the symptoms of hysterical palsy of one arm. Other symptoms occasionally seen in hysteria, are persistent anorexia, polyuria, sometimes incontinence of urine, disturbance of the secretion of saliva or perspiration, and very rarely hysterical fever.

The general condition of hysterical patients is usually below the normal. They are poorly nourished and anæmic; they sleep badly; they have capricious appetites, feeble digestion, and faulty assimilation.

Diagnosis.—Hysteria is apt to be overlooked because its occurrence in children is not considered as often as it should be. In most cases the diagnosis is easy if hysteria is suspected. A combination of vague disconnected symptoms is usually present which admits of no other explanation. Organic disease can be excluded only by careful and repeated examinations. It is to be borne in mind, however, that hysteria not infrequently complicates organic or constitutional disease. Much importance is to be attached to a family history of hysteria or of other neuroses. From poliomyelitis, hysterical paralysis is differentiated by the presence of faradic contractility even though atrophy exists. Hysterical convulsions are differentiated from true epilepsy by the absence of any elevation of temperature, of biting of the tongue, evacuation of the viscera, of a violent fall, and often by the rapid disappearance of the symptoms under appropriate treatment.

Prognosis.—This is better than in adults, especially if the cases are taken in hand early, before the disease has become deeply seated. Very much depends upon how well the directions for treatment can be carried out. The prognosis is less favourable where the hereditary tendency is strongly marked. In many cases there are relapses later in life.

Treatment.—Prophylaxis is of much importance. When a hereditary tendency to nervous diseases exists in a family, or whenever very nervous children are placed under the physician's care, every means should be taken toward muscular development, keeping the nervous system in the background. Such children should lead an out-of-door life as much as possible, preferably in the country; they should keep early hours, have regular exercise, and their education should be directed with moderation and judgment; special attention being paid to regularity of work, and the prevention of overpressure in schools. Theatres and exciting books should be avoided. All stimulants, including tea and coffee, should be absolutely forbidden. The diet should be plain and nutritious. It is highly important that such children should be removed from association with a hysterical mother, when this is possible.

In the general management of a case of hysteria, it is of the first importance that the child should be cared for by a person of firmness, who can exercise proper control. Hysterical children are always managed more easily when they are removed from their homes and placed under the charge of a good trained-nurse. Isolation is absolutely essential in many cases. The general health should be carefully looked after, and arsenic, iron, cod-liver oil, and other tonics given according to indications. Horseback exercise and other out-of-door sports should be encouraged, and every means taken to interest the child in something which requires physical exercise. In cases of simulated disease, the child should be put to bed, no books or toys allowed, and no effort made toward his amusement. No sympathy should be exhibited, but the child should be treated with kindness and firmness. This moral treatment is quite as important as any other part of the therapeutics. In cases with hysterical joint symptoms the most valuable thing is counter-irritation to the spine, preferably by the Paquelin cautery. Some cases are benefitted by galvanism. The moral effect of hypodermics, even of cold water, is sometimes striking. Under no circumstances should mechanical force be used to overcome deformity. Many cases of hysteria improve under hydrotherapy; the cold douche, the cold pack, or the shower bath may be used. This is valuable in conjunction with massage and the " rest treatment."

In attacks of hystero-epilepsy the cold douche may be used, or pressure made upon the testicle or ovary. In severe cases ether may be given. In all hysterical cases the condition of the bowels should receive careful attention, as these patients are very prone to obstinate constipation.

HEADACHES.

Headaches are not common in little children except in connection with disease of the brain or meninges; in older children they occur from causes similar to those seen in adult life. The most frequent headaches may be grouped in the following classes:

1. *Toxic headaches.*—Such are the headaches resulting from uræmia, from carbonic acid in poorly ventilated rooms, and from malaria. But the largest number are due to absorption of toxines from the intestines, and are associated with chronic indigestion and constipation.

2. *Headaches from anæmia and malnutrition.*—These are most frequently seen in girls from ten to fourteen years old. Some are intellectually bright, and have been crowded in their school work; others are dull and learn only with difficulty, and in consequence worry over their work until their health becomes undermined. They sleep badly, lose appetite, and often become choreic. The anæmia may be either the cause or the result of these symptoms. The urine in these cases often contains a large excess of uric acid.

3. *Headaches of nervous origin.*—These may occur in children who are highly neurotic, either from their inheritance or surroundings, and in those who are the subjects of epilepsy or hysteria, and they may be symptomatic of organic disease of the brain, such as tumour or tuberculous or syphilitic meningitis. True facial neuralgia is rare in childhood except from carious teeth; from this cause, however, it is not infrequent.

4. *Headaches due to disease of some of the organs of special sense.*—In connection with the eyes there may be conjunctivitis, keratitis, iritis, errors of refraction, or strabismus; connected with the nose there may be polypi, hypertrophic rhinitis, or adenoid vegetations of the pharynx; connected with the ears there may be otitis or foreign bodies in the canal. Each one of these conditions requires special treatment.

5. *Headaches due to inherited gout or rheumatism.*—These are not very frequent, but they may be severe, and may at times simulate the onset of meningitis. They are often accompanied by pains in the joints, muscles, or nerve trunks; they may be associated with a urine which is highly acid and contains deposits of oxalates or of free uric acid.

6. *Disturbances of the genital tract* are rarely a cause of headaches in children, although this may be the case in girls about the time of puberty, especially where menstruation is delayed or difficult.

Diagnosis.—The diagnosis of headaches includes the discovery of the cause, and this is often difficult. In an infant or a young child, organic disease of the nervous system should always be suspected as a cause of severe headaches. In older children the important things to be considered, because the most frequent, are digestive disturbances, nervous exhaustion, malnutrition, and visual disorders. An absolute diagnosis in a case of persistent headache can be made only by a careful physical examination, not omitting a study of the urine; often there must be a close observation of the patient for some time.

Treatment.—The only successful treatment is that which is directed toward a removal of the cause. Each one of the different groups above mentioned is to be managed differently, according to the principles elsewhere laid down regarding the treatment of these conditions. For the relief of the symptom, cold to the head, a hot foot-bath, and phenacetine in moderate doses are perhaps the most certain of all remedies.

DISORDERS OF SPEECH.

In this chapter will be discussed only functional speech defects,* those depending upon organic conditions being considered in connection with diseases of the brain. The most common varieties are stuttering, stammering, lisping, alalia, backwardness, and functional aphasia. All forms are much more frequent in boys than in girls, the proportion being more than four to one.

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* See Wyllie, Edinburgh Medical Journal, October, 1891.

Stuttering.—This is the most common form of speech disturbance. Articulation is distinct and the separate sounds are properly produced, but there is a difficulty in connecting the consonant with the succeeding vowel; this seems like an obstacle to be overcome. Stuttering is occasionally seen in most children. It is more frequent in the third and fourth years, before speech is thoroughly mastered. At this age it is aggravated or produced by disturbances of nutrition, but is usually of temporary duration, lasting for a few weeks or months. Only recently a little boy of four was under my care, who became very anæmic, slept poorly, and suffered from malnutrition as a result of the confinement incident to a home in the city. He soon began to stutter, and in a short time it became painfully marked. After a few weeks in the country he improved very much in his general condition, gained four or five pounds in weight, and his stuttering completely, and I think permanently, disappeared. Such disturbances as this are analogous to chorea. In other cases stuttering follows some acute illness, and under such conditions also it is usually of short duration.

Most children who become habitual stutterers do not begin until they are six or seven years old, and sometimes even later. Stuttering may arise from imitation, and probably inheritance is an occasional factor. It is frequently a mark of degeneration.

It is important that all such cases receive early treatment before the habit becomes firmly fixed. The prognosis is good for spontaneous recovery in nearly all the cases seen in very young children, and also in those coming on after acute illness. Other cases in which the condition has become habitual, should have the benefit of systematic training under a competent teacher in breathing, vocal and speech gymnastics.

Stammering.—This term is sometimes used synonymously with stuttering. Kussmaul makes the distinction between them that, in stammering, individual sounds are difficult of production, while in stuttering it is syllabic combinations. Stammering is often accompanied by some defect in the organs of articulation—the teeth, lips, tongue, or palate—which is not present in stuttering.

The treatment consists in careful training and in the correction of whatever abnormal local conditions may exist.

Lisping.—In this there is imperfect production of certain sounds, owing usually to a faulty position of the organs of articulation. The sounds may be so indistinct that they can not be understood. In this condition also there may be defective formation of some of the organs of articulation, although in the milder forms this is not the case. The treatment is similar to that of stammering.

Alalia.—This consists in a total inability to articulate. It is seen in all young infants during their earliest attempts at talking. In older children it is usually associated with some mental defect.

Backwardness.—Backwardness is carefully to be distinguished from a late development of speech due to idiocy. At two years old children not deaf are almost invariably able to speak. Speech may be late in consequence of prolonged or very severe illness, and where it has been acquired it may be lost from similar causes.

Functional Aphasia.—The term has been applied to a temporary loss of speech which sometimes occurs in chorea, and sometimes from severe fright or anything else which has produced a marked nervous impression. West records an instance in a girl of eight years, who was suffering from an attack of chorea induced by fright. Speech first became difficult and then was lost altogether. For a month the child could say only "Yes" and "No." The case improved very slowly, but at the end of nine weeks had recovered completely.

Loss of speech sometimes follows the acute infectious diseases, especially typhoid fever.

In all disorders of speech, the functional cases are to be distinguished from those which depend upon deafness and mental deficiency. The frequency with which these disorders are due to disturbances of general nutrition, and to local causes in the mouth and throat, should be borne in mind, and these conditions should receive their appropriate treatment early, before the habit of defective speech becomes firmly established. For the latter class of unfortunates, special training at the hands of a competent teacher should be advised, preferably in an institution.

DISORDERS OF SLEEP.*

Disturbed Sleep, Sleeplessness.—Disturbed or restless sleep is much more common in infancy and childhood than is true insomnia, although the causes of the two conditions may be the same.

Etiology.—In infancy these symptoms are most frequently due to hunger or to indigestion resulting from overfeeding or improper feeding. Very often disturbed sleep is the result of bad habits, such as rocking during sleep or night-feeding. Sometimes it arises from dentition, or the pain of colic or otitis; at other times it may be simply the expression of a condition of nervous irritability, the result of inheritance or of the child's surroundings.

In later childhood the first thing to be suspected when sleep is much disturbed is some derangement of the digestive organs; in this will be found the explanation of fully half the cases. The most frequent type, where the symptom is of long duration, is chronic intestinal indigestion, often associated with indicanuria, a condition in which the diagnosis of the mother is usually worms. Other cases are due to obstructed respiration from adenoid growths of the pharynx or enlarged tonsils, sometimes to nocturnal attacks of asthma. A lack of fresh air in the sleeping room, excessive or insufficient bedclothing, and cold feet, are other frequent causes. Disturbed sleep with "starting pains" is one of the earliest symptoms of hip-joint disease. In the nervous exhaustion resulting from overpressure in schools, and in malnutrition and anæmia, disturbances of sleep are well-nigh constant. They are also seen in organic cardiac disease and in all pulmonary conditions accompanied by dyspnœa or cough. Sleep may be disturbed in consequence of bad dreams which have their origin in exciting stories heard or read just before bedtime, or in too violent or exciting play. To discover the cause in almost any case it is necessary to investigate carefully the whole routine of the child's life.

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* For the characteristics of the sleep of infancy, and the average amount taken at the different ages, see pages 5 and 6.

Symptoms.—The condition may be one of real insomnia which may last for weeks or months; or the sleep may be simply disturbed and restless, the child waking many times during the night, and when asleep will not lie quietly, but constantly changes his position. Sometimes children wake suddenly with a scream, but immediately drop off to sleep again.

Treatment.—The essential treatment consists in the discovery and removal of the cause of the disturbance. This will often involve a radical change in the manner of feeding, in the hygiene of the nursery, and in all the surroundings of the child; but in this way only should these cases be managed. Under no circumstances should the physician countenance the use of drugs to promote sleep in children, except in the case of severe acute disease. Soothing syrups and all nostrums for "teething" should be absolutely forbidden. Mothers and nurses are only too ready to fall into the habit of using them, because the injurious effects are not appreciated. When the cause of sleeplessness is found and removed the child will sleep, but compulsory sleep obtained under other conditions is always productive of more harm than good. If food, diet, and all bad habits have been corrected, nervous causes should be investigated. When no cause can be discovered the treatment should consist in putting the child upon the simplest possible diet, and in attention to such general conditions as anæmia, malnutrition, and neurasthenia, some of which are almost certain to be present. In many cases a warm bath at bedtime will be found beneficial. A quiet, darkened room, plenty of fresh air, and the stopping of both eating and drinking during the night, are essential to a cure in most cases. When the condition accompanies some acute disease, the drugs which are most useful are codeine and trional. A child of two years may take $\frac{1}{20}$ of a grain of codeine or two grains of trional as an initial dose, to be increased if necessary.

Night Terrors—Pavor Nocturnus.—Two classes of cases have been grouped under this head, both having this in common, that sleep is disturbed by fright. In an excellent article upon this subject,* Coutts calls attention to the necessity of sharply distinguishing between them.

The condition in the first group partakes of the nature of nightmare. It may be due to partial asphyxia from adenoid growths of the pharynx, or to other causes mentioned under disturbed sleep, or it may be gastric or intestinal in its origin. These cases are quite frequent. Sleep may be disturbed from the outset, and the attack may be merely the culmination of such disturbance. The child wakes in a state of fright and excitement, and often says he has had a bad dream. His mind is clear, he recognises those about him, but it may be a long time before he is sufficiently calm to sleep again. The attack may be remembered perfectly the next day. Cases like this are to be managed in the same general way as those of disturbed sleep above mentioned.

In the second group are the only cases to which the term " night terrors " should really be applied. These are relatively rare, but the condition is a much more serious one. The symptom is due to some disturbance of the central nervous system. According to Coutts, it occurs especially in those of neurotic antecedents, or those who have previously suffered from infantile convulsions, and it is often the precursor of other nervous attacks—migraine, hysteria, epilepsy, and even insanity. The attack usually comes suddenly where a child has previously been sleeping quietly, and more frequently in the early part of the night than later. He is generally found sitting upright in his bed in a bewilderment of terror, being " afraid of the dog," or " the bear," or there is some other vision or hallucination which has produced the fright. Often this is associated with something of a red colour. The child does not recognise those about him, does not know where he is, and may go to sleep again without coming to full consciousness. The next day there is no recollection of what has happened. Usually no after-effects are seen, but sometimes a large amount of pale urine is passed. The attacks may be repeated at intervals of a few months, or they may occur every few nights; but whatever the peculiar nature of the vision, it is likely to be repeated in nearly the same form. Such attacks have something in common with epileptic seizures, and the diagnosis between them may at times be difficult. They are always to be regarded seriously, not only on account of what they are in themselves, but on account of what may follow.

Treatment.—All mental and nervous strain should be most carefully avoided, and where the attacks are frequent the bromides should be given at bedtime. Some person should sleep in the same room with the child, or in an adjoining one with the door open.

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* American Journal of the Medical Sciences, February, 1896.

Excessive Sleep.—It is rare that either infants or children sleep an unnatural amount of the time unless one of two causes is present—organic brain disease or the use of drugs. The latter is always to be suspected if with the sleep there is associated obstinate constipation. Opium in the form of "soothing syrup" or paregoric, is the drug which has usually been given.

INJURIOUS HABITS OF INFANCY AND CHILDHOOD.

On account of the close connection of such habits with disturbances of the nervous system, they may be properly considered with the functional nervous diseases. Although some of these habits may not be of serious importance, yet as a group they have received altogether too little attention at the hands of the physician.

Sucking.—This is a very common habit in infants, and during the first few months it is seen to some degree in most of them. If they are carefully watched the habit is easily stopped; otherwise it may continue indefinitely. Young infants usually suck the fingers when hungry, and this can scarcely be considered abnormal, but an effort should always be made to stop it, lest the habit become fixed. Lindner * distinguishes between simple sucking and sucking with combinations. In the former, the child sucks some part of the body, such as the thumb, fingers, toes, tongue, lips, back of the hand or arm, or it may be some foreign substance, such as part of the clothing, the blanket, a rubber nipple, or the "pacifier." This is the most common form that is seen. In the second variety the sucking is accompanied by the rubbing of some other parts, which seems to afford a pleasurable excitement; this may be the ear, the genitals, or any other portion of the body. Sometimes sucking is accompanied by some practice which produces actual pain, such as pulling of the hair or scratching the body. Habits of sucking often persist throughout infancy, and not infrequently throughout childhood; they have often been known to continue up to puberty. The longer the habit has lasted the more difficult is it to break.

The results of sucking may be serious. Deformities of the thumb or finger, of the lips and teeth, and even of the jaws, are sometimes produced. I know a lady, now in advanced life, whose thumbs to this day show a deformity resulting from the habit of thumb-sucking while a child. In her case the habit was not broken until she was eight or nine years old. Probably the most pernicious result of sucking is its tendency to develop the habit of masturbation. Habitual sucking of one hand or finger may lead to spinal curvature.

Treatment.—In the management of these cases the most important thing is to arrest the habit early, before it becomes fixed. Too often the habit of thumb-sucking, or of sucking a rubber nipple, is encouraged by mothers, nurses, and sometimes even by physicians because of the temporary quiet which is thereby produced. Under no circumstances should it be resorted to as a means of putting children to sleep or otherwise quieting the nervous system. With infants, the only treatment which is at all successful is mechanical restraint. It is of no use to cover the part which is sucked with bitter solutions. The hands of young infants may be covered with mittens, or with the long sleeves of a night-gown which is pinned to the bed, so that it is impossible for the child to get the part to the mouth; or pasteboard splints may be applied at the bend of the elbow, so as to prevent flexion of the arms. In the milder cases the habit is often discontinued spontaneously; but when it has been indulged until a child is four or five years old, it is broken only with the greatest difficulty. Punishments are of little avail, but rewards are often successful.

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* Jahrbuch für Kinderheilkunde, vol. xiv, p. 68.

Masturbation.—This is not uncommon even in infancy. Many cases have been observed during the first year, and some as early as the seventh or eighth month. It is seen in children of all ages and in both sexes; but in infants and young children it is, in my experience, much more common in girls than in boys.

Etiology.—Local causes are present in a large number of the cases, and this is usually something which produces undue irritation. The most frequent are, long or adherent prepuce, phimosis, balanitis, vulvovaginitis, eczema of the labia, threadworms, and tight clothing. A urine which is irritating because of excessive acidity or the presence of crystals of uric acid may be a cause. Any irritation may lead the child to rub the parts in some way, and a pleasurable sensation being excited, this action is repeated until a habit is formed. Other causes are exercises in which the legs are rubbed together, or the body against a pole, as in climbing. To these causes must be added, in infants at least, the habit of sucking. After infancy the habit of masturbation is usually acquired from other children, sometimes taught by vicious nurses.

General causes are also important as predisposing factors. These are the same as underlie most of the neuroses of childhood—viz., marked anæmia, general malnutrition, and a highly neurotic constitution, which is often an inheritance, and is always aggravated by surroundings which tend to unnatural stimulation of the nervous system. When masturbation develops in a young child without any local cause, it may be an early sign of either mental deficiency or moral delinquency; if looked for, other stigmata of degeneration will usually be found, and in most cases other vicious traits will soon appear.

Symptoms.—In infants and very young children masturbation is often accomplished by thigh friction or by rubbing the body against a pillow, chair, or some other object. The variety of ways is almost endless. Frequently the child will simply lie upon the floor with the thighs crossed and rigidly held, and only a backward and forward motion of the body made. This lasts for a few moments, is accompanied by flushing of the face and some appearance of excitement, followed by relaxation, and often by perspiration. It frequently happens with little children that these "queer tricks," as they are often regarded, have been continued for months before their true nature is suspected.

A consciousness that they are doing something wrong early leads even young children to get by themselves when they repeat the habit. It is especially likely to be practised when children lie long awake alone after they go to bed, or if they wake early. The habit is always made worse by any deterioration of the general health. I have known children, who were thought to be cured, to relapse under such conditions.

It is somewhat difficult to separate the general symptoms with which masturbation is associated, and upon which it largely depends, from those which are the direct result of the habit. There are some children in whom the condition is chiefly or entirely dependent upon a local cause, or when it is only occasionally practised, in whom no general symptoms are seen, or at most only an unnatural shyness and a disposition to seek seclusion. Others are precocious and excitable with an excessive amount of nervous sensibility. There are others in whom more marked nervous symptoms are present; the most striking are absent-mindedness, loss of power of concentration, loss of interest in all amusements, and mental depression. In some cases nymphomania, or even insanity, may be the result. Epilepsy, chorea, or hysteria may develop, particularly where a strong predisposition to them already exists in the family. The effect of masturbation upon the physical and mental development of the child may be serious when it is begun at an early age or is frequently practised. But even more striking is the change sometimes brought about in a child's moral nature. Even little children of eight or nine years may become centres of moral infection, which may involve a group of playmates or even a whole school.

Local symptoms of masturbation are not always present; in the male there may be redness and slight swelling of the prepuce; the organs may be abnormally large or simply much relaxed. In the female similar conditions may exist, and sometimes there is vaginitis.

Prognosis.—Masturbation in children is at all times a most difficult condition to deal with. The outlook is better in infants and young children than in those who are older, because the latter are more difficult to watch and control; besides, in them the habit has usually become more firmly fixed. In young children local causes are frequently found to be at the root of the trouble; in those who are older general causes are more often present, and these it may be impossible to remove. When masturbation is a symptom of degeneracy it is usually hopeless.

Treatment.—The most important thing is an early recognition of the condition. The physician should put parents and nurses on their guard, and the first suspicions should be reported and the child carefully watched until all doubt is removed. In young infants much may be accomplished by mechanical restraint. The kind of restraint which is necessary will depend upon the manner of masturbating. If by the hands, they should be tied during sleep, so that the child can not reach the genitals; if by the thigh-friction, the thighs should be separated by tying one to either side of the crib. In inveterate cases, a double side-splint, such as is used in fracture of the femur, may be applied. In children that are over three years old, all such contrivances are almost invariably unsuccessful. It is of the utmost importance in every case to have the child under the close surveillance of a competent and trustworthy person. He should be especially watched just after being put to bed and immediately after waking. Corporal punishment is often useful in very young children, but of little or no benefit in those who are over three years old. In fact, in such cases it may do positive harm, for deception and lying are soon added to the previous vice. The mother should secure the child's confidence, and in every way possible seek to strengthen his will and stimulate his self-control, using her influence to help him break the habit. The local causes, too, must be examined into and removed whenever found. Circumcision should be done if phimosis exists, and even where it is not, the moral effect of the operation is sometimes of very great benefit. In girls improvement sometimes follows a separation under anæsthesia of the preputial hood from the clitoris. If a dorsal slit is made in the prepuce a recurrence of the adhesions can easily be prevented. Complete circumcision is sometimes done with advantage,and in very obstinate cases the clitoris may be cauterized. Blistering the inside of the thighs, the vulva, or the prepuce is sometimes useful. Care should be taken that the clothing does not irritate the parts. The child should not only be removed from all vicious companions, but constant watchfulness should be exercised in the home and at school, that the child should have no opportunity to teach other children the habit. In the most serious cases the child should be sent away from home and kept from other children. The co-operation of a trustworthy nurse or companion is indispensable. General treatment should be directed to the child's condition; it is required in most of the cases. A child suffering from malnutrition and anæmia should be sent to the country, kept out of doors and away from books, studies, and from everything which stimulates or excites the nervous system. Almost all exercises except horseback may be recommended. Every means should be employed to build up the child physically. Cure results in most cases only by using all these measures and for a long time.

Nail-biting and Tongue-sucking are two forms of habit which are less frequent and less important than those already mentioned. The former is best remedied by keeping the nails cut very short. Tongue-sucking seldom becomes a fixed habit, and the child usually ceases it of his own accord as he grows older.

CHAPTER III.

DISEASES OF THE BRAIN AND MENINGES.

MALFORMATIONS.

The malformations of the brain are of great variety, and many of them are solely of anatomical interest, as the conditions are incompatible with life. Only the most frequent and the best-known types will be mentioned, and those which are of interest from a clinical point of view.

Meningocele, Encephalocele, and Hydrencephalocele.—These three conditions have in common a protrusion of some part of the cranial contents through an opening in the skull. In meningocele (Figs. 123, 126) there is protrusion of the membranes alone. These form a sac, which is usually, but not invariably, distended by fluid. In encephalocele (Fig. 124) there is a protrusion of a portion of the brain substance; this is connected with the rest of the brain by a constricted neck or pedicle. The tumour may or may not contain fluid. In hydrencephalocele (Fig. 125) there is a protrusion of a portion of the brain substance which contains within it a cavity filled with fluid, this cavity communicating with the distended lateral ventricles.

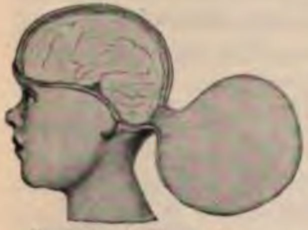
Fig. 123.—Meningocele.

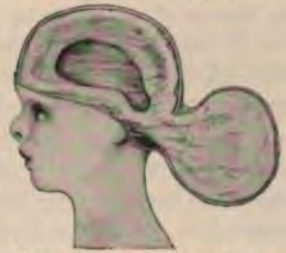
Fig. 124.—Encephalocele.

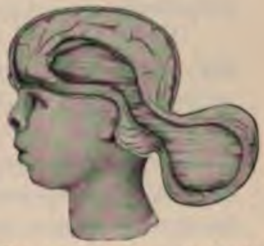
Fig. 125.—Hydrencephalocele

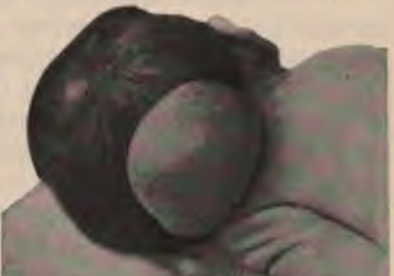
Fig. 126.—Meningocele.

From a patient in the Babies' Hospital. The autopsy showed that the sac communicated with the lateral ventricles.

In all these conditions there is a tumour, usually pedunculated, of a round or pyriform shape, with a smooth or lobulated surface. The ordinary size is that of a mandarin orange; it may be as small as a walnut, or as large as the patient's head. It is generally covered by the scalp, which is often denuded of hair; but it may be covered only by granulation-tissue, or it may show a central cicatrix, like that of spina bifida. Other deformities, such as spina bifida, club-foot, and hare-lip are frequently present.

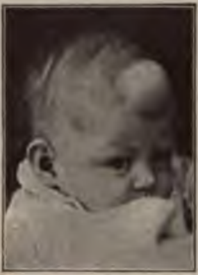
FIG. 127.—Frontal meningocele. From a patient in the Babies' Hospital.

All these conditions are rare, but the most frequent and most serious one is hydrencephalocele, this being usually associated with hydrocephalus. The next in frequency is encephalocele, which has the best prognosis. This is frequently termed *hernia cerebri.* If fluid is present, it is external to the brain. In meningocele (Figs. 126 and 127) there is simply an accumulation of fluid in the arachnoid cavity, which communicates by a small opening with the general arachnoid cavity of the brain.

Of one hundred and five cases collected by Schatz, fifty-nine occupied the occipital region and forty-six were frontal. The aperture through which the occipital protrusion takes place is usually in the median line. It may communicate with the posterior fontanel, with the foramen magnum, or with the cleft of a spina bifida. The occipital bone may be divided in the median line, or rarely it may be absent.

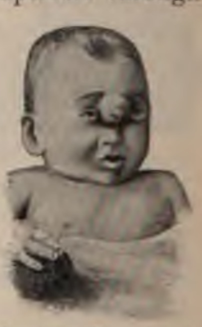
FIG. 128.—Naso-frontal meningocele (after Demme).

In the naso-frontal form (Fig. 128) the tumour is usually at the root of the nose, a little to one side of the median line. The aperture is most frequently between the cribriform plate of the ethmoid and the frontal bones. It may be between the lateral halves of the frontal bone, causing a median tumour. The point of protrusion may also be the lateral region of the skull, generally about the lateral fontanel, or along the line of the sutures; it may project into the mouth or the pharynx. These anterior tumours are usually small, although large ones containing the anterior lobes of the brain have been seen.

The theory of the origin of these malformations which is most widely accepted is that they are primarily cases of intra-uterine hydrocephalus, and as the cranial cavity is gradually closed by the development of the bones, a certain portion of the brain is left outside.

Symptoms.—The tumour is always congenital, although after birth it frequently increases very much in size. A typical tumour is round and elastic, usually giving evidences of fluid; it pulsates synchronously with the heart; during screaming or forced inspiration, it increases in size; partial and in some cases complete reduction is possible, but this is usually followed by marked cerebral symptoms, even by convulsions. After partial reduction, an opening in the skull may often be made out. Microcephalus may be present, or there may be unequal development of the two sides of the head.

The following differential points given by Treves, indicate the most characteristic features of the three varieties: In meningocele, the tumour is at first small, but increases; it has a smooth surface; it is pedunculated; there is distinct fluctuation, perfect translucency, rarely pulsation; often it is completely reducible; compression of the tumour causes cerebral symptoms; the skull is normal. In encephalocele, the tumour is small and smooth; it is rarely pedunculated; fluctuation is absent; it is not translucent; there is distinct pulsation; it is usually reducible; pressure causes cerebral symptoms; the skull is normal. In hydrencephalocele, there is a large pendulous tumour with an irregular or lobulated surface; it is pedunculated; translucency is rarely complete; fluctuation is distinct; it is irreducible; pressure rarely causes symptoms; microcephalus and other deformities are often associated.

The occipital tumours are usually more serious than the frontal ones. The majority of cases die in the course of the first few weeks of life, death resulting from meningitis, convulsions, or rupture. In meningocele the tumour usually grows slowly, and ultimately may be shut off from the cranial cavity; but gradual thinning of the membrane may take place, and spontaneous or accidental rupture occur. In encephalocele the tumour grows slightly, or not at all. Most of these patients exhibit signs of mental impairment or other evidences of organic brain disease.

Treatment.—According to Treves, operation is justifiable only in case of impending rupture. The conditions present are essentially the same as in spina bifida. Meningocele may be aspirated, injected with iodine, or with Morton's iodine and glycerin solution; the sac may be laid open and a plastic operation performed for the closure of the communication with the cranial cavity; or the skin may be divided, and a ligature or clamp applied to shut off the communication with the brain. All these methods have been at times successful, but cure has in many instances been followed by the development of chronic hydrocephalus. Encephalocele is to be treated by protection and compression. Aspiration may be resorted to if fluid is present. In hydrencephalocele the prognosis is absolutely bad under all circumstances. Schatz * gives the following statistics, showing the results with and without operation, all varieties being included: Of twenty-four occipital tumours not operated on, three recovered; of thirty-five operated on by excision, ligation, or injection, six recovered. Of forty-six frontal tumours, there were six recoveries in thirty-two cases without operation, and two recoveries in fourteen cases with operation.

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* Berlin. klin. Wochenschrift, No. 28, 1885.

Microcephalus.—This is generally regarded as due to premature ossification of the skull; but this theory is certainly inadequate to explain all the cases. In many children suffering from marasmus, the sutures ossify and the fontanels close much earlier than in healthy infants of the same age, chiefly because, with the rest of the body, the brain also has ceased to grow. So it is true of some of the cases, at least, of microcephalus, that the early ossification of the skull is due to arrested growth of the brain, and not the reverse. The reasons for the developmental arrest in the brain are for the most part unknown. The condition usually dates back to intra-uterine life, although in some cases it appears to begin after birth.

It is well known that there is not an invariable relation between the size of the head and the size of the brain, although generally the two correspond. If the circumference of the head is much below the average for the age (page 20), and relatively much less than the measurements of the rest of the body, microcephalus may be assumed to exist. Sachs calls attention to the fact that the circumference of the head may be nearly normal and yet the essential conditions of microcephalus exist, owing to imperfect development of the anterior part of the brain.

The symptoms of microcephalus are those of idiocy and cerebral paralysis, existing in all possible combinations and with variable degrees of severity.

A new surgical interest in these cases has been awakened during the last few years by the operation of craniectomy. The purpose of this operation, which was devised by Lannelongue, is to relieve the intracranial pressure by making a longitudinal opening in the skull, on one or both sides. The opening made is usually about half an inch wide and four or five inches long. It is one or two inches from the sagittal suture, to which it is parallel. For the time being the cranial capacity is increased, but it is doubtful if even this is permanent. Jacobi* gives a report of thirty-three cases operated upon by American surgeons, with fourteen deaths and nineteen recoveries. At the time of report the condition in the cases which survived the operation was as follows: no improvement in seven; slight, in seven; "some," in one; much, in two; no history, in one; uncertain, in one. I quite agree with him that such results do not justify the performance of this operation.

Congenital Hydrocephalus.—These cases may fairly be considered as belonging in this group, although they have been discussed elsewhere.

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* New York Medical Record, May 19, 1894.

Porencephalus (literally, a hole in the brain) is a condition in which there is a large depression in some part of the brain, but with surrounding parts well developed. Such depressions may involve a whole lobe, and they may be deep enough to reach the lateral ventricles.

Porencephalus is described as congenital or acquired. In the congenital form, the defect is usually found in the anterior or middle part of the brain. The origin of these conditions is still a disputed question. They are probably due to early vascular changes. Children sometimes live several years with very large defects, the symptoms depending upon the seat of the lesion. The acquired form of porencephalus is usually one of the late results of meningeal hæmorrhage. It may affect one or both sides. Such cases present the symptoms of spastic paralysis—usually diplegia. In all cases with large brain defects, the space is filled with fluid.

PACHYMENINGITIS.

Pachymeningitis, or inflammation of the dura mater, occurs both as an acute and a chronic disease.

Acute Pachymeningitis.—This is very rare in children. Only pachymeningitis externa is generally included under this term, as acute pachymeningitis interna does not occur alone, but usually with inflammation of the pia mater (leptomeningitis). It may be associated with disease or injury of the bones of the skull, but is most frequently seen in connection with middle-ear disease. It generally begins as a localized process, but the inflammation may extend to the inner layer of the dura, and to the pia mater; or it may remain circumscribed, and terminate in the formation of an abscess between the dura mater and the bone.

The symptoms of acute pachymeningitis are distinctive only when the process is localized. They are then usually associated with middle-ear disease, and are indistinguishable from those of cerebral abscess. The treatment is surgical.

Chronic Pachymeningitis.—This, in children, almost invariably affects the inner layer of the dura mater (pachymeningitis interna); it is also known as *pseudo-membranous* and as *hæmorrhagic pachymeningitis* or *hæmatoma of the dura mater*. Its causes are for the most part unknown. It is not very rare, being usually discovered at autopsy in children, chiefly cachectic infants, who have died of other diseases. In the Report of the New York Pathological Society for 1890 Northrup records six such cases. I have seen five similar ones, as well as one other associated with chronic hydrocephalus.

Two classes of cases are to be distinguished—those with, and those without extensive hæmorrhages. In the latter group there is found a thin, translucent, vascular membrane lining the inner surface of the dura. It may be only a delicate film which can be scraped off; it may be as thick as ordinary blotting-paper, or even twice that thickness. The membrane is often œdematous; it is exceedingly vascular, and the vessels have very thin walls. There are usually scattered, punctate hæmorrhages, and there may be a few of larger size. This membrane may cover the whole inner surface of the dura, but in most cases it is principally over the convexity and may be found only here; it is apt to be more upon one side than upon the other. In cases of long standing there may be adhesions between the dura and the pia. When large hæmorrhages have taken place, quite a different pathological appearance is presented. The lesions found in a case upon which I made an autopsy in the New York Infant Asylum, are fairly typical: The infant was six months old, and the symptoms had existed for six days. The fontanel was bulging to a marked degree, and the sagittal and coronal sutures were separated. A thin recent clot from one eighth to one fourth of an inch in thickness covered nearly the whole of the right hemisphere and part of the convexity of the left. The entire dura was lined both at its convexity and base by a pseudo-membrane of grayish color, about one sixteenth of an inch in thickness. The brain was anæmic.

In cases of longer standing partial organization of the clot may be seen; in more recent ones the blood is partly or entirely fluid. I once found acute leptomeningitis with a purulent exudation, associated with hæmorrhagic pachymeningitis. In cases where life is prolonged for years, there may be partial or even complete absorption of the clot, followed by the formation of cysts, considerable inflammatory thickening of the pia with deposits of blood pigment, and finally atrophy and sclerosis of the cortex. The source of the hæmorrhage may be the rupture of a single large vessel, but more frequently the blood comes from many small vessels.

Symptoms.—These are due to the hæmorrhage, and not to the inflammatory process. Until hæmorrhage occurs there are no symptoms by which the disease can be recognised. Thus in many of the cases in which pachymeningitis is found at autopsy, its existence is not suspected during life. The occurrence of hæmorrhage is sometimes marked by vomiting or convulsions, and usually there is loss of consciousness. It may be a question whether the convulsions are the cause or the result of the hæmorrhage. In most cases they seem to be the result. They are usually general and repeated. If the hæmorrhage occurs slowly, there may be stupor without convulsions until nearly the close of the disease. In the fatal cases the symptoms generally continue from two days to a week. There are dulness, stupor, and finally coma, death occuring in coma or convulsions. If the hæmorrhage is diffuse—and this is apt to be the case—there is rigidity of all the extremities; if it is of one side only, the rigidity affects only one arm and leg. The pupils are more frequently contracted, but may be dilated or unequal. There is diplegia, hemiplegia, or monoplegia, according to the seat and extent of the hæmorrhage. The respiration is slow and irregular and may be of the Cheyne-Stokes variety. The pulse is slow, irregular, and sometimes intermittent. The temperature is at first normal, but rises slowly until death occurs, when it is from 100° to 103° F. Generally the cranial nerves are not affected, and opisthotonus is absent. The knee-jerk is often exaggerated. In cases which do not prove fatal—these being chiefly in older children—we have a similar onset, but after a few days consciousness is regained, and only hemiplegia or monoplegia remains. The course of the paralysis is that seen after meningeal hæmorrhage due to other causes. Wagner has reported a case in which recurring hæmorrhages took place at intervals of several months, the autopsy showing distinct evidences of both old and recent lesions.

Pachymeningitis, I believe, plays a much more important rôle in the production of meningeal hæmorrhages in children than has generally been accorded to it. From the frequency with which this lesion is found as a cause of sudden meningeal hæmorrhages which are fatal, it is not unlikely that many of the cases which recover with hemiplegia or monoplegia, may be due to the same cause.

The prognosis depends upon the age of the patient and the extent of the hæmorrhage. Extensive hæmorrhages are usually fatal in infancy, but small ones are seldom so, for they are rarely at the base. The prognosis of the paralysis in cases not terminating fatally, is the same as after meningeal hæmorrhage due to other causes, with perhaps an added liability to recurrent attacks.

Without large hæmorrhages, pachymeningitis interna can not be diagnosticated; and it is impossible to differentiate the hæmorrhagic cases from other varieties of meningeal hæmorrhage. It is important to make a diagnosis between pachymeningitis with hæmorrhage, and acute simple meningitis. In the former we have a sudden onset; stupor occurring early, usually on the first day, gradually diminishing in cases of recovery, or deepening into coma in fatal cases; localized or general paralysis, also occurring early; there is no fever in the beginning, and only moderate fever at the close. In acute meningitis we usually have a higher temperature, especially early in the disease; coma develops later, and rigidity of the extremities is less pronounced. In certain cases, however, where the hæmorrhage occurs in the course of some other disease, a differential diagnosis may be impossible.

Treatment.—The treatment of pachymeningitis hæmorrhagica is symptomatic. The indications are, to relieve cerebral congestion by applying ice to the head, to allay irritative symptoms by the use of bromides, and to keep the patient perfectly quiet.

ACUTE MENINGITIS.

Three distinct varieties of acute meningitis are met with in children.

1. Cerebro-spinal meningitis. This is the only variety of meningitis which prevails epidemically, but it also occurs sporadically. It is due to a specific cause, the *diplococcus intracellularis* of Weichselbaum, known also as the *meningococcus*. It may be regarded as a general infectious disease, but with its essential lesions in the brain and cord.

2. Simple acute meningitis, which may be due to a wide variety of micro-organisms. Although this is sometimes primary, it is usually a secondary disease.

3. Tuberculous meningitis.

CEREBRO-SPINAL MENINGITIS—EPIDEMIC MENINGITIS—CEREBRO-SPINAL FEVER.

Epidemics of cerebro-spinal meningitis are separated by quite long intervals and occur without any assignable cause. The following chart (Fig. 129) represents the prevalence of the disease in New York city during the last fifty years. This shows that very little was seen of cerebro-spinal meningitis until the epidemic of 1872. After this time a certain number of deaths from this cause occurred each year, there being two or three times as many in some years as in others; but there

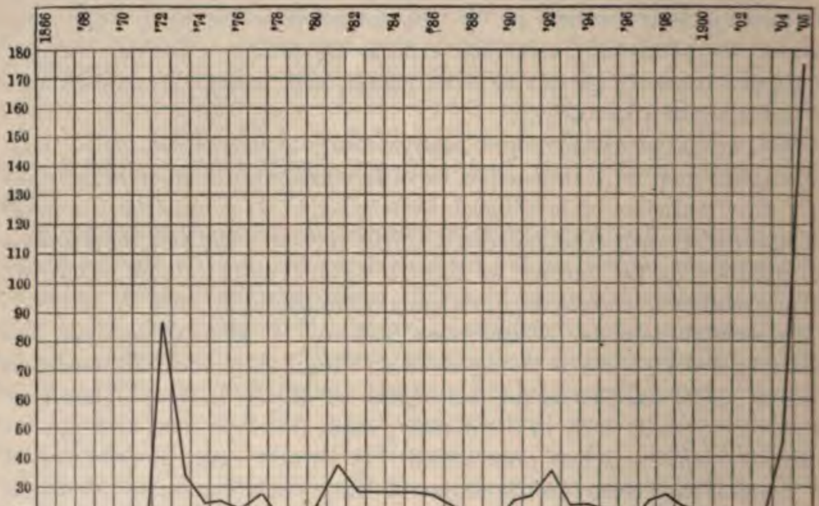

Fig. 129.—Chart showing deaths from cerebro-spinal meningitis in New York city, for fifty years, per 100,000 of population.

was no extensive epidemic until that of 1904–5. What has been said of New York is true of almost every large city. In remote country towns, epidemics are occasionally witnessed, and after prevailing a few months the disease disappears as mysteriously as it came. Epidemics are usually seen in the winter and early spring, lasting for several months, generally reaching their height in March or April and slowly subsiding as warm weather approaches.

With reference to the cause of epidemics very little has been settled. When the disease prevails in cities it usually occurs in crowded tenements, being relatively infrequent in private houses. Many cases may occur in certain districts, while in others not very far removed there may be very few. These facts suggest a connection with unsanitary conditions, but nothing that is positive has been demonstrated.

Cerebro-spinal meningitis is not contagious. Whether the disease is in any way communicable is not yet established. The fact that in a considerable number of cases (about 15 per cent according to the observations of the New York Health Department) an organism closely resembling the meningococcus, if not identical with it, has been found in the noses of children and adults exposed to the disease, affords some grounds for believing the disease to be communicable; probably very much as lobar pneumonia sometimes is. However, when we consider that in fully 70 per cent of the cases but one person in a household is affected, although no effort at isolation is made, it will be apparent that the danger of spreading the disease in this way is slight. I have never known the disease to originate in a hospital, although in New York patients with cerebro-spinal meningitis are regularly received into hospital wards with other children. Sporadic cases of meningitis occur after epidemics from no apparent cause and without any connection with one another. Children of all ages are about equally susceptible to this disease. The youngest case I have seen was in a child of two and a half months.

Cerebro-spinal meningitis is due to a specific organism, the diplococcus intracellularis or meningococcus. This is present in the meningeal exudate, in the cerebro-spinal fluid obtained by lumbar puncture, and in some cases can be demonstrated in the blood. It is almost invariably found in pairs or tetrads within the leucocytes. It is decolourized when stained by Gram's method. The portal of entry is as yet not settled; but from the fact that early in the disease the organism has been so often obtained from the upper part of the nose, the inference has been drawn that infection of the brain takes place through this channel. Outside the body the organism is unknown.

Lesions.—In epidemic meningitis death may take place so early that the changes found at autopsy are slight. There may be only a serous exudation and intense hyperæmia, which is doubtless much less marked after death than during life. The cerebro-spinal fluid is turbid and much increased in amount. The microscope, however, may show, even in these early cases, an abundant exudation of leucocytes in the pia mater. After the third day the lesions are quite uniform. The convolutions appear somewhat flattened from pressure due to distention of the ventricles. The inner surface of the dura is usually normal or only congested. There may be thrombi in any of the cerebral sinuses, or in the meningeal veins of the convexity. There is an exudation of greenish-yellow fibrin, which is sometimes very abundant. It is generally widely distributed, but is most marked over the anterior half of the brain and at the base. In some cases it is limited to the base, but very rarely limited to the convexity. There is an increase in the quantity of cerebro-spinal fluid. The ventricles are moderately distended with serum or sero-pus, and their walls may be slightly softened. The brain substance of the cortex may be reddened or may appear normal. In the meninges of the cord, lesions similar to those of the brain are usually seen. The exudation is principally upon the posterior surface, and may extend throughout the entire length of the cord, or be limited to its upper or to its lower portion.

Microscopical examination shows the exudation to consist of fibrin and pus cells, which infiltrate the pia mater. The superficial layers of the cortex in the inflamed areas often show minute hæmorrhages and very marked cell-infiltration. Minute abscesses may be present. Very marked degenerative changes can usually be demonstrated in the nerve cells themselves. The cells of the neuroglia are also affected; they are swollen and increased in number; and there may be proliferation of the connective tissue about the blood vessels. Changes in the cord similar to those just described may be found, but these are less frequent and as a rule much less severe than those in the brain. Inflammatory products are sometimes present in the central canal of the cord and in the walls of the lateral ventricles of the brain. The inflammatory process frequently extends along the cranial nerves, especially the optic and auditory, and this may result in choroiditis or otitis; from the cord, it may extend along either the anterior or posterior nerve roots. Descending degeneration is found in the nerves both of the brain and cord.

In patients that die after the disease has lasted two or three months, the later results of these lesions may be seen. There is usually present a chronic meningo-encephalitis, sometimes diffuse, sometimes localized. The pia mater is cloudy, thickened, and frequently adherent to the brain. Here and there are seen small, yellow, opaque patches which are the result of fatty changes in the cells and fibrin of the exudate, with some proliferation of connective tissue. The lesions are usually most marked at the base, where the thickening of the meninges and the adhesions may lead to the development of a secondary hydrocephalus.

In cases which have lasted a much longer time the most marked changes are in the brain substance. There may be generalized meningeal adhesions,* with a diffuse cortical atrophy, but more frequently there are areas of sclerosis, especially over the frontal and temporo-sphenoidal lobes, with which there are almost always associated marked descending degenerative changes in the cord. Such lesions are, of course, permanent, and seriously interfere not only with the functions, but also with the growth and development of the brain.

The visceral lesions most frequently found in epidemic meningitis are pulmonary. There may be lobar or broncho-pneumonia, and in the exudation may be found the same organism as in the brain. Acute degeneration of the liver and kidneys is also frequent. The other viscera are seldom affected.

Symptoms.—The symptoms of cerebro-spinal meningitis do not differ essentially in the sporadic and epidemic cases, except that the most severe forms of the disease are seen in the latter. They may be divided into several quite distinct groups:

1. *Hyper-acute form.*—Cases of this kind are rarely seen except in an epidemic, and usually occur at its height. The onset is very abrupt, the course short and intense, and death may take place in from twelve to thirty-six hours. The following case illustrates this type: A little girl of ten years was well enough at 2 P.M. to carry a bundle of clothes a dozen city blocks. Returning home, she complained of intense headache, vomited frequently, and was so weak that she was obliged to go to bed. In a few hours she passed into deep coma, with very high fever, and died at 11 P.M.

The earliest symptoms are usually intense headache, repeated attacks of vomiting, and very high fever. There is great prostration and the nervous symptoms increase so rapidly that in a few hours the patient may become comatose and death occur in a short period. The temperature rises rapidly to 104° or 106° F. A few petechial spots may be discovered over the face, chest, or extremities. There is usually no rigidity, but rather general relaxation. The pulse is weak, in most cases rapid, but sometimes slow and irregular. The respiration is usually irregular both in frequency and depth.

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* This lesion and its effects are well illustrated by one of my own patients who died six months after an attack. She was a bright little girl of four and a half years, and had a typical attack of meningitis of moderate severity. Convalescence was slow, but at the end of two months recovery was perfect in everything but her mental condition. She remembered nothing which she had previously learned in the kindergarten, where she had been an exceptionally bright pupil. Her mind was a blank. She was dull, listless, and her face had a vacant, idiotic expression. The special senses seemed unaffected, and speech was retained. She died during an attack of convulsions. At the autopsy the pia was everywhere thickened and adherent, while in the cortex were present the earlier changes of a general encephalitis.

The symptoms appear to be due to two factors: first, the intensity of the infection; second, the rapid accumulation of cerebro-spinal fluid, causing coma with cardiac and respiratory paralysis. Usually both these factors are present, but I believe that the second one is the more important. In support of this view is the striking infrequency of cases of this type in infants with an open fontanel. Should the patient survive the violence of the onset, a period of reaction occurs, and after a day or two the disease follows the regular course.

2. *Usual form.*—In this also the onset is generally abrupt, but not so violent as in the cases just described. It may be marked by intense headache, vomiting, convulsions, delirium, chills, and fever with general hyperæsthesia and rigidity. The initial temperature is from 101° to 104° F. Opisthotonus, with severe pains in the back of the neck and along the spine, and general muscular rigidity are usually present. There is often active delirium, but rarely stupor or coma. The pulse is generally rapid, 120 to 150, and sometimes irregular. The respiration is often slightly irregular, and it may be rapid or slow. The eruption is not so frequently seen as in the very acute cases.

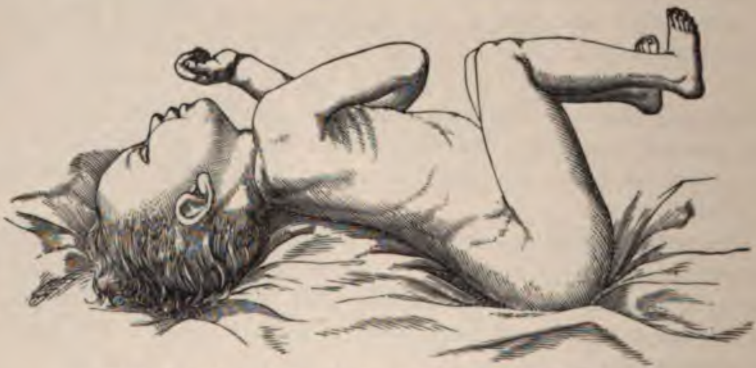

FIG. 130.—Posture in cerebro-spinal meningitis. (Smith.)

As the disease progresses, the nervous symptoms often change but little from day to day for two or three weeks. They are mainly of the irritative type—moderate delirium, extreme hyperæsthesia, tremor and muscular rigidity. The posture is quite characteristic (Fig. 130). Owing to the opisthotonus the child cannot lie upon the back, but rests upon the side, with arched spine and neck, and general flexion of the extremities. There is a rather rapid loss in weight, steadily increasing prostration, and a weak, rapid pulse. The bowels are usually constipated. From time to time attacks of vomiting occur. In most cases there is considerable difficulty in feeding. The duration of this form of the disease is from three to six weeks. The course is often marked by periods of remission and exacerbation. If recovery is to take place, the temperature gradually falls to normal and often at times it is subnormal. The mind becomes clear, and one by one the nervous symptoms disappear, the muscular rigidity being usually the last to go. Convalescence is always protracted.

In cases ending fatally, the patient usually passes into a deep stupor or coma, with extreme prostration, a slow, weak, irregular pulse, shallow respiration of the Cheyne-Stokes variety, sunken abdomen, general relaxation, and death occurs from exhaustion or from broncho-pneumonia.

Occasionally the attack is much prolonged, the fever and all the active symptoms continuing from eight to twelve weeks. Emaciation sometimes becomes extreme, and with a few nervous symptoms may continue long after the fever ceases. In infants, death is often due to marasmus. While a fatal outcome is more frequent in these prolonged cases, not a few recover completely, even where symptoms have lasted for eight or ten weeks.

3. *Mild form.*—Especially toward the end of an epidemic, and sometimes occurring sporadically, there are seen cases which in their onset and for the first two or three days resemble those just described; but instead of running the usual course, the fever and the nervous symptoms subside rapidly and convalescence is established early.

4. *Chronic form.*—Owing sometimes to the extent, sometimes to the position of the lesions, the disease does not subside at the usual time, but nervous symptoms continue after the temperature and most of the other constitutional symptoms have passed away. These cases are chiefly of the basilar type, and often lead to the development of chronic basilar meningitis with secondary hydrocephalus. They are more fully considered in a later chapter.

Onset.—One of the most striking features of this disease is the abruptness with which it develops. Occasionally there are indefinite symptoms for a day or two before active symptoms begin; but in the great majority not only the day, but the hour of the onset is definitely marked. The most frequent initial symptoms are the simultaneous occurrence of severe headache and vomiting, followed by high fever and marked prostration. The vomiting is usually repeated, projectile, and has no relation to meals. Convulsions occurred in the beginning of 30 per cent of my cases. Occasionally a decided chill is seen. After twenty-four hours acute general pains and hyperæsthesia are usually present, together with rigidity of the muscles of the neck and extremities, giving rise to opisthotonus and muscular contractions.

Skin.—Eruptions upon the skin vary much in frequency in different epidemics. The most characteristic one is the appearance of small punctate hæmorrhages, resembling flea bites; they are not numerous, but may be found on almost any part of the body, most frequently upon the extremities, the upper part of the chest and neck. In my experience they have been present in about 14 per cent of the cases. From this symptom the name "spotted fever" has arisen. This petechial eruption belongs to the early stage of the disease, fades quickly, and is rarely visible after the third or fourth day. In some cases a general erythema is present; in others, an eruption closely resembling measles. Herpes upon the lips and face is common in older children, but is rare in infants. Bedsores have been seen in about one-third of my cases. They are found over pressure points—the trochanter, the malleoli, and the side of the head; in several instances the ear has been the part affected.

Nervous system.—Headache is a frequent initial symptom and is usually severe; it is more often frontal than elsewhere, and may be associated with vertigo. There are acute pains in the back of the neck, along the spine, and marked general hyperæsthesia, which is often so intense that any movement of the body causes agonizing cries. This is one of the most striking symptoms of the disease, and may continue throughout the acute stage. The mental state varies much in different cases. Delirium is frequent in the early stage of the severe form; it is usually wild and active. After delirium a stage of dúlness or apathy ensues, giving place to great irritability when the patient is disturbed. Convulsions are sometimes seen early, but are seldom repeated in the course of the disease or toward its close. There is rarely continuous deep coma except toward the end of fatal cases. In some cases with high temperature and quite severe symptoms, after the subsidence of a short early stage of excitement or delirium, the mind remains perfectly clear throughout the attack. Under these circumstances an erroneous diagnosis is often made, particularly if the physician has not observed the case from the beginning.

Tonic spasm of the various muscular groups is seldom absent, and, like the hyperæsthesia, is persistent. The rigidity and contraction of the muscles of the neck and back produce cervical or general opisthotonus; cervical opisthotonus is most marked with lesions chiefly at the base, but may be wanting in the rare cases when the lesion is almost entirely at the convexity. Tonic spasm of the extremities usually causes general flexion of the thighs, legs, and arms. Late in the disease this may be replaced by complete extension of the lower extremities with dropping of the feet. The tonic muscular spasm gives rise to Kernig's sign, viz., inability to extend the leg when the thigh is flexed upon the body. In young children one should not place too much dependence upon this sign. While rarely, if ever, wanting in cerebro-spinal meningitis, it is often present in other conditions. Muscular rigidity is one of the most common symptoms and one of the last to disappear. Almost the only times when it is absent is in the early stage of the hyper-acute cases, and very late in fatal cases, when there may be general relaxation. Other nervous symptoms frequently present are ankle clonus, muscular tremor, especially of the hands, and paralysis, which may be facial, monoplegic, or hemiplegic. Early in the disease the knee-jerks are usually increased; in the later stages they are often lost.

Eye and ear.—The pupils in the early stage are generally contracted; toward the close they are usually widely dilated. Ocular paralyses are not so frequent nor so marked as in tuberculous meningitis. The same is true of the changes in the optic disc, although these vary much in different epidemics. There may be congestion of the fundus, retinitis, or optic neuritis. In some epidemics such changes have been observed in fully half the cases. In that of 1904–5, in my own hospital cases, they were rarely seen, and then were but slightly marked. Conjunctivitis is most frequently present and may be severe. There may be choroiditis and sometimes complete destruction of the eye, but usually this is unilateral. In most epidemics the ears are more frequently affected than the eyes. Early deafness may be due to a lesion of the auditory nerve, is generally bilateral, and often permanent. Acute otitis media occurs as a complication, and the meningococcus is occasionally found in the discharge. This was true of three of my hospital cases. Permanent deafness is sometimes due to changes in the brain itself.

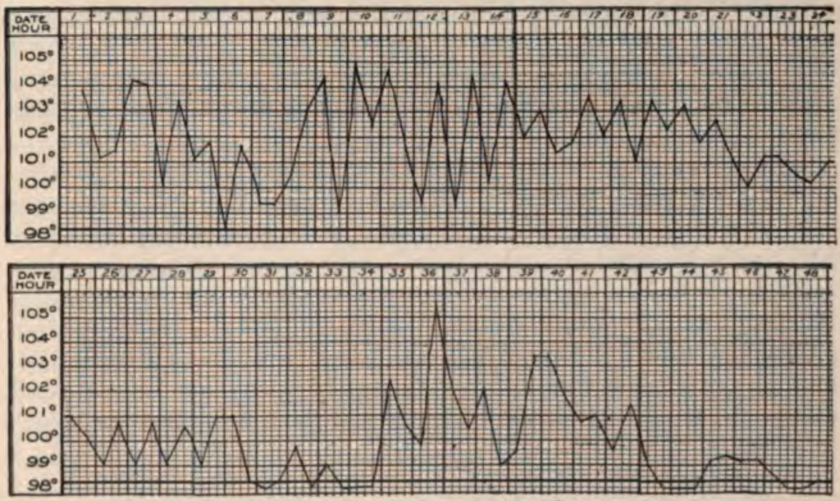

FIG. 131.—Cerebro-spinal meningitis. Recovery.

Fairly typical chart of prolonged case, showing remissions and exacerbations. Private patient, three and a half years old; unconscious, blind, and deaf for two and a half months; practically complete recovery.

Fever.—This disease is usually attended by high fever, but the curve is apt to be an irregular one and shows wide variations. The temperature is nearly always high at the onset; in the hyper-acute cases it may reach 106° F. or higher. The usual range during the disease is from 100° to 105° F. (Fig. 131). Sometimes it is steadily high; not infrequently a few days after a sharp acute onset it falls nearly or quite to normal and remains there for several days. Cases seen in this afebrile period are most difficult of diagnosis. This stage may be followed by another sharp rise, and afterward continuous fever. Periods of remission and exacerbation in the temperature are seen in a large proportion of the prolonged cases. Often it becomes subnormal. The temperature may bear no relation to the severity of the other symptoms. The duration of the febrile period is usually from three to six weeks.

Respiration is disturbed very early in the disease, when it is often irregular and may be slow or rapid. Throughout the greater part of the attack it may be nearly normal. Occasionally it is of the typical Cheyne-Stokes variety.

Pulse.—Through the greater part of the disease the pulse is rapid. In the early stage it is often weak, and sometimes irregular. The average frequency in young children is from 130 to 150. A slow, irregular pulse is occasionally seen late in the disease in patients who are in deep coma.

Blood.—A leucocytosis is present in nearly all cases. The count has varied in my experience between 7,000 and 64,000. The average of fifty-six observations was as follows: during the first week, 19,000; second week, 17,000; third week, 30,000; fourth week, 20,000; fifth week, 16,000. The increase is chiefly in the polynuclear cells. Blood cultures made early in the disease have in a few instances shown the presence of the characteristic organism.

Digestive system.—Vomiting is one of the most frequent symptoms of onset but rarely persists throughout the attack. Late in the disease it may be most troublesome. As a rule constipation is present. The tongue is coated, dry, glazed, sometimes covered with sordes. In a small proportion of cases jaundice has been observed. On account of the loss of appetite, great irritability, delirium, and stupor, the greatest difficulty is often experienced in feeding these patients. In young children gavage is much more satisfactory than rectal feeding. Early in the disease the abdomen is natural. In the late stage it is often very much retracted.

General nutrition.—This is impaired in nearly all cases. There is a progressive wasting, greater than would be explained by the disturbance of digestion. In the protracted cases it may be extreme. Infants and young children often die of inanition or marasmus long after the active symptoms of the disease have subsided.

Other symptoms of importance are the tense, bulging fontanel, in infants rarely absent early in the attack, but often wanting in the late wasting stage; incontinence of urine and fæces, and retention of urine, very frequent and often overlooked; occasionally swelling of some one of the large joints is seen.

Course, Duration, and Termination.—Excluding the hyper-acute cases in which death occurs very early, the usual duration of active symptoms is from three to six weeks. Of the cases which have come under my personal observation, more than half have lasted over four weeks, while active febrile symptoms of from six to eight weeks have not been uncommon. A very considerable proportion of these protracted cases terminate favourably. I have seen one child recover completely after 84 days of fever, and another after 102 days. In infants, the duration is shorter than in older children, as their resistance is sooner exhausted. The progress of the disease is an irregular one; most of the prolonged cases are marked by periods of exacerbation with increase in the fever and nervous symptoms, followed by periods of remission, with such improvement that it is thought that the disease is surely at an end. Not until the temperature has been normal for several days, the mind become clear, and the hyperæsthesia and rigidity of the neck and extremities have entirely disappeared, can we consider convalescence as established. Recovery is slow, and it may be many months before the child is able to walk and talk as usual, and has regained its lost weight.

In cases ending fatally, death may come early from coma, convulsions, or heart failure. It may occur in the middle period from complications, most frequently pneumonia, or the terminal stage of the disease may be seen with extreme wasting, continuous stupor, general relaxation, sunken abdomen, shallow, irregular respiration, feeble pulse, and finally death from exhaustion.

Complications and Sequelæ.—Most of the complications have already been mentioned. The chief ones are pneumonia, otitis, conjunctivitis or choroiditis, and bedsores. Rarely, nephritis and arthritis are seen. Sequelæ are, unfortunately, very common. There may be perfect recovery so far as physical functions are concerned, but the child be left mentally deficient. This may be seen in all degrees. In some cases the defect is so slight as not to be evident for several months or even years; in others the mental faculties are entirely lost. There may also be various types of paralysis—strabismus, facial paralysis, monoplegia, hemiplegia or diplegia, and often contractures, which are sometimes temporary, but apt to be permanent. The acute attack may be followed by chronic meningitis with hydrocephalus. Of the special senses, hearing is most frequently affected, deafness being quite common, usually of both ears, and deaf-mutism is not an infrequent result in young children. Blindness is rare and is usually unilateral. As a late result epilepsy may develop.

Prognosis.—The mortality of cerebro-spinal meningitis varies much in different epidemics and in the same epidemic at different periods. It is usually greatest at the height, and lowest toward the end of the epidemic. The average is about 70 per cent. Of fifty consecutive cases treated in my hospital wards in one epidemic, the mortality was 86 per cent. All these patients were infants or very young children. Of twenty cases under one year, not one recovered. Of cases seen in private practice, chiefly in older children, the mortality was 50 per cent. In no disease is it more difficult to foretell the outcome than in this one when it affects older children. Some cases, apparently the most hopeless, recover, while others which do not appear to be especially severe ultimately prove fatal. One should never despair of a patient no matter how unpromising the outlook. The symptoms indicating a good prognosis are a clear mental state, absence of marked or continuous opisthotonus, a good pulse, and a good digestion. The temperature is no guide.

Diagnosis.—Lumbar puncture is by far the most important means of diagnosis we possess. By it we can not only differentiate meningitis from other diseases with nervous symptoms, but can determine the variety of meningitis. Furthermore, this is possible very early in the disease. With suitable precautions I believe it to be absolutely free from danger, so that it may be employed whenever there is any question of meningitis. Properly performed, it gives conclusive information in nearly every case. The procedure is simple, but the technique is important.* The quantity of fluid which may be removed at one time varies from a few drops to three ounces, the average being two or three drachms. Its character varies with the stage of the disease. During the first day or two it is usually a turbid serum; later it is purulent, often thick and containing flocculi of fibrin. The gross pus gradually diminishes, and after the fourth or fifth week may nearly or quite disappear, but may reappear with an exacerbation of the symptoms. Many leucocytes may be found in the sediment for a much longer time. The fluid in other diseases and in non-inflammatory brain conditions is a perfectly clear serum. The presence of many leucocytes always indicates meningitis, but the variety can be determined only by microscopical examination of the sediment after standing, or, better, after centrifuging. In cerebro-spinal meningitis there are found within the pus cells many diplococci; some are also free in the fluid. The number of organisms may be few or many, but their presence establishes the diagnosis, which is possible in no other way during life. Sometimes when not obtained in the smears the diplococci are found by culture.

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* Puncture may be made with an ordinary surgical exploring needle, but the special lumbar needle devised by Quincke is preferable. This is merely a fine trocar and cannula and is made somewhat stronger than an exploring needle, which sometimes breaks. The child is placed upon the right side with the thighs tightly flexed against the abdomen to separate the spines and laminæ of the vertebræ as much as possible. The point chosen for puncture is in the median line between the third and fourth lumbar vertebræ. This is on a level with the highest part of the iliac crest. The skin should be carefully cleansed and the needle boiled. The pain is no greater than from exploratory punctures elsewhere, and no anæsthetic is necessary unless the child is extremely nervous or sensitive. The introduction is not difficult and the canal is reached at the depth of about one inch. The trocar is now withdrawn and the fluid usually flows freely through the cannula, sometimes spurting forth some distance, owing to high pressure. A dry puncture may be due to the fact that the canal has not been entered; but more frequently it is because the exudate is too thick to flow through the small needle, or the needle has been plugged by fibrin. Raising the patient to a sitting posture usually causes a freer flow, as does also flexing the head upon the chest if opisthotonus is extreme.

The diagnostic value of lumbar puncture, when properly performed, is very great; not only are positive findings conclusive, but a negative puncture in the first two weeks almost certainly excludes meningitis. Observations upon thirty-nine of my hospital cases gave the following findings: of twenty-one punctures during the first week, all gave positive results, i. e., fluid containing the organisms; of thirty-two made in the second or third week, twenty-eight gave positive results, and in four no fluid was obtained, though former punctures had given positive results. Fluid which did not show the organisms either in smears or culture was found only once during the first five weeks of the disease. In one case, very prolonged but not especially severe, the organisms were still present as late as the ninetieth day of the attack.

The diagnosis of cerebro-spinal meningitis by symptoms alone presents peculiar difficulties at the beginning of the attack, most of which disappear when the disease is fully developed. The most valuable early symptoms for diagnosis are, a sudden onset with intense headache, vomiting, high temperature, prostration and a petechial eruption, early rigidity of the neck and extremities, great mental excitement, irritability or delirium. Later in the disease three symptoms are rarely wanting—persistent hyperæsthesia, muscular rigidity of the neck and extremities, and fever. Kernig's sign is frequently seen in other conditions and is not diagnostic. These spinal symptoms are more to be relied upon for diagnosis than the cerebral symptoms, which are subject to greater variation. The mind in some cases remains perfectly clear; in others there is delirium and excitement, but not often continuous, deep coma. One should not lay too much stress upon the presence or absence of any single symptom, but rather consider the whole clinical picture.

At its beginning, cerebro-spinal meningitis may be confounded with scarlet fever, pneumonia, acute indigestion or influenza; the first is distinguished by the eruption and sore throat; the second, by rapid respiration and physical signs; the third and fourth, by less intense nervous symptoms, and the course of the disease. From all these, cerebro-spinal meningitis is differentiated by lumbar puncture. It is often difficult to distinguish between cerebro-spinal and tuberculous meningitis. At certain stages the symptoms of the two may be almost identical.

The most distinctive features are the following:

CEREBRO-SPINAL MENINGITIS.	TUBERCULOUS MENINGITIS.
1. Infrequent except when epidemic.	1. Occurs at all times and seasons.
2. Affects the robust quite as often as the delicate.	2. Much more frequent in the delicate and in those giving signs of other tuberculous lesions in bones, joints, lungs, etc.
3. Previous history not significant.	3. Often a history of exposure to tuberculous infection.
4. Onset abrupt with definite symptoms.	4. Gradual with indefinite prodromal symptoms.
5. Temperature usually high and widely fluctuating; 100° F. to 105° F.	5. Generally low, 99°. to 101° F., unless complicated by tuberculosis elsewhere.
6. Pulse generally rapid until late; respiration often is not disturbed.	6. Pulse frequently slow, irregular, and intermittent through greater part of the illness; respiration usually disturbed; in most cases of Cheyne-Stokes variety.
7. Petechial rash may be seen early.	7. None present.
8. Fluid obtained by lumbar puncture always cloudy in early weeks; microscope shows meningococcus.	8. Never a cloudy fluid; often no organisms found.
9. Great mental irritability; irritative symptoms often present throughout the attack.	9. Only present early, followed by drowsiness merging into deep stupor.
10. Rigidity and hyperæsthesia marked and continuous.	10. Seen in early stage only, never very marked; relaxation after the onset.
11. Course prolonged; often lasts three to eight weeks; progress irregular.	11. Seldom more than three weeks after beginning of definite cerebral symptoms; progress then steadily from bad to worse.
12. Mortality about 70 per cent.	12. Practically always fatal.

Treatment.—The treatment of cerebro-spinal meningitis is at present very unsatisfactory, and it is doubtful whether the results are greatly modified by any special plan of treatment; they seem to depend rather upon the age of the patient and the severity of the attack, than upon the management. The course of the disease is so irregular that physicians have often been inclined to attribute great benefit to particular plans of treatment, which larger experience proved to be valueless. Of the various specific measures proposed, the only one to be seriously considered is lumbar puncture. Regarding its therapeutic value opinion is still much divided. From my own experience, I am inclined strongly to advocate its use as early as possible in the attack, especially in cases characterized by the rapid development of severe nervous symptoms. The withdrawal of one or two ounces of fluid at this time may not only relieve coma, but very greatly improve the pulse and respiration. I think it should be tried in every case. Too often, to be sure, the relief is only temporary, but I am convinced that some cases are saved by early lumbar puncture. Of its value later in the disease, one must speak more guardedly. At times lumbar puncture seems to be distinctly beneficial to the pulse, respiration, and nervous symptoms; at others it is without any effect. It surely does no harm and deserves further trial. An ice-cap should be applied to the head, and at times an ice-bag along the spine. The bowels should be kept freely open by calomel or saline cathartics.

Treatment otherwise is directed toward the symptoms of the disease. Drugs for the purpose of affecting the inflammatory process I believe to be absolutely useless. Of the symptoms which call for special treatment, the most prominent one is pain, which when severe requires morphine or codeine sometimes in quite large doses. It is often necessary to give it hypodermically. For other nervous symptoms—delirium, sleeplessness, etc.—the bromides and chloral, sulfonal, or trional may be given, or warm sponge or tub baths. Stimulants are required in most of the cases at some time in the course of the disease. They are indicated by a weak, rapid, and irregular pulse. Alcohol and digitalis or strophanthus should be used, but not strychnine.

One of the most important duties of the physician is to look after the nutrition of the patient. The difficulties in feeding are sometimes great, but they can often be overcome by the use of gavage (page 64), which may be advantageously employed as a routine practice in a very large number of the severe cases. One should be on the watch for bedsores, and endeavor to prevent them by cleanliness, frequently changing the patient's position, etc. The bladder also must not be forgotten, as retention of urine is not uncommon and may require the use of the catheter.

For the residual paralysis, massage, warm baths, and friction should be employed, but electricity only when all symptoms of central irritation have subsided. The prolonged use of iodide of potassium, especially in combination with mercury, seems to have some influence in promoting absorption of the inflammatory products in cases where there is a persistence of symptoms for two or three months.

SIMPLE ACUTE MENINGITIS.

This term may be used to include all the varieties of acute meningitis due to other causes than the diplococcus intracellularis and the tubercle bacillus. Although the cases in this group may differ widely in etiology, they are closely related clinically, and may therefore be advantageously considered together.

Etiology.—A larger number of cases are probably due to the pneumococcus than to any other single organism. From this cause we may have not only secondary meningitis following pneumonia, empyema, and other forms of pneumococcus infection, but also primary meningitis. A considerable number of this variety sometimes occur in a single season, and to them the term "epidemic" has been improperly applied. It is from such data that some writers have drawn the conclusion that epidemic meningitis may be due to this organism as well as to the diplococcus intracellularis. Such a group of cases is very different from a general epidemic of cerebro-spinal meningitis. It therefore seems best, with our present knowledge, to limit the term epidemic meningitis to the disease caused by the diplococcus intracellularis.

When meningitis is due to other causes than the pneumococcus, it is nearly always a secondary disease. It may be caused by the streptococcus, staphylococcus, gonococcus, influenza bacillus, typhoid bacillus, or the colon bacillus.

Meningitis from the streptococcus is seen in the newly born following umbilical infection, and in older children associated with otitis media or mastoiditis. It also occurs from traumatism, from general pyæmia, and with erysipelas of the scalp. Under many of the same conditions the staphylococcus may be the bacterial cause of meningitis. I have once seen meningitis in the newly born from the colon bacillus, originating probably after an umbilical infection. The pus from lumbar puncture during life contained this organism in pure culture. Meningitis due to the gonococcus, to the bacillus of typhoid fever, or of influenza, is very rare in children.

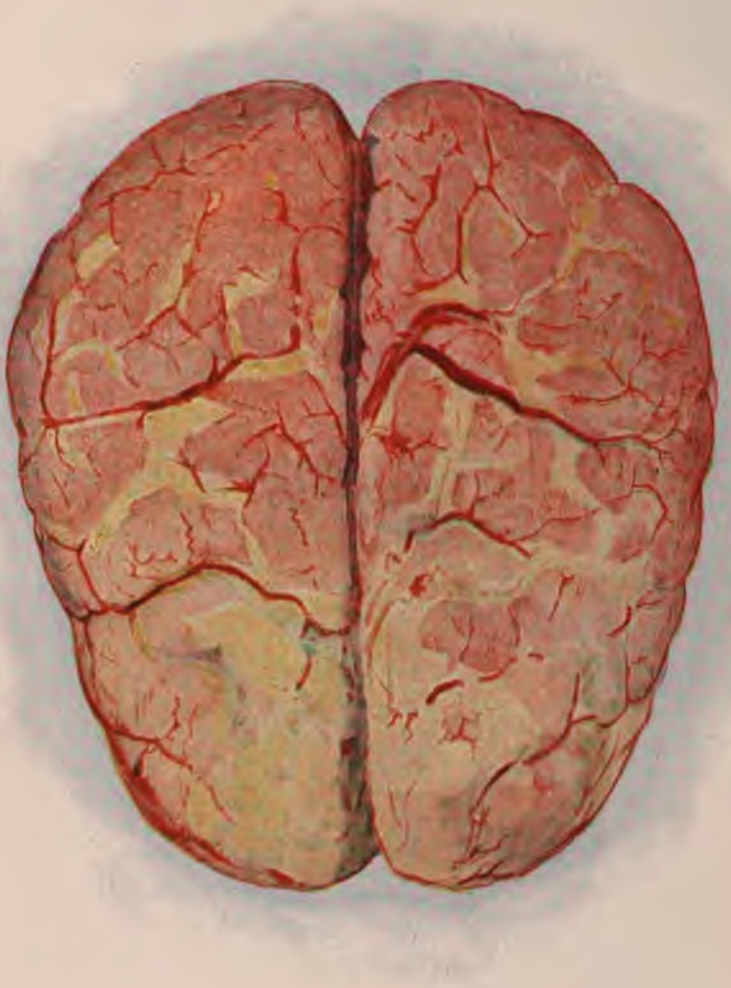
Lesions.—In a general way the anatomical changes resemble those described in cerebro-spinal meningitis, with the exception that the marked changes in the brain substance which are usually dependent upon the long course of that disease are wanting. As a rule, also, in simple acute meningitis the lesions are limited to the meninges of the brain. If the cord is involved, it is only to a slight degree. Almost the only cases in which cord involvement is seen are those in which the exciting cause is the pneumococcus.

Acute simple meningitis due to the pneumococcus is characterized by a more abundant exudation of fibrin and pus than is seen in any other variety of meningitis. It affects the convexity as well as the base, and is especially marked over the anterior lobes. Often the exudate almost conceals the convolutions. (See Plate XIV.) There is usually less distention of the ventricles than in cerebro-spinal meningitis. When due to other causes than the pneumococcus, the lesions are not distinctive, and do not differ greatly from the cerebral lesions of cerebro-spinal meningitis.

Symptoms.—The primary cases are nearly always of the pneumococcus variety. As in these the membranes of the cord are sometimes involved, the symptoms may be almost or quite identical with those of cerebro-spinal meningitis, the only possible method of differentiation being by lumbar puncture. The course, however, is usually shorter and the termination almost invariably in death.

Acute secondary meningitis presents quite a different clinical picture,

PLATE XIV.



ACUTE MENINGITIS, COMPLICATING PLEURO-PNEUMONIA.

Child twenty months old; on twenty-third day of a protracted attack of pneumonia, vomited six times, and the temperature, which had been nearly normal for four days, rose to 103° F. On the following day general convulsions, which were repeated frequently during the next few days; temperature, 101° to 104° F.; death in convulsions on twenty-eighth day.

Autopsy.—Pleuro-pneumonia of left side; lung resolving. Anterior portion of brain enveloped in lymph and pus, more marked at the convexity, but present also over the base.

and the symptoms are greatly modified by those of the original disease. Meningitis is often latent, and the lesions may be found at autopsy where no very marked cerebral symptoms have existed during life. This is particularly true when the pathological process is chiefly at the convexity.

The symptoms of acute secondary meningitis are essentially the same no matter what the bacterial cause. The involvement of the brain may be indicated by the abrupt occurrence of vomiting or convulsions, rapidly followed by stupor and coma, or there may be simply headache and a gradual increasing apathy or drowsiness. The later symptoms resemble the later stage of cerebro-spinal meningitis, except that the spinal symptoms—general hyperæsthesia, rigidity, and contractions—are wanting, while the cerebral symptoms may be more prominent. The most significant are the following: continuous deep stupor; dilated or unequal pupils which do not respond to light; strabismus, ptosis, or some other localised paralysis; in infants, a tense, bulging fontanel; a slow, irregular, or intermittent pulse, especially when associated with high temperature; irregular, shallow, sighing respiration, interrupted by long pauses; general relaxation or paralysis, and constipation. Often present, but of less diagnostic value, are opisthotonus, retracted abdomen, the *tâche cérébrale,* marked irritability, increased knee jerks, sharp cries, delirium, and convulsions.

As compared with the cerebro-spinal form, simple acute meningitis runs a much shorter course, rarely lasting a week. Its progress is steadily from bad to worse, periods of remission in the symptoms being infrequent. It almost invariably terminates fatally.

Diagnosis.—The toxic symptoms of many acute diseases, notably pneumonia, typhoid and scarlet fever, gastro-enteric intoxication, and ileo-colitis, may very closely simulate acute meningitis. Almost every single symptom of meningitis may be present, even though the brain is not involved; but rarely, if ever, is such a combination of symptoms seen as is present in meningitis. Without such a grouping of symptoms one should hesitate to make a diagnosis of meningitis when another acute disease is present, especially if that one be any form of diarrhœal disease. The mistake is more frequently made of diagnosticating meningitis where there is none than of overlooking it when present. Our only certain means of differential diagnosis is by lumbar puncture. This not only distinguishes meningitis from other diseases with nervous symptoms, but determines the form of meningitis. In most of the varieties a turbid fluid is present, which shows by smears the particular organisms causing the disease.

Treatment.—This is symptomatic purely, and should be carried out along the same lines as have been already laid down under cerebro-spinal meningitis.

TUBERCULOUS MENINGITIS.

Synonyms: Acute hydrocephalus; basilar meningitis; water on the brain.

Tuberculous meningitis is a tuberculous inflammation of the pia mater of the brain, sometimes involving also that of the cord. It is doubtful if it ever occurs as the only tuberculous lesion of the body. It is quite frequently seen, and is more uniformly fatal than any other disease of early life. In infancy it is usually associated with general or pulmonary tuberculosis; in older children with tuberculosis of the bones, joints, or lymph nodes. Of my own cases, twenty-five per cent of all deaths from tuberculosis in children were due to meningitis.

Lesions.—The lesion consists in the production of miliary tubercles, with which are frequently found tuberculous nodules of variable size, and in almost every case there are also the products of ordinary inflammation of the pia mater—fibrin and pus—together with an accumulation of fluid in the lateral ventricles of the brain. Frequently there are tubercles in the pia mater of the upper portion of the cord. The miliary tubercles appear as small gray or white granules, situated along the vessels of the pia mater. When few in number they are usually only at the base, especially along the Sylvian fissures and in the interpeduncular space. When numerous they are most abundant at the base, but are also seen scattered over the convexity in small groups. In about half of my autopsies they were limited to the base, and in no case were they seen exclusively at the convexity. Tubercles are often found in the choroid coat of the eye. The amount of fibrin and pus present is rarely great, and never equal to that seen in simple acute meningitis. It is often a matter of surprise at autopsy to find the lesions so few, after very marked symptoms. The inflammatory products are most abundant at the base. In addition to the patches of greenish-yellow fibrin, there are adhesions between the lobes of the brain and thickening of the pia. In cases which have lasted for several weeks, the pia mater in places is often very much thickened, owing to cell infiltration and the production of new connective tissue, and it is studded with miliary tubercles, sometimes with small yellow tuberculous nodules; frequently there is arteritis, which is sometimes obliterating.

In the most acute cases the brain substance immediately beneath the pia is intensely congested, slightly softened, and shows under the microscope a superficial encephalitis. The lateral ventricles are usually distended with clear serum, sometimes with serum containing flocculi of fibrin or pus; the amount present varies from one to four ounces in each ventricle, being always greater in the subacute cases. The walls of the ventricles may be softened. The distention of the ventricles leads to flattening of the convolutions from pressure against the skull, to bulging of the fontanel, and sometimes to separation of the sutures, if they are not completely ossified.

Tuberculous nodules varying in size from a small pea to a walnut are frequently seen associated with meningitis in older children, but not so often in infants. These nodules may be connected with the meninges, or they may be situated within the brain substance, usually in the cerebellum. The larger ones are classed as brain tumours. Inflammatory products are rarely found in the spinal canal.

Although it is not infrequent to see meningitis without symptoms of tuberculosis elsewhere, I have never failed at autopsy to find other tuberculous lesions in the body. In my own experience the following are those most often met with, given in the order of frequency:

(1) In infants, associated with general or pulmonary tuberculosis; (2) in children from three to twelve years of age, with tuberculosis of the vertebræ, hip, knee, or ankle; (3) at any age, with tuberculosis involving only the tracheal, bronchial, or mesenteric lymph nodes; (4) much less frequently with the pulmonary tuberculosis of older children. There seems now to be good reasons for believing that meningitis may follow tuberculous adenoids.

Etiology.—Tuberculous meningitis is produced only by the transportation of the tubercle bacilli to the brain. They may find their way by the blood-vessels or lymphatics.

The following table shows the age at which the disease is most frequently observed:

AGE.	Personal cases.	Oxley.	Total.
Under one year	14	3	17
One to two years	9	16	25
Two to five years	24	26	50
Five to nine years	15	18	33
Nine to sixteen years	5	0	5
Totals	67	63	130

In this series males were a little more frequently affected than females. In two or three instances traumatism was apparently an exciting cause. Tuberculous meningitis is occasionally seen in young children who have been previously healthy, whose family history is free from tuberculosis, and where no exposure can be traced. It is probable that in all such cases there has been latent tuberculosis somewhere in the body, and that the exposure was long antecedent to the symptoms. In the majority, however, this is not the case. There is usually a history of exposure to infection; or there have been previous evidences of tuberculosis in the lungs, bones, or lymph nodes.

Symptoms.—In forty-three of sixty-three cases the onset was gradual; but in a considerable number of those classed as sudden, careful inquiry elicited a history of previous indisposition. The most frequent early symptoms are: disinclination to play, or drowsiness, sometimes constant fretfulness or irritability. Often there is a distinct change in disposition. In a case recently under observation this was most striking; a little girl previously devoted to her mother, could not endure her presence in the room. There is loss of appetite, and usually constipation. Sleep is restless and disturbed; there may be grinding of the teeth. Older children often complain of headache. At all ages a suggestive symptom is frequent attacks of vomiting without apparent cause. In addition to these there may be a slight but continuous elevation of temperature. Indefinite symptoms may last for four or five days, or they may be spread over two or three weeks without perhaps being sufficiently severe to attract much notice. Finally, unmistakable evidence of brain disease develops, and then it is recollected that symptoms like the above had existed for some time. These early disturbances are often ascribed to dentition, to worms, or to indigestion; and sometimes they are regarded simply as the result of the constipation.

In the midst of such indefinite symptoms there may come an attack of convulsions, and, in the course of a few hours, deep stupor. The early symptoms of the active stage are indicative of cerebral irritation. There is headache, often located in the frontal region, and occasionally photophobia; sometimes there is sudden screaming out at night without waking. The skin is usually somewhat hyperæsthetic; the reflexes are apt to be exaggerated; the muscles of the neck may be rigid and the head is drawn back, or there may be rigidity of one or more of the extremities. The pupils are normal or contracted; there may be nystagmus. The child is fretful, wishes to be left alone, and cries if disturbed; but otherwise is apt to be unnaturally drowsy. Such symptoms may continue for a day or two, or even for a week. If prolonged, they are likely to alternate with periods of more marked apathy and dulness. During this stage there is occasional vomiting, and the bowels are obstinately constipated. The pulse is usually somewhat accelerated, but may be slow and occasionally is irregular. The respiration is of normal frequency, but a careful observation during sleep or perfect quiet will often show a slight irregularity which is very significant. This becomes more marked as the disease progresses. The temperature is invariably elevated, but never very much so, generally being from 99° F. to 101° F. When a high temperature is seen, it is usually due to tuberculosis elsewhere than in the brain.

During the intermediate or second stage, the irritative symptoms subside, and stupor becomes deeper and more continuous. If undisturbed, the child may sleep a great part of the time, but can be roused, and then appears quite rational. Later the stupor becomes so profound that the child can not be roused at all; or, again, this condition may alternate with periods of complete lucidity. Active delirium is rare. The pupils respond slowly to light or not at all; they may be unequal; occasionally there is seen strabismus, ptosis, or paralysis of the face. More often there is hemiplegia, or paralysis of one arm or leg. Such paralyses are often transient, disappearing after a day or two. Automatic movements of the extremities, particularly of the arms, are frequent. Muscular twitchings may be noticed. Opisthotonus is marked and well-nigh constant. In infants the fontanel is tense and bulging; the abdomen is retracted, giving the typical "boat-belly." On drawing the finger-nail along the skin of the abdomen, there appears, after a few seconds, a distinct red streak one or two inches wide, which remains for three or four minutes. This is the *tâche cérébrale,* and while not pathognomonic, it is almost always present. Other vaso-motor disturbances may be seen. The reflexes are variable; in the early part of the disease they are usually increased, later they are diminished or abolished. The pulse now becomes slow and irregular, often intermittent. The respiration assumes the characteristic type, which consists in the movements becoming deeper and deeper until there is a long sigh, then a complete arrest of respiration for several seconds, after which the movements begin again, at first shallow, but gradually increasing in depth until the sigh is repeated. The accompanying tracing illustrates the type (Fig. 132). An examination with the ophthalmoscope usually shows the presence of choked discs and possibly choroid tubercles.

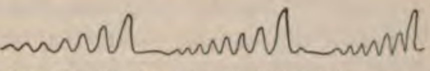


FIG. 132.—Tracing of respiration in tuberculous meningitis.

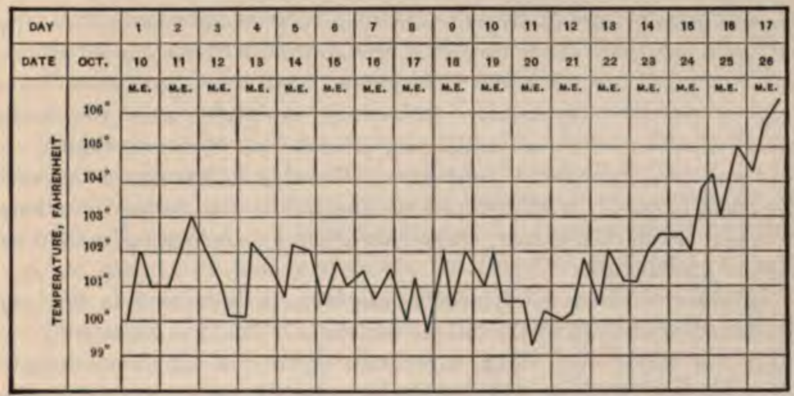


FIG. 133.—Fairly typical temperature curve in tuberculous meningitis; boy, twenty months old; death on seventeenth day.

The duration of this stage is from three to ten days. The progress is irregular, and subject to great variations, especially as regards the mental symptoms. Sometimes a child will be seen in quite deep stupor. and on the following day will be sitting up in bed playing with his toys.

In the third stage there is complete coma. The child can not be roused at all. The pupils are widely dilated, and do not respond to light. There is general muscular relaxation. There may be retention of the urine. Deglutition is difficult, sometimes almost impossible. The boat-belly and opisthotonus are still marked. The respiration is more rapid, but still irregular. There are sordes on the lips and teeth, emaciation, and anæmia. Toward the end the temperature rises rapidly to 104° F., sometimes to 106° or 107° F. (Fig. 133). The pulse becomes very rapid and feeble, often 160 to 180 a minute. Death usually takes place from exhaustion in deep coma; or convulsions develop and continue from twelve to twenty-four hours until death. The duration of the stage of coma is from two days to a week. Often the patient will live for four or five days in a condition of prostration so extreme that death is hourly expected. A rapidly rising temperature or the occurrence of convulsions indicates approaching death. Of fifty-seven cases, fifty died in coma, seven in convulsions.

The entire duration of the disease from the beginning of definite symptoms, in sixty-five of my own cases, was as follows:

One week, or less	17
One to two weeks	15
Two to three weeks	17
Three to four weeks	14
Five weeks	2
	65

Variations in the course of the disease.—There are few diseases which present a greater variety of symptoms than tuberculous meningitis. Typical cases like those above described are seen most frequently in children over two years old, in whom the cerebral symptoms predominate over those of general tuberculosis. In infancy, especially when the disease follows acute tuberculous pneumonia, the duration of the cerebral symptoms may be only three or four days. The stages then are not marked. The onset is usually with convulsions, and in less than twenty-four hours there may be marked stupor, and all the symptoms belonging to the third stage of the disease.

In older children the symptoms are from pressure due to the great accumulation of fluid in the lateral ventricles. There is persistent drowsiness, but rarely deep coma, sometimes rigidity of all the extremities, and sometimes paralysis. Opisthotonus is nearly always marked in these cases.

Diagnosis.—There are no diagnostic symptoms in the first stage. If the patient has previously suffered from local or general tuberculosis, and symptoms develop which are enumerated as prodromal, meningitis may be suspected. If the child has previously given no evidence of tuberculosis, an early diagnosis is impossible. The indefinite symptoms that belong to this stage of the disease are frequent in young children suffering from chronic indigestion associated with constipation. Such is the usual explanation of the indisposition rather than incipient meningitis. Some cases of cyclic vomiting may present many of the symptoms of meningitis.

The most frequent symptoms of tuberculous meningitis enumerated in the order of their occurrence in fifty-eight cases were as follows: obstinate constipation, persistent drowsiness, irregular respiration, vomiting without apparent cause, irregular pulse, convulsions, opisthotonus, and fever which was usually slight. Equally important for diagnosis, and especially significant when associated with the above, are strabismus, facial paralysis, and loss of the pupillary reflexes.

The fluid drawn by lumbar puncture is usually perfectly clear. In some cases, after standing, a very few leucocytes may be found in the deposit. Tubercle bacilli can be demonstrated in but a small proportion of the cases, even after centrifuging. A few bacilli are, however, present, as may be shown by inoculation of a guinea-pig. This affords a positive, though late, means of diagnosis. The symptoms which distinguish tuberculous from cerebro-spinal meningitis have already been considered in the discussion of the latter disease.

The cerebral symptoms of ileo-colitis and other diarrhœal diseases sometimes closely resemble those of tuberculous meningitis; but whenever in a young child there is some other disease present which may furnish an explanation for the cerebral symptoms, the diagnosis of meningitis should be made with great caution. The development of meningitis in the course of an ordinary attack of pneumonia may simulate very closely pulmonary tuberculosis with tuberculous meningitis. A diagnosis may be impossible during life. In doubtful cases the probabilities are greatly in favour of tuberculosis, since it is so much more common.

Prognosis.—Although there have been recorded a few isolated instances of recovery after the tubercle bacilli have been found in the fluid obtained by lumbar puncture, such an outcome is so exceedingly rare as not to be expected. I have never seen it. Cerebro-spinal meningitis may at times very closely simulate the tuberculous variety, and it is probable that most of the cases of alleged recovery were not tuberculous.

Treatment.—From what has been said regarding prognosis, it follows that if the diagnosis is correct the case is practically hopeless, no matter what treatment is employed; but as a positive diagnosis is not always possible, all cases should be treated like those of simple acute meningitis.

CHRONIC BASILAR MENINGITIS IN INFANTS.

It was first pointed out in 1898 by Still (London) that this disease is usually due to the diplococcus intracellularis; in other words, that it is a chronic form of cerebro-spinal meningitis. Chronic basilar meningitis is most frequently seen after epidemics of cerebro-spinal meningitis, but it is occasionally met with at other times as a sequel of a sporadic case. It occurs after an acute attack, when the basilar lesion persists and becomes chronic. As acute cerebro-spinal meningitis in infants is invariably fatal if the attack is severe, it follows that the chronic form is seen only after the mild attacks. It is chiefly for this reason that the early symptoms often are not recognized as types of cerebro-spinal meningitis. The patient frequently does not come under observation until all acute symptoms have passed away, the persistent opisthotonus being the chief feature of the case.

There is also seen in children, though very rarely, a chronic basilar meningitis of syphilitic origin. At least two such cases have come under my observation in the Babies' Hospital. One was cured by anti-syphilitic treatment, and the other diagnosis was confirmed by autopsy.

Lesions.—This process is usually limited to the base of the brain. The pia mater is thickened about the interpeduncular space, also over the medulla, pons, and cerebellum. These different parts may be adherent to each other, or to the inner surface of the dura. The cranial nerves may be compressed. The openings in the fourth ventricle are usually obliterated, and there results a distention of the lateral ventricles with clear serum, sometimes in sufficient amount to be regarded as hydrocephalus. Rarely, pus may be found in the ventricles.

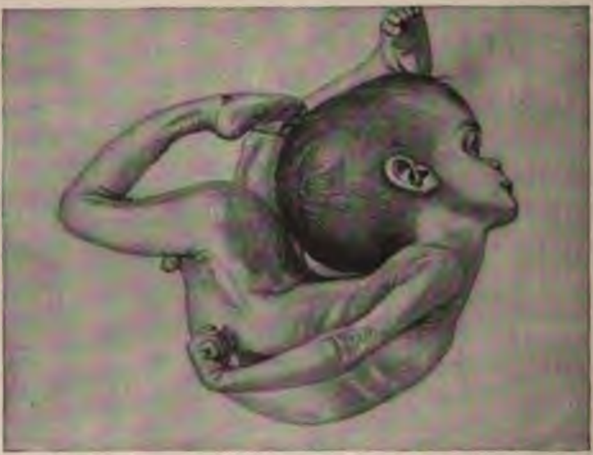


FIG. 134.—Chronic basilar meningitis—extreme deformity.

Ill for five months; followed cerebro-spinal meningitis; posture shown in the picture maintained for the last six weeks; death at ten months. Autopsy showed typical lesions.

Symptoms.—The onset is usually gradual, although in most cases there can be obtained a fairly distinct history of an early active period. The most prominent symptoms are cervical opisthotonus, moderate hydrocephalus, and usually general muscular rigidity. The opisthotonus is often extreme (Fig. 134) and is greater than is seen in any other disease.

If placed upon its back the body of the child often touches the table only at the occiput and the sacrum (Fig. 135). The head is usually somewhat enlarged, but never to the degree seen in primary hydrocephalus; the fontanel bulges, and the sutures are separated. These symptoms are due to an accumulation of fluid in the lateral ventricles; they are never so marked as in primary hydrocephalus. The rigidity of the extremities is very great and in most cases constant; the legs and feet are usually extended, while the forearms are flexed and the hands clenched. All the reflexes are greatly exaggerated. There is rarely coma, but mental dulness alternating with periods of great irritability in which general convulsions may occur. Vision may be impaired or wanting entirely. The fact that in most cases optic neuritis is absent is of some value in differentiating this disease from tumour. Nystagmus is often present and attacks of vomiting occur without evident cause. There is no fever except for a few days at a time during acute exacerbations. The usual duration of the disease is from two to five months; death may occur from convulsions, from some intercurrent disease, such as pneumonia, but most frequently from marasmus. The prognosis is very bad except when the cause is syphilis, when recovery may take place.

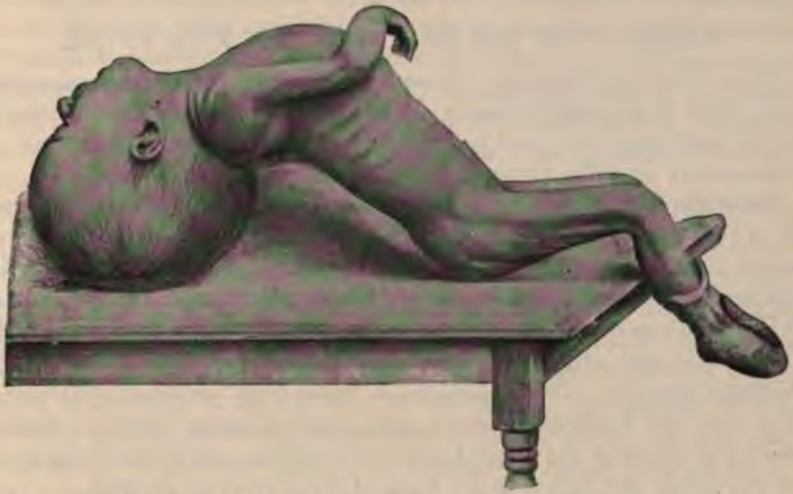


FIG. 135.—Chronic basilar meningitis; a patient in the Babies' Hospital (diagnosis confirmed by autopsy).

Diagnosis.—The disease is to be distinguished from tuberculous meningitis, and from the opisthotonus of reflex origin which is occasionally seen in infants suffering from marasmus. It differs from tuberculous meningitis in its more protracted course, in the absence of fever, paralysis, and also in the greater prominence of the opisthotonos and hydrocephalus. The opisthotonus which is seen in cases of marasmus is never so extreme or so continuous, and is not accompanied by any enlargement of the head, or by other cerebral symptoms.

Treatment.—If there is any reason to suspect syphilis, iodide of potassium should be administered. At least fifteen grains daily should be given for several weeks to an infant six months old, and still larger doses if the stomach will tolerate it. Lumbar puncture is useful for diagnosis only. The establishment of auto-drainage of the ventricles, as practiced in primary hydrocephalus, has recently been advocated for this condition, and tried with some measure of success.

THROMBOSIS OF THE SINUSES OF THE DURA MATER.

This is not very frequent. It may depend upon certain general conditions, when it is usually classed as cachectic or marantic thrombosis; it may be associated with local pathological processes, when it is known as inflammatory or septic thrombosis.

Cachectic Thrombosis.—This is seen in infants and young children, but is very rare after the age of five years. It occurs in the course of various diseases, the most frequent being pneumonia, pertussis, diphtheria, nephritis, tuberculosis, and the acute intestinal diseases. In connection with the last-mentioned group, altogether too much has been made of it, as it is really rare, and in only a very few cases does it explain the cerebral symptoms present. This statement is made from personal observations upon over two hundred autopsies upon cases of acute intestinal disease. The actual cause of the thrombosis is the altered condition of the blood and the feeble circulation, as the walls of the sinuses are normal.

The most frequent seat of cachectic thrombosis is the superior longitudinal sinus. At autopsy one must be careful not to confound the soft, partly-decolorized, non-adherent thrombi of post-mortem origin, with those of ante-mortem formation. The latter are firm, and when of long standing may be very hard and even show a laminated structure. They usually fill the sinus completely, and are adherent. The thrombus extends from the sinuses to the veins emptying into it, which stand out like dark worms upon the surface of the brain. The brain itself may be deeply congested, or it may be covered with a diffuse hæmorrhage, but more frequently the brain and the membranes are simply œdematous.

The *symptoms* of cachectic thrombosis are few and uncertain, and in a large number of cases the disease is latent. Very rarely is a positive diagnosis possible during life. When the thrombosis occurs just before death, its symptoms are so mingled with those of the original disease that they can not be separated. In some cases there may be localized or general convulsions, or paralysis, loss of consciousness, and strabismus.

The *prognosis* is bad, cases generally proving fatal in the course of a few days. The diagnosis is so uncertain and obscure that the *treatment* must be symptomatic, and directed toward the general rather than the local condition.

Inflammatory Thrombosis—Septic Thrombosis—Sinus-Phlebitis.—This condition is most frequent in children in connection with acute meningitis. It may exist either with the simple or the tuberculous variety. It also follows otitis—especially old and neglected cases—usually with necrosis of the petrous bone, but sometimes without it. It is much less frequently associated with disease of the ear in children than in adults. It may arise from traumatism, necrosis of the cranial bones, or from septic processes involving any of the cavities or any of the structures adjacent to the brain, such as the scalp, orbit, nasal fossa, mouth, or pharynx. Infection from the mouth or pharynx is most frequent in children in connection with scarlet fever or diphtheria; while usually secondary to otitis it may occur without it, the infection being carried by the blood-vessels. Infection from the nose may have its origin in ulceration from syphilis or tuberculosis. In the orbit, the source may be malignant disease.

The seat of the thrombosis will depend upon the original disease. If this affects the cranial bones or the scalp, it will be the longitudinal sinus; if the ear, the lateral sinus; if the base of the skull, the orbit, the mouth, the jaw, or the nose is affected, it will be the cavernous sinus. When thrombosis occurs with meningitis the lesions are much the same as in the cachectic form, with the exception that there are sometimes slight changes in the walls of the sinuses. If the patient has suffered from a local septic process, there may be puriform softening of the clot, and general pyæmia, with the development of secondary abscesses in the brain, in the lungs, and in other organs. With such cases there may be associated a general or localized meningitis.

Symptoms.—The symptoms of septic thrombosis are more decided than those of the cachectic form. When occurring in the course of meningitis, it usually adds no new symptoms to those of the original disease. In the pyæmic form the symptoms are more characteristic, particularly when associated with otitis. There are recurring chills with very high and widely-fluctuating temperature. There is headache, and often localized tenderness of the scalp; the other symptoms which are present are usually the same as those of meningitis. If metastasis occurs, there may be evidences of abscesses of the brain or in other organs, and sometimes there are signs of suppuration in the jugular vein.

The local symptoms of the thrombosis differ somewhat according to the sinus affected: if its seat is the superior longitudinal sinus, there may be cyanosis of the face, dilatation of the temporal and frontal veins, and sometimes epistaxis; if the lateral sinus is involved, the process may extend to the jugular vein, which may be felt in the neck as a hard cord, and there may be dilatation of the veins of the mastoid region, and even localized œdema; when the cavernous sinus is affected, there may be protrusion of the eyeball of the affected side, œdema of the lid, and with the ophthalmoscope the retinal veins appear enlarged and tortuous, sometimes being the seat of thrombosis. The process may affect either one or both sides. The course of septic thrombosis is rather irregular, varying from a few days to three weeks. In fatal cases death takes place from meningitis, cerebral abscess, or pyæmia. The prognosis is very grave, unless the disease is so situated that it is accessible to surgical operation.

Treatment.—The only successful treatment is surgical. Operation is easiest in thrombosis of the lateral sinus, being much more difficult if involving the superior longitudinal sinus. So many cases are now on record of successful operation upon septic thrombosis of the lateral sinus, that it should always be urged when the diagnosis is clear. Recurring chills and high, fluctuating temperature, associated with disease of the ear, either with or without symptoms of meningitis, are sufficiently characteristic to justify operative interference.

CEREBRAL ABSCESS.

Cerebral abscess is quite rare in children, decidedly more so than is cerebral tumour. In Gowers' collection of 223 cases, only 24 were under ten years of age. In infants, abscess is one of the least frequent diseases of the brain, and up to five years it is exceedingly rare.

Etiology.—By far the most frequent cause in children is otitis. This is the origin of the great majority of the cases. Abscess rarely complicates acute otitis, but is seen with the chronic form. Exactly how otitis causes cerebral abscess it is not always easy to determine. Toynbee was the first to call attention to the fact that cerebellar abscess was most frequent with disease of the mastoid cells, and cerebral abscess with otitis media. Usually there is caries of the petrous bone, but there may be none. The infection may extend through the small veins traversing this bone, or along the lateral sinuses to the cerebellum. Abscess is often attributed to the retention of pus in the ear, but it may occur when the discharge is free.

Traumatism is the second important etiological factor. Abscess may be associated with fracture of the skull, or follow simple concussion. The abscess is generally in the neighbourhood of the injury, but occasionally is produced by [illegible]. In one instance, reported by Wagner, thrush was believed to be the cause of cerebral abscess, the same fungus that existed in the mouth being found in the brain, which in this case was studded with small abscesses. Abscess may be the result of infectious emboli associated with general pyæmia, though this is rare in early life; and finally it may occur without any assignable cause.

Lesions.—The most frequent seat of the abscess is, first, the temporo-sphenoidal lobe; secondly, the cerebellum; thirdly, the frontal lobes. Other locations are very rare. Abscesses are usually single. In size they vary from that of a small cherry to an orange. One case was observed by Meyer, in which an abscess occupied one entire hemisphere. The contents are usually thick greenish-yellow pus, which may be very fetid. When abscesses have lasted for some time they are usually surrounded by dense pyogenic membrane, and may become encysted. The pathological process may be slow, and often is apparently stationary for a long period. Abscesses may rupture into the ventricles, less frequently upon the surface of the brain, causing meningitis, or the pus may even escape externally through the auditory meatus, as in Lallemand's case.

Symptoms.—These are general and local. The general symptoms are much the more important for diagnosis, and often are the only ones present. The local symptoms are those of a tumour. The clinical history of a case of abscess of the brain may be divided into three stages: First, the period of onset, or early acute inflammatory symptoms, fever, etc., which attend the formation of pus. Secondly, the latent period, or period of remission, in which very few symptoms are present. In many acute cases this stage is wanting altogether; in the chronic cases it may last for months, or even years. Thirdly, the final period, with recurrence of active cerebral symptoms, followed by death in a few days.

The onset may be accompanied by symptoms so slight as almost to escape notice. In most cases, however, headache and fever are present. The headache is usually severe, and often localized upon the affected side; in cerebellar abscess it may be occipital. The fever is moderate in intensity, and continuous. In addition there may be vertigo, vomiting, general convulsions, and cessation of the aural discharge, if one has been present. The duration of this stage is variable; it may be only a few days, or several weeks. It is shorter in traumatic cases, and in those which are due to pyæmia.

The latent stage, or period of remission of symptoms may be quite short—only a few days' duration—and it is often absent. During this period the temperature may fall quite to the normal, and the headache disappear, or be only occasional and slight. However, if any focal symptoms have been present they remain unchanged.

The symptoms of the terminal stage are due to a rapid extension of the inflammatory process, with œdema and softening about the abscess, sometimes to rupture into the ventricle, and sometimes to meningitis. The fever now returns, and may be high. There is headache, often very intense and continuous; there may be delirium and convulsions, and the gradual development of coma. In addition there may be vomiting, paralysis, opisthotonus, retracted abdomen, and the other symptoms of meningitis. Occasionally all the earlier symptoms may be latent, and the terminal symptoms may be the only ones present. In infants, the fontanel is usually large and bulging; convulsions are rather more frequent than in older children.

The local symptoms of abscess are rather indefinite, owing to its usual situation. Abscesses of considerable size may exist in the temporo-sphenoidal lobe, in the central part of the frontal lobe, or in the cerebellum, without any definite local symptoms. If the abscess is near the motor area, there are the usual symptoms of disease in this location, spasm, or paralysis of the face, arm, or leg. A cortical or sub-cortical abscess is likely to cause convulsions. Cerebellar abscess may give rise to occipital headache, frequent vomiting, and when the abscess is large enough to press upon the middle lobe, there may be inco-ordination of the muscles of the extremities. Optic neuritis may be present, but other symptoms relating to the cranial nerves are rare. Localized tenderness over the scalp, when persistent, is a symptom of importance, and may serve to locate the abscess, if it is superficial.

Diagnosis.—Of the general symptoms, the most important for diagnosis are fever, headache, delirium, and terminal coma. These become particularly significant when following otitis or traumatism. The differential diagnosis of abscess is to be made principally from tumour and meningitis, and from these conditions more by the history and general course of the disease than by any special symptoms. The diagnosis of abscess from tumour is considered in connection with the latter disease. It is more difficult to distinguish between meningitis and abscess, since the two processes are often associated. With meningitis convulsions are more common, but they are rarely localized; rigidity and the inflammatory symptoms are more intense; the course is usually more rapid and more regular, being rarely interrupted, as is the course of abscess. From the cerebral symptoms occurring with otitis it is extremely difficult to distinguish abscess, for, according to Gowers, optic neuritis may be present in the former as well as in the latter condition. The more intense and prolonged are the cerebral symptoms and the more marked the neuritis, the greater are the probabilities of abscess.

Prognosis.—The prognosis in cerebral abscess is always grave, unless accessible to surgical operation. The progress may be slow, or rapid, but it is inevitably from bad to worse, and sooner or later the disease, if not interfered with, proves fatal.

Treatment.—The medical treatment of abscess in its active stage is that of any acute intracranial inflammation,—ice to the head, absolute quiet, free catharsis, and full doses of the bromides or antipyrine or morphine, if pain is intense. The absolutely hopeless condition of these cases when left to themselves, and the recent brilliant results from surgical operations, should lead the physician to urge operation in every case.*

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* For a discussion of the surgical aspects of this question, see "Brain Surgery," by M. Allen Starr, M. D., and "Pyogenic Infectious Diseases of the Brain and Cord," by William McEwen, M. D.

CEREBRAL TUMOUR.

Very little has been added to our knowledge of cerebral tumour in children since the exhaustive monograph of Starr, which appeared in Keating's Cyclopædia in 1890. It is to this article that I am indebted for most of the facts in this chapter.

Varieties and Location.—Tumour of the brain is not very infrequent, and may be seen even in infancy. From this time up to puberty there is no period of special susceptibility. In two hundred and sixty-nine of the cases in Starr's collection, in which the nature of the tumour was stated, the following were the varieties:

Tubercle	152	cases.
Glioma	37	"
Sarcoma	34	"
Glio-sarcoma	5	"
Cyst	30	"
Carcinoma	10	"
Gumma	1	"
	269	"

Tuberculous tumours are more often multiple than are other varieties. Their most frequent seat is the cerebellum; next to this the pons and crura cerebri. They are rarely cortical or central. Glioma is most often found in the cerebellum or in the pons, and next in the cortex; but it is rarely central. Sarcoma is most frequently in the cerebellum; next to this, in the order of frequency, in the pons, the basal ganglia, and the cortex. Cystic tumours are either central or cerebellar. Taking the cases as a whole, the most frequent seat of tumour in children is, first the cerebellum, second the pons, third the centrum ovale.

Tuberculous tumours are occasionally seen in infancy, but they occur most frequently between the ages of five and twelve years. They are usually secondary to tuberculosis elsewhere, especially in the lungs and in the bronchial lymph nodes. They most frequently start from the membranes, rarely being centrally situated, and extend inward, infiltrating the superficial portion of the cerebellum or cerebrum. There is almost invariably localized meningitis at the site of the tumour; there may be adhesions between the dura and pia mater, and the disease may extend to the cranial bones. In size, these tumours vary from a small pea to a child's fist. They may be softened and broken down at the centre, or cheesy throughout. They are the result of a localized tuberculous inflammation, which does not differ essentially from that seen in other parts of the body.

Glioma is not infrequent in infancy. It is probably connected in every case with the ependyma of the ventricle. It repeats the structure of the neuroglia, being composed of connective tissue and branching cells.

Sarcoma may be of the spindle-celled or the mixed variety. It grows much more rapidly than glioma. The two varieties are not infrequently combined in the same tumour—glio-sarcoma.

Cystic tumours are sometimes sarcomatous in origin, the wall of the cyst containing sarcoma cells, and they may also be parasitic, from the growth of the echinococcus. They may be found in any part of the brain.

The other varieties of sarcoma, gumma and vascular tumours, are exceedingly rare until after puberty.

As the tumour grows, secondary lesions are produced in most of the cases. These are the result of pressure upon arteries, causing localized anæmia, or even cerebral softening; or upon veins, producing congestion and œdema. When affecting the middle lobe of the cerebellum, pressure upon the venæ Galeni may lead to effusion into the ventricles. Localized meningitis over tumours superficially situated is the rule, and this may be the cause of some of the symptoms. Rarely, cerebral hæmorrhage may be associated.

Etiology.—The causes of cerebral tumours are for the most part unknown. In a few instances there is a history of definite traumatism. Sarcoma or carcinoma may be secondary, and tuberculous tumours are probably always so.

Symptoms.—These may be divided into two groups: first, the general symptoms which are common to tumours of all varieties, and are independent of location; secondly, the local symptoms depending upon the situation of the growth.

General symptoms.—One of the most frequent is headache. Though it varies much in its severity, character, and position, it is rarely absent. It is apt to be severe, and may continue for a long period, or it may be intermittent. The location of the pain has no definite relation to the situation of the tumour. It may be accompanied by sensations of tightness, compression, or tension in the head. It may be associated with localized tenderness of the scalp; when this is constant it is a valuable symptom for diagnosis, as it often occurs with tumours superficially located.

General convulsions are frequent in the early stage, but separated by quite long intervals; they become more frequent and more severe as the disease progresses. All degrees of severity are seen, from slight twitchings and temporary loss of consciousness, to typical epileptiform seizures. They are most common when the growth is rapid and when complicating meningitis is present. Attacks of vomiting or of localized spasm may for a considerable time precede general convulsions; and in a single attack there may be first localized and then general convulsions.

Mental symptoms are generally present in great variety and complexity. There may be only fretfulness and irritability, or a marked change in disposition. These symptoms are so frequent from other causes in children that they excite no apprehension, unless to them are added dulness, apathy, and somnolence. Later in the disease there may be attacks of hypochondriasis, or of melancholia; there may be periods of wild, almost maniacal excitement; and, finally, the mental impairment may approach a condition of imbecility.

Optic neuritis and optic-nerve atrophy are very frequent, occurring, according to Starr, in eighty per cent of the cases. This is only recognised by the ophthalmoscope, as there may be no disturbance of vision. The optic neuritis is generally double, appears earlier, and is more constant in basal tumours than in those at the convexity, or those centrally located.

Vomiting is very frequent, but diagnostic only when it occurs suddenly without assignable cause, and without nausea or other symptoms of indigestion. It is especially significant when frequently repeated, and of more importance in older children than in infants.

Vertigo is often associated with vomiting. At first it is occasional and seen upon changing position, but later it may be quite constant, especially with tumours in the posterior fossa.

Disturbances of sleep are frequent. There is usually insomnia, but sleep may be broken by hallucinations, accompanied by attacks of screaming; rarely is there persistent drowsiness until toward the end of the disease.

Local symptoms.—These depend upon the situation of the tumour, but not at all upon its anatomical character. Local symptoms may be wanting entirely, and they may vary much in different cases even with tumours in the same situation. They are modified by the size and by the rapidity of growth, and by the existence of local meningitis.

In tumours of the cortex, the meninges are likely to be involved, especially with tuberculous and gliomatous growths. The pathological process may extend from within outward or from without inward. The most frequent general symptoms in such cases are headache, circumscribed tenderness of the scalp, convulsions, and mental symptoms. Optic neuritis, vomiting, and vertigo are not so common. Tumours situated in the frontal lobe, as a rule, present few symptoms and may be entirely latent. Irritation of the frontal lobe may extend to the motor area and cause convulsions either local or general; but not often is there paralysis. Tumours of the left side (of the right side in left-handed persons) in the third frontal convolution may cause motor aphasia.

Tumours in the motor convolutions along the fissure of Rolando produce the most definite and uniform local symptoms. When situated at the upper portion the leg is affected, at the middle portion, the arm, and at the lower, the face. Irritative symptoms, such as rigidity or clonic spasm, commonly precede for some time the paralysis which results from pressure or destruction. These attacks of localized convulsions may begin in the face, arm, or leg; but they usually extend more or less rapidly until all three are involved. There is no loss of consciousness, but there may follow a slight transient paralysis. Such attacks are known as "Jacksonian epilepsy," and form one of the most diagnostic symptoms of cerebral tumour. Localized spasm may be associated with anæsthesia or other disturbances of sensation. The paralysis generally first affects one extremity—the arm or leg, according to the location of the tumour—and afterward it may involve the entire side, including the face.

If the tumour is centrally located, or at the base, hemiplegia may be an early symptom from pressure on the motor tract. With cortical paralysis there may be associated ataxia and anæsthesia.

Tumours of the parietal lobe may give no local symptoms. At times there are disturbances of muscular sense, tactile sensibility, or sensations of pain and temperature. If the inferior parietal lobule of the left side is affected, there may be word-blindness, or inability to understand written language.

Tumours of the occipital lobe produce, as the only constant local symptom, hemianopsia. This is usually bilateral, affecting the same side of both eyes, being on the side opposite to that of the lesion—i. e., a tumour on the right side causes blindness in the left half of both eyes, so that the patient sees nothing to the left of a line directly in front of him. Instead of hemianopsia, there may be only irritation and various disturbances of sight.

Tumours of the temporo-sphenoidal lobe may be latent, or, if on the left side, may cause word-deafness—i. e., inability to understand the significance of spoken language.

Tumours in the island of Reil when situated upon the left side (right side in left-handed persons) may cause motor aphasia or disturbances of speech. If they are large they may produce symptoms by pressure upon the motor tract,—hemiplegia or monoplegia.

Tumours of the basal ganglia cause marked general symptoms, but none of a definitely local character. The important symptoms relate to the various tracts or bundles of fibres which pass from the cortex through the internal capsule. These include the motor and the various sensory tracts, the olfactory, auditory, visual, and speech tracts. Any of these may be pressed upon, and the nature of the symptoms will depend upon the size of the tumour and the extent of the pressure. If only the anterior part of the capsule is affected there may be no symptoms; if the middle fibres, hemiplegia and disturbances of articulation; if the posterior fibres, hemianæsthesia. All these may be associated, and any of them may be complete or partial. Tumours in this situation are apt to implicate the cranial nerves. Optic neuritis is quite constant, and appears early. Localized or general convulsions are rare.

The peculiar symptoms pointing to tumours of the crura cerebri are nystagmus, strabismus, and loss of pupillary reflex, sometimes with general muscular inco-ordination, and a staggering gait. There is usually third-nerve paralysis on the side of the tumour, and on the side opposite to the hemiplegia with which it is often associated. This variety of crossed paralysis is quite diagnostic. The symptoms of third-nerve paralysis are external strabismus, dilatation of the pupil, and ptosis. In these cases optic neuritis appears early. There may be a complicating hydrocephalus. While hemiplegia is commonly present with large tumours, it may be absent with small ones, or may appear later than paralysis of the third nerve.

Tumours of the pons are quite common. The diagnostic symptoms consist in crossed paralysis, the cranial-nerve symptoms being on the side of the tumour, and the general motor and sensory symptoms on the opposite side. When the seat is the upper half of the pons, the third and fifth nerves are apt to be implicated, giving rise to ptosis, dilatation of the pupils, external strabismus, trophic disturbances such as ulceration of the cornea, and neuralgic pain in the face. Tumours in the lower half of the pons involve the sixth, seventh, and eighth nerves, causing internal strabismus, contracted pupils, facial paralysis, sometimes deafness, and auditory vertigo. Other symptoms associated with tumours of the pons are headache, vomiting, and optic neuritis; convulsions being rare.

Tumours of the medulla are recognised by the involvement of the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves. There are difficulty of deglutition, irregular respiration, irregular pulse, and vaso-motor disturbances, such as flushing of the face and perspiration. There may be projectile vomiting, polyuria or glycosuria, opisthotonus, difficulty in articulation or in sucking, and in protrusion of the tongue. When large, these tumours may produce symptoms of pressure upon the motor or sensory tracts,—paralysis, partial anæsthesia, with rigidity and exaggerated reflexes.

Tumours of the cerebellum are especially important, this being the most frequent location in childhood. When only one hemisphere is affected there may be no local symptoms. Tumours involving the middle lobe, or those large enough to produce pressure upon the middle lobe, give rise to vertigo and cerebellar ataxia. Vertigo is especially frequent; it may occur with headache. Cerebellar ataxia is different from the ataxia due to a spinal-cord lesion, and strikingly resembles that of intoxication. It may increase until the patient is unable to walk, although there is no loss of muscular power. Vomiting is a frequent symptom, as are also optic neuritis, and headache which is usually occipital. When there is secondary hydrocephalus, as is not uncommon, mental symptoms are present, and there may be enlargement of the head. Opisthotonus is occasionally seen, but general convulsions are rare.

Diagnosis.—The size of the tumour is to be determined mainly by the general symptoms, special attention being given to the order of their development. A diagnosis as to the nature of the tumour is really not of much importance; but some information upon this point may be gained from the consideration of its etiology, the rapidity of its growth, and the age of the patient. Cerebral tumour may be confounded with abscess, tuberculous meningitis, chronic basilar meningitis, and chronic hydrocephalus. The symptoms distinguishing tumour from abscess are the following: Tumour may occur at any age; without definite etiology, excepting when tuberculous; the progress is steady, but generally slow, new symptoms being continually added; headache is more constant and more severe; optic neuritis more frequent; cranial nerves more often involved; mental disturbances more marked; focal symptoms are often definite; fever is absent; duration, six months to two years. As compared with the above, abscess is not so frequent, being especially rare in infancy; there is a definite history of traumatism or ear disease; progress more irregular; symptoms often intermittent; headache less severe; mental symptoms less marked; optic neuritis and involvement of the cranial nerves less frequent; focal symptoms usually indefinite; localized tenderness over the scalp more constant; fever present except in the latent period; the most frequent complication is acute meningitis.

Cases of tuberculous meningitis which may be confounded with tumour are those of slow course sometimes seen in older children. The difficulty in diagnosis is increased by the frequent association of tuberculous tumours with tuberculous meningitis. The main points of difference are that in tumour the symptoms are more localized and the course generally much slower. Almost every individual symptom, however, may be present in the two conditions.

Chronic basilar meningitis may produce symptoms almost identical with those of tumour in the posterior fossa. It is, however, confined to infancy, and is frequently syphilitic. Hydrocephalus and opisthotonus are much more marked than are usually seen with tumour.

Chronic hydrocephalus may resemble tumour; this occurs so frequently as a lesion secondary to tumour that the question often arises whether there is only hydrocephalus, or there is in addition a tumour. Primary hydrocephalus is usually congenital, and commonly attains to a greater degree than is seen in secondary hydrocephalus.

Prognosis.—The prognosis in cerebral tumour, while bad, is not hopeless. Cases are occasionally seen which exhibit all the characteristic symptoms of tumour, even including optic neuritis, which recover perfectly. These are probably syphilitic, although often no such history can be obtained. In other cases, most frequently of a tuberculous nature, an arrest of the growth occurs and the patient recovers with some function of the brain impaired; usually there is loss of vision or some paralysis. In most cases, however, the progress is steadily downward until death.

Treatment.—If there is any reason to suspect syphilis, the iodide of potassium should be given in large doses and continued for a long period;

the effect of this drug even in tumours not syphilitic is sometimes beneficial. Starr refers to a case in which symptoms of six months' duration, including optic neuritis, entirely disappeared under the use of mercury and the iodide. The tumour was supposed to be gumma, but an autopsy obtained six months later showed it to be a sarcomatous cyst. For a discussion upon the surgical aspect of the treatment of brain tumours, the reader is referred to Starr's work on Brain Surgery.

HYDROCEPHALUS.

Hydrocephalus or "water on the brain," consists in an accumulation of serum in the cranial cavity. This may be between the dura mater and the pia (external hydrocephalus) or in the ventricles of the brain (internal hydrocephalus). The former is secondary and is quite rare, while the latter is not uncommon. Hydrocephalus may be acute or chronic.

Acute Hydrocephalus is secondary to basilar meningitis, which is usually of tuberculous origin. The terms tuberculous meningitis and acute hydrocephalus are sometimes used synonymously. A moderate distention of the ventricles is frequent in all varieties of acute meningitis. The amount of fluid in acute hydrocephalus is not great, there being rarely more than three or four ounces present.

Chronic External Hydrocephalus except in its mild form is extremely rare, and is nearly always a secondary lesion. It may follow meningeal hæmorrhage, pachymeningitis or any lesion causing cerebral atrophy. It is seen in its most marked form associated with congenital malformations of the brain, particularly imperfect development of the hemispheres. (See Fig. 137.) On incising the dura mater a few ounces, or sometimes even a pint, of serum may escape. The convolutions are somewhat flattened, and may be greatly atrophied. Other lesions are found either in the brain or in the dura mater. External hydrocephalus may cause enlargement of the head and separation of the sutures, and in fact most of the symptoms of the internal variety; but usually it is not severe enough to give rise to any decided symptoms.

CHRONIC INTERNAL HYDROCEPHALUS.

This is the important variety, and when no qualifying term is mentioned this is the form of hydrocephalus which is always understood.

Etiology.—This occurs both as a primary and a secondary condition. When secondary it is usually associated with tumours of the base of the brain or with chronic basilar meningitis, either simple or tuberculous. It is in these cases a mechanical condition caused by pressure which obliterates the openings from the lateral ventricles into the fourth ventricle, or the foramen of Magendie.

The causes of primary hydrocephalus are as yet very little understood. In a large proportion of the cases the disease is congenital, generally beginning in the latter months of intra-uterine life. Some of these cases are clearly syphilitic. D'Astros[*] has collected nine cases and added three others, in which hydrocephalus was associated with lesions undoubtedly syphilitic. When due to syphilis, the disease may at the same time be congenital. Rickets and hydrocephalus are occasionally associated, but so infrequently as to make a definite etiological connection between them very doubtful. The rachitic head has been so often mistaken for hydrocephalus that an erroneous notion has arisen as to the frequent association of these two diseases. This point will be referred to more fully under diagnosis. Chronic hydrocephalus is often attributed to tuberculosis, but here again the connection is a very doubtful one. Heredity is a factor of some importance; numerous instances are on record where two children in the same family have been affected. Hydrocephalus not infrequently develops after successful operations upon spina bifida or encephalocele.

Lesions.—The difference between the primary and secondary cases is chiefly one of degree. The amount of fluid in secondary cases is rarely more than three or four ounces. In primary cases it is usually from half a pint to one pint, but it may be very great. In one of my own cases there was removed from the head of a child, who died at four months, five pints of fluid. Larger quantities than this have been reported, but not at so early an age. In composition this resembles the cerebro-spinal fluid. An examination in one of my cases showed it to be a clear, translucent fluid, slightly alkaline in reaction, specific gravity 1005, containing sodium and potassium chlorides, alkaline phosphates, and a trace of albumin. In some specimens sugar is found. In cases of inflammatory origin the amount of albumin is generally larger, and the fluid may be slightly turbid. The effusion may become purulent from accidental infection resulting from operation, from rupture, or, as in one of my cases, from infection through the sac of a spina bifida with which it was complicated, the process extending to the brain through the central canal of the cord.

The changes in the brain result from the gradual accumulation of fluid in the ventricles. The septum lucidum is usually broken down, and all the avenues of communication between the ventricular cavities are greatly enlarged. The continuous distention results in a gradual thinning of the brain substance which forms the ventricular walls; often these are found only one fourth of an inch in thickness, or even less than this, the cortex being a mere shell (Fig. 136). In one of my autopsies the ependyma of the ventricle and the pia mater were in places actually in contact, all of the brain tissue having been absorbed; the brain resembled a large double cyst. In a case of Peterson's, with the exception of a small portion of one temporo-sphenoidal lobe, all of both hemispheres had disappeared, the cerebellum and basal ganglia alone being intact. The brain is always anæmic, and the gray and white substance may be indistinguishable. The changes are largely mechanical, the microscope showing, in my case just referred to, only granular matter and round nuclei evidently from broken-down nerve cells. In less severe cases the changes may be slight. It is, however, always surprising to see the amount of compression which the cortex will tolerate without interference with its functions, provided the pressure comes gradually. The ependyma may be normal, but it is usually somewhat thickened and pale, sometimes granular, and may be infiltrated with new cells. When infection takes place an acute ependymitis may be set up. Chronic inflammation of the ependyma is thought to be the essential lesion in many of the primary cases, whether of simple or syphilitic origin.

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[*] Revue Mensuelle des Maladies de l'Enfance, ix, 481, 543.

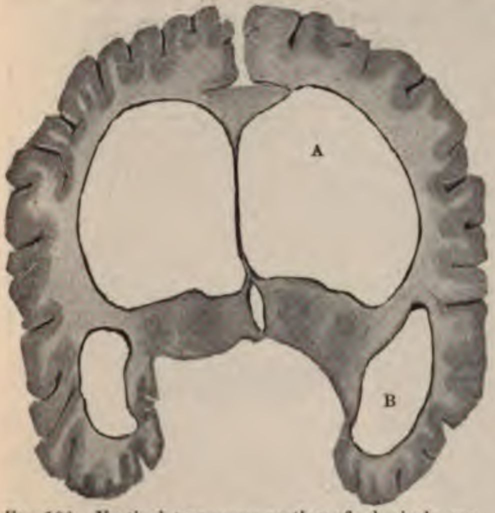

FIG. 136.—Vertical transverse section of a brain in congenital hydrocephalus, from a child who died at the age of three weeks. *A*, distended lateral ventricle; *B*, its descending horn.

The bones of the skull are markedly affected; the sutures at the vault are widely separated, and sometimes even those at the base. After the removal of the fluid the head collapses, giving an appearance which has been well likened to a "bag of bones." It should not be forgotten, however, that hydrocephalus may coexist with premature ossification, in which case the head may be small. In the cases which recover, the wide gaps in the skull may be closed by the development of wormian bones; but ossification is often not complete until the fifth or sixth year.

The most frequent lesion associated with congenital hydrocephalus is spina bifida, in which cases there may also be a patency of the central canal of the spinal cord; more rarely meningocele or encephalocele are met with. Sometimes there are deformities in other parts of the body, such as club-foot or hare-lip.

Symptoms.—Hydrocephalus may exist with a small head. In this condition there is usually premature ossification of the cranial bones. Four such cases have come under my notice, one child having lived to be fourteen months old. These children are usually idiotic, and die at an early age, often from convulsions. In such cases other malformations of the brain are frequently associated.

Hydrocephalus, with the exceptions mentioned, is recognised by the increased size of the head. In order to estimate the amount of enlargement, it must be remembered that at birth the circumference of the normal head is about 14 inches, and at one year from 18 to 19 inches. The degree of enlargement in hydrocephalus may be very great. In one of my cases, the head at four months measured 24½ inches. In another at ten and a half months, 26¾ inches. Steiner has reported a remarkable case in which the head at eight months measured 32¾ inches. When the enlargement of the head is not great the diagnosis is not so easy. Hydrocephalic enlargement is commonly symmetrical and in all directions. The head is sometimes globular in outline and sometimes pyramidal (Fig. 138). The forehead is exceedingly high and projecting, and there is a prominence of the frontal eminences seen in no other form of enlargement. The sutures may be separated from half an inch to two or three inches; the fontanel is very large, tense, and bulging;

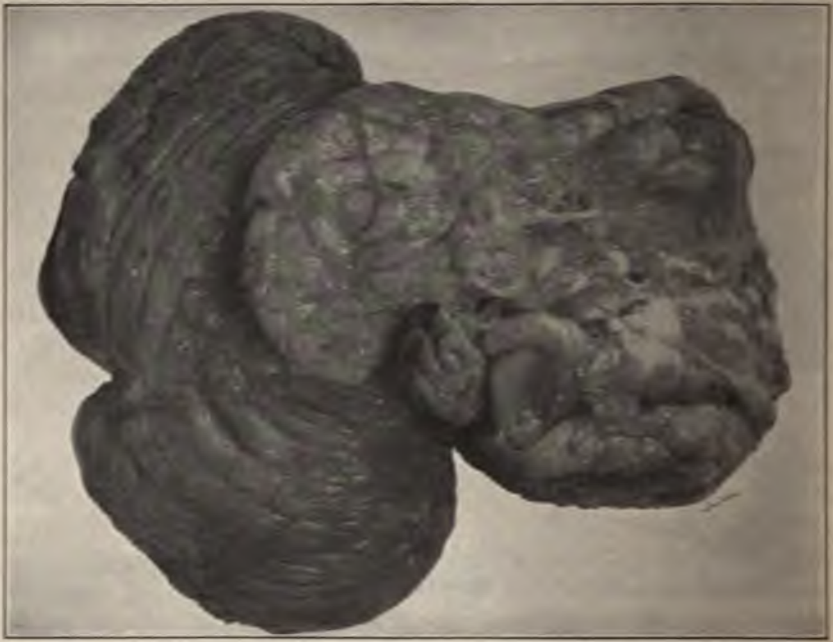

FIG. 137.—Brain in external hydrocephalus, showing imperfect development of the hemispheres.

Patient three and a half months old; head measured 20¼ inches; increase in size, 2 inches in the six weeks before death; symptoms were typical of ordinary internal hydrocephalus. In the picture the small size of the cerebrum is best judged by comparison with the cerebellum, which is normal. The hemispheres were rudimentary; the basal ganglia were normal; the cranial cavity contained about one pint of fluid.

the veins of the scalp are enlarged and prominent. In marked cases fluctuation may be readily obtained, and the head may even be distinctly translucent.

In the acquired form all these symptoms are less marked, and if ossification of the skull has taken place it is often impossible to discover any increase in size. The rate of growth of the head varies much in different cases, and it is the surest measure of the progress of the case. The increase in circumference is usually from one to three inches a month.

The primary cases are for the most part of congenital origin, and the child may die *in utero*. At other times the process may have advanced so far before birth that puncture of the head is necessary before delivery is possible. In perhaps the majority of cases no symptoms are observed at birth, or the head is only slightly larger than normal. Usually nothing is noticed until the child is two or three months old, when it is discovered that the head is increasing in size at an abnormal rate. If the progress is rapid, other symptoms are soon evident: the infant can not hold up its head; it is lethargic, and all its perceptions are dulled, sight and hearing included; there may be a general flaccid condition of all the

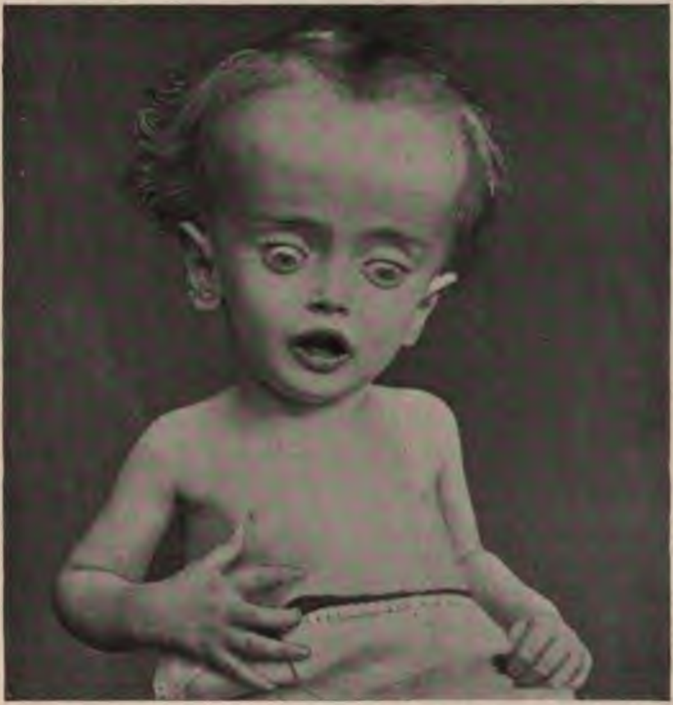

FIG. 138.—Chronic hydrocephalus of average severity; head of pyramidal shape; showing characteristic expression of the eyes.

muscles of the extremities due to a slight general paresis, but more often there is rigidity, which is usually most marked in the legs, but sometimes in the arms; the hands are often clenched, with the thumbs adducted; the reflexes are exaggerated; the pupils are generally contracted and equal, though they may be dilated; nystagmus and convergent strabismus are often present. Convulsions may occur from time to time, or may be deferred until near the close of the disease. As the head enlarges the body usually wastes, and the disproportion between the two may seem greater than it really is.

Such congenital cases rarely see the end of the first year, and are often fatal during the first six months. The causes of death are marasmus, convulsions, and intercurrent disease, rarely rupture of the head.

In the cases which develop more slowly, the symptoms are quite different. The head may not attain at eighteen months the size reached in the other cases at the third or fourth month. The surprising thing about many of these cases is that the distinctly cerebral symptoms are so few. Where the pressure develops gradually, the brain seems able to tolerate an almost indefinite amount of it. The more readily the bones of the skull yield to pressure the fewer are the nervous symptoms; hence, other things being equal, they are less marked where the disease begins before the sutures are firmly ossified than in the later cases. A comparatively small amount of effusion may cause very marked symptoms in a child two or three years old, while a much larger amount in an infant of a year, may produce much less disturbance. It is for this reason that secondary hydrocephalus causes such striking symptoms, although the accumulation of fluid is small.

Whether the progress of these cases is slow or rapid, the development of the children is greatly retarded. Many are not able to support the head until two or three years old; frequently they do not walk until five or six years old. The special senses are generally not noticeably affected, but intelligence in most cases is interfered with,—in some only slightly, in others very markedly, while some are idiotic. Contractions of the extremities are occasionally seen, but usually more of the hands than the legs. Sensation is not often affected. The course is a very chronic one. From time to time there are exacerbations of the symptoms, and even intercurrent meningitis may be excited.

Prognosis.—Most of the congenital cases are fatal before the end of the first year. It is very rare that a hydrocephalic child reaches the age of seven years. The process may go on up to a certain age and then cease spontaneously, and the child may go through life with a head very much larger than normal and usually with a mental condition somewhat impaired. Retrogression of the symptoms is, however, never to be looked for.

Diagnosis.—The most important symptom is the enlargement of the head, and this can only be arrived at by careful measurement and comparison with the normal size. The rapidity of growth is quite as important for diagnosis as the fact of enlargement. If the head grows more than an inch a month there can be little doubt. The enlargement most frequently confounded with hydrocephalus is that which occurs in rickets. In the latter disease it is almost invariably irregular; there are prominences over the two frontal eminences and over the parietal bones, often with furrows between them; the size of the head is chiefly due to thickening of the bones of the skull; the marked prominence of the forehead is not seen, and the increase in the bi-parietal diameter is not present; furthermore, there are other signs of rickets.

Treatment.—If there is any suspicion of syphilis, mercurial inunctions should be employed, and potassium iodide given internally in full doses. Of all the operative measures that have been proposed for this condition, and their name is legion, the only one at the present time which seems to hold out any reasonable prospect of permanent improvement is auto-drainage. This consists in establishing a communication between one of the lateral ventricles and the sub-arachnoid space. By this means the fluid is conducted to a place from which it can be absorbed. A considerable number of cases have now been treated in this way. The dangers of the operation are very great; fully half the patients having died as the direct result of it. Of those who have survived, a number have shown improvement and a few very striking improvement, but no complete cures have been reported.*

INFANTILE CEREBRAL PARALYSIS.

Synonyms: Spastic diplegia, paraplegia, or hemiplegia.

Under the term cerebral paralysis are included several groups of cases with causes quite dissimilar, but having certain definite clinical features in common. While the symptomatology is quite clear, there are many questions relating to the pathology that are not yet fully settled, although much has been added to our knowledge within the last few years. Paralysis depending upon cerebral tumour, abscess, or hydrocephalus is not included in this chapter.

The cases of cerebral paralysis may be divided into three groups, according as the paralysis depends upon conditions existing prior to birth, upon those connected with birth, or upon those of subsequent development.

I. **Paralysis of Intra-Uterine Origin.**—This is the least frequent condition. In such cases there is some congenital defect in the brain, due sometimes to arrest of development, at others to such intra-uterine lesions as hæmorrhage or thrombosis. There may be porencephalus, or cysts extending deeply into the substance of the brain, sometimes communicating with the ventricles. The origin of this condition is for the most part unknown. In rare cases the paralysis is due to cortical agenesis,* a condition in which the brain may seem normal to the naked eye, but the microscope shows a complete arrest in the development of the cells of the cortex, usually affecting both hemispheres. In still other cases there are found gross defects in development in the motor centres of the cortex. Such a lesion is shown in Fig. 149, page 806. Cases in which there is conclusive evidence of intra-uterine hæmorrhage are very rare.

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* For a discussion of the surgical aspects and literature, see A. S. Taylor, American Journal of Medical Sciences, August, 1904.

Symptoms.—In most of the paralyses due to intra-uterine lesions, loss of power is only one of the symptoms, and usually not the most prominent. It is rare that there is not some mental impairment, and usually idiocy is present. The type of paralysis is nearly always diplegic or paraplegic. Where this is due to arrested cortical development, a general flaccidity of the muscles may be seen instead of the rigidity so characteristic of the other forms of cerebral paralysis.

II. **Birth-Paralysis.**—Cerebral birth-paralysis is due in nearly all cases to meningeal hæmorrhage. The primary lesions and the early symptoms have already been described in connection with the Diseases of the Newly Born. The secondary lesions present considerable variety. There may be found (1) meningo-encephalitis, (2) atrophy and sclerosis of the cortex, (3) cysts upon the surface, (4) secondary degenerations in the spinal cord.

1. Meningo-encephalitis.—This lesion is often quite diffuse. There is thickening of the pia mater, and it is usually adherent to the brain substance. The cortex is involved to a variable degree, depending somewhat upon the time which elapses between the initial lesion and the autopsy. The following were the microscopical changes found by Sachs † in the brain of a child in my wards at the Babies' Hospital, who died at the age of one year of measles: The lesions were found everywhere in the cortex. The pia was universally adherent, and showed general cellular infiltration; its blood-vessels showed marked cellular proliferation, and the veins in the sub-pial space were dilated and filled with blood. In the pia dipping in between the convolutions similar changes were present. In the cortex few if any normal pyramidal cells were found, but in the outer layers were an enormous number of small glia cells. Many of the blood-vessels showed a cell-proliferation of their walls. There was also

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* For fuller description, see Sachs's Nervous Diseases of Children, 1895, p. 601.

† The clinical features of this case are quite as interesting as the pathological findings. The child was a first-born, delivered after a dry labour of forty-eight hours. It was asphyxiated, and from the first days of its life it had attacks of convulsions, usually repeated many times a day. During one of these convulsions the photograph from which Fig. 140 was made, was taken by Dr. Peterson. The child had the symptoms of typical spastic paraplegia—the arms being, however, slightly involved—retarded mental development, and convergent strabismus.

a degeneration in the pyramidal tracts of the anterior columns of the cord.

2. Atrophy and sclerosis.—These changes vary much in extent and degree. There may be only a circumscribed area in which the convolutions are small, firmer than usual, and covered with an adherent pia, or there may be an atrophy so extensive as to involve a large part of one hemisphere (Fig. 139), or sometimes of both hemispheres. Usually the lesion is somewhat diffuse over the convexity of both sides, and much more frequently of the anterior than of the posterior half of the brain. Where a depression of the brain exists the space is filled with cerebrospinal fluid, and in many cases there is a deformity of the skull.

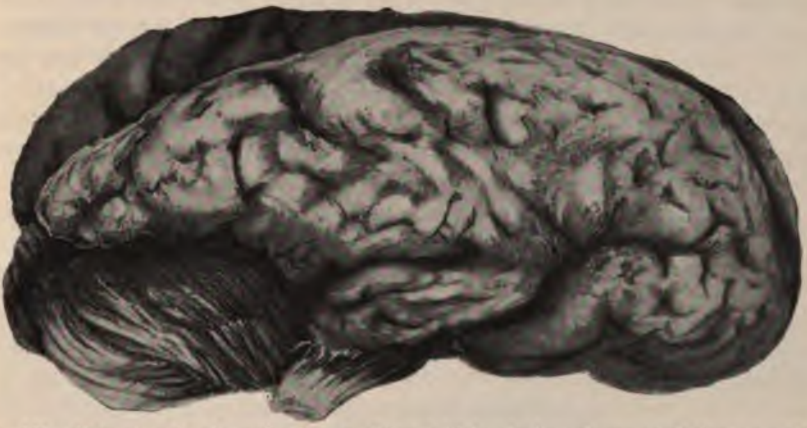

Fig. 139.—Extensive atrophy and sclerosis of the right hemisphere, from an infant seven and a half months old; probably the result of a meningeal hæmorrhage at birth (lateral view).

History.—Twelve hours after birth was seized with general convulsions, which continued for three days. No other symptoms noticed till one month before death, when weakness of left arm was observed. Never held head erect. Was plump and well nourished; died from erysipelas.

Autopsy.—Pia not adherent; a large cyst occupied the region of the occipital and posterior part of the parietal lobes, showing in its floor discolouration and pigmentation, evidently from an old hæmorrhage. Right optic nerve, tract, and crus much smaller than the left.

3. Cysts upon the surface may occur alone or in connection with the lesions just mentioned. These are usually small, about the size of a walnut, but they may cover a large part of a hemisphere. Such large cysts are sometimes classed as cases of external hydrocephalus.

4. Secondary degenerations of the internal capsule and the lateral columns of the cord are found in most of the cases associated with extensive atrophy and sclerosis, and in many of those in which only meningo-encephalitis is present.

Symptoms.—The type of paralysis will of course depend upon the extent and position of the original lesion. A diffuse lesion is followed by diplegia; one not quite so extensive by paraplegia; one affecting one side only by hemiplegia, or even monoplegia, though this is very rare. The relative frequency of the different forms will vary according to the age at which the patients come under observation. Thus in the statistics of Sachs and Peterson* there were twenty-seven cases of diplegia or paraplegia and twenty-two of hemiplegia. These cases were drawn from miscellaneous sources, chiefly from a general neurological clinic. According to my own observations, which have been chiefly upon infants, the cases of diplegia and paraplegia have outnumbered those of hemiplegia more than four to one. My belief is that the great majority of the congenital cases, or those due to hemorrhage occurring at birth, are diplegias or paraplegias, and that very many of them succumb during the first two years, and never come under the observation of the neurologist; however, the cases of hemiplegia, because of the less serious lesion, live much longer, and hence are more likely to be seen by the specialist. Diplegia and paraplegia will therefore be considered as the characteristic types of cerebral birth-palsy, as the cases of hemiplegia do not differ from those due to later causes—i. e., the acquired form.

In the most severe cases that survive the symptoms of the early days of life there remains some rigidity of the extremities, chiefly of the legs, which is constant or intermittent, slight or well marked. There is often spasm of the muscles of the neck and trunk, giving rise to opisthotonus. In many cases there are frequent attacks of convulsions (Fig. 140). The general physical development of the child is often interfered with, so that he remains small and delicate, and perhaps dies of some acute disease in early infancy, never having been able to sit erect, or even

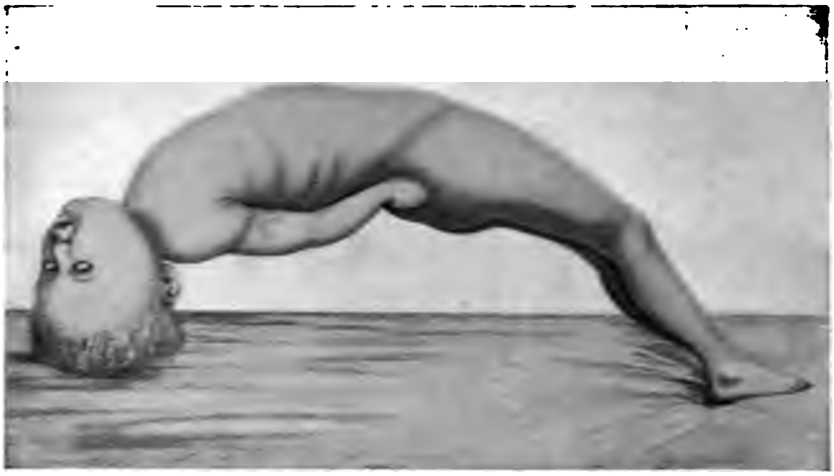

FIG. 140.—Convulsions in spastic paraplegia; from a photograph by Dr. Frederick Peterson during an attack.

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* Journal of Nervous and Mental Disease, May, 1890.

support his head. In other cases the general nutrition is not affected, and life may be prolonged indefinitely, but usually with some degree of mental impairment. This is seen in all degrees; it may be so slight as not to be noticed until the child is two or three years old, or the child may be idiotic. Often these children are not able to stand until they are over three years old, and do not walk alone until they are four or five years old, and then with a peculiar cross-legged gait, owing to spasm of the adductors of the thighs. This may be so great as to entirely prevent walking, and while sitting or lying the thighs may cross each other. These form the typical cases of spastic paraplegia (Fig. 141). All the reflexes are greatly exaggerated. The arms are much less affected than the legs and in about half the number they are not involved at all.

In the mild cases the early symptoms may be overlooked, and nothing excite suspicion until the infant is six or eight months old. There is then discovered unmistakable muscular weakness; the child does not sit up, or even hold up the head when the trunk is supported. Often there is observed before this time a tendency to stiffen the body and to throw the head backward, owing to spasm of the cervical or spinal muscles. The muscular weakness is often mistaken for rickets, or regarded simply as backwardness. A closer examination usually discloses the presence of some rigidity of the extremities, particularly of the legs, and exaggeration of the knee-jerks. As the child grows older other symptoms of imperfect development become more and more evident.

There are changes in the shape of the skull, this being usually smaller than normal in all its diameters, or there may be asymmetry. There is an arrest of development in the paralyzed limbs. These are both smaller and shorter than normal. In many cases abnormal movements are seen, which may be of an irregular choreic type, or they may be athetoid. Epilepsy develops in from 33 to 50 per cent of all these patients.

III. **Acute Acquired Paralysis.**—This is usually of the hemiplegic type, although diplegia and paraplegia may in rare instances be met with. This group includes cases developing at any time after birth, but the great majority of those seen in childhood begin before the fifth year.

Etiology.—The etiology is often obscure. The paralysis sometimes follows traumatism. It is occasionally seen in the course of scarlet fever, measles, diphtheria, variola, and pneumonia. Much more frequently than with any of these diseases it occurs during pertussis, being usually the outcome of a severe paroxysm. The frequency with which these cases are ushered in with convulsions has led many to assign this as the cause of the paralysis. It is possible that the convulsions are sometimes the result and sometimes the cause of the lesion.

Lesions.—The lesions of acute cerebral palsy may be grouped under three heads: (1) those of the blood-vessels; (2) those of the membranes; (3) those of the brain substance.

1. Lesions of the blood-vessels.—There may be hæmorrhage, embolism, or thrombosis. Hæmorrhage is by far the most important. It is usually meningeal, rarely cerebral. It occurs more frequently at the convexity than at the base, and is often diffuse. Meningeal hæmorrhage may result from pachymeningitis. It may be due to traumatism, where it is also from the dura mater; or from the acute hyperæmia accompanying paroxysms of pertussis, where it may be from the dura or the pia; or it may be secondary to thrombosis of the superior longitudinal sinus. The association of hæmorrhage with sinus-thrombosis is not very infrequent. It was found in one of my autopsies upon a patient who died of pneumonia. Cerebral hæmorrhage is extremely rare, but it occurs even in infants; I once saw it in one only two months old.

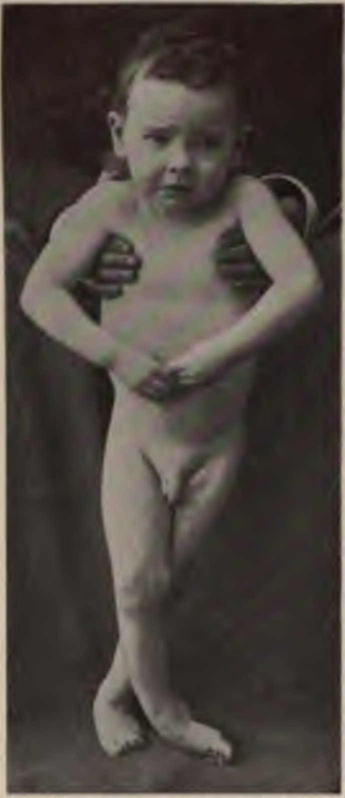

Fig. 141.—Spastic paraplegia.

Child two and one half years old, New York Foundling Hospital, unable to walk or even to stand without assistance. The habitual position of the limbs, which is due to strong adductor spasm, is shown in the picture.

Embolism is rarely found unless associated with acute rheumatic endocarditis, and then usually in children who are over seven years old. As in adults, the usual seat of the embolus is a branch of the middle cerebral artery. It may be single or multiple. Thrombosis has been met with in a small number of cases, but it is extremely rare.

2. Lesions of the membranes.—These are generally the result of an old cerebrospinal meningitis; sometimes they may be of syphilitic origin. In both, however, the process is rarely confined to the membranes; it is a meningo-encephalitis.

3. Lesions of the brain substance.—Atrophy and sclerosis are terminal conditions found in a large number of the autopsies made upon cases where the paralysis has been of long standing. They vary in severity and extent, and are followed by secondary degeneration in the cord, as in cases of birth paralysis. There may be the same development of cysts of the pia mater, or an accumulation of fluid in the arachnoid cavity, these taking the place of the atrophied convolutions. What the primary lesion is in these cases is still a matter of debate. A certain number of them are due to acute poliencephalitis, analogous to acute

poliomyelitis. In other cases a chronic diffuse encephalitis with atrophy is found at autopsy, closely resembling the conditions which follow a meningeal hæmorrhage occurring at birth, yet the children were normal up to the second or third year and there was no acute onset.

Acute paralysis sometimes occurs for which no explanation can be found at autopsy. An infant with pneumonia was admitted to the Babies' Hospital, who had developed, a few days before, typical right hemiplegia. It came on suddenly, with convulsions, and involved the face, arm, and leg. The arm and leg appeared to be completely paralyzed, but in the face the paralysis was incomplete. The paralysis had begun to improve somewhat at the time of the child's death, which occurred a little over a week after its onset. At the autopsy no gross lesion could be discovered. A careful microscopical examination was made by two excellent pathologists, Drs. C. A. Herter and J. S. Thacher, who could find no explanation of the paralysis. Nothing abnormal was found except "a slight increase of small spheroidal cells about some of the meningeal and cortical vessels of the motor area. The frontal and occipital lobes were normal."

Symptoms.—While diplegia and paraplegia are occasionally seen, the great majority of cases of acquired cerebral palsy are of the hemiplegic variety. When diplegia and paraplegia occur, it is usually in early infancy, and their symptoms and course differ in no wise from the birth palsies. We may therefore regard hemiplegia as the chief manifestation of acquired cerebral palsy.

The onset of the paralysis is almost invariably sudden, with convulsions, which are usually repeated, and in severe cases followed by loss of consciousness. In the secondary cases these are generally the only symptoms. In one of my cases the patient went to bed apparently well, and awoke in the morning with hemiplegia. Such an onset, however, is very exceptional. When the paralysis is apparently primary, fever is usually present, and in addition to the convulsions there may be vomiting, delirium, and other symptoms, strongly suggestive of an acute inflammatory process in the brain, which continue for a variable time, usually from one to three days, before paralysis is seen. The temperature in most cases is from 100° to 103° F., and the rise of temperature sometimes follows, sometimes precedes, the convulsions. After the child recovers consciousness, and sometimes before this, the paralysis is discovered. If there is a very extensive lesion there may be diplegia, deep coma, and death, but this is very infrequent. Usually the lesion is more limited, and the symptoms are those of typical hemiplegia. When the face is involved, it soon recovers, and often it escapes altogether. The paralysis of the arm and leg is at first complete, but may improve very rapidly in the course of a few days. Disturbances of sensation are usually of a transient character. After a variable period, from one to several weeks, the patient begins to use the paralyzed extremities, first the leg, afterward the arm, as in adult hemiplegia. The convulsions may be repeated for the first day or two, but prolonged or continuous convulsions are rare. With lesions of the left side of the brain, speech may be affected, and not infrequently in young children when the lesion is upon the right side. The reflexes are increased upon the affected side, and a slight ankle-clonus may be present.

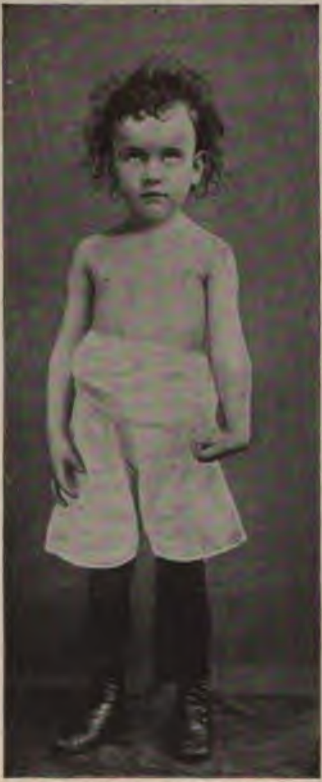

FIG. 142.—Deformity of left hand the result of contractures following an attack of hemiplegia four years before; child seven years old.

In the course of a few weeks the child may be able to walk, dragging the affected leg; the recovery in the leg is sometimes complete, but in most cases a slight halt in the gait remains. The arm usually recovers more slowly than the leg, and contractures are likely to develop after a variable time, generally two or three years. In Fig. 142 is shown a frequent deformity of the upper extremity. Contractures of the leg lead to various forms of talipes, generally equinus, from shortening of the tendo-Achillis. Sometimes the arm or the leg recovers so perfectly that the case may be regarded as one of monoplegia. In old cases the paralyzed limbs are atrophied; there is more or less rigidity, and the spastic condition may be quite marked. I have seen this limited to a single group of muscles in the leg. Aphasia is common in right hemiplegias, and it is not very rare in those of the left side, because infants appear to use both sides of the brain with nearly equal facility.

The mental condition of these children is often normal, in striking contrast with the cases of congenital diplegia. The earlier the paralysis occurs the more likely are mental symptoms to be present, since we have here not only the direct effect of the lesion, but an arrested development of some part of the brain. Epilepsy is not an uncommon sequel; it may be of the Jacksonian type, or there may be attacks of general convulsions. In other cases there are post-hemiplegic movements of a choreic or athetoid character, or irregular incoordinate movements.

Prognosis of Infantile Cerebral Paralysis.—In diplegia and paraplegia the outlook is always unfavourable. A very large number of these cases which are due either to intra-uterine or birth lesions, never reach the third year, but die in infancy from marasmus or acute intercurrent disease. Those who survive usually show serious mental defects, and many are practically helpless on account of the extreme spastic condition of the muscles of the extremities.

In hemiplegia the prognosis is much more favourable. In most of these cases the paralysis is of the acute acquired variety, and the later the period of onset, the less likely is the brain to be seriously damaged. In some of these patients complete recovery takes place; in others the residual paralysis is so slight as to be easily overlooked except on careful examination, the occurrence of epilepsy being perhaps the first thing which leads one to suspect that a previous paralysis has existed. The great majority of children who have suffered from infantile cerebral palsy have some degree of permanent paralysis and usually some deformities from contractures, the extent of both varying, of course, with the severity of the primary lesion. In all cases seen in young infants it is exceedingly difficult to give a prognosis in regard to future mental development. As a rule, the impairment is directly proportionate to the extent of the paralysis and its intensity; although in exceptional cases we find a good deal of mental disturbance with only moderate paralysis, and *vice versa.*

Diagnosis.—The diagnosis between the congenital and acquired forms of cerebral palsy is of no great practical importance, and it may be impossible; for the symptoms in congenital cases are often not sufficiently marked to attract attention until children are old enough to sit alone or to walk.

It may be quite difficult to distinguish cerebral paralysis from infantile spinal paralysis. The history of an acute onset, the atrophied limbs, the deformities, and the absence of sensory disturbances, may be found in both conditions. Spinal paralysis is, as a rule, monoplegic, and often affects but a single group of muscles. Cerebral paralysis is either diplegic or hemiplegic in character, and even though only a leg or an arm may seem to be affected, a critical examination will usually reveal the fact that the other limb of the same side has also suffered. The presence of rigidity and exaggerated reflexes is quite as important evidence of this as loss of power. The electrical reactions, however, are conclusive; the reaction of degeneration is absent in cerebral paralysis, while it is present in spinal paralysis.

Simple as the differentiation may seem in most cases, the mistake is frequently made of confounding cerebral diplegia, particularly of the flaccid type, with rickets. But a careful history and a thorough examination will usually dispel all doubt (see article on Rickets). Cases of acute acquired paralysis at the onset may be mistaken for acute meningitis, but early loss of consciousness, the early development of the paralysis, its permanent character, and the short duration of the acute symptoms, distinguish cases of hæmorrhage from those of meningitis;

but when it follows traumatism, and when it occurs in the course of some other disease such as pneumonia or scarlet fever, it may be difficult or impossible to make a diagnosis between the two conditions.

Treatment.—The course and the result of cerebral paralysis depend upon the extent of the injury to the brain, its nature, and the age at which it is inflicted,—all these being conditions which are beyond the power of the physician to modify or control. The treatment of cerebral palsy is therefore extremely unsatisfactory. For the congenital cases practically nothing can be done, except for the deformities and complications. The acquired cases during the acute onset are to be managed like all other cases of acute cerebral congestion or inflammation,—absolute rest, ice to the head, and bromides. Electricity is never to be used in early cases, and little or nothing is to be expected from it in the late ones. Much can be accomplished in an educational way for the mental derangements resulting from cerebral palsy. An important part of the treatment relates to the deformities. Many of these may be prevented by the early use of orthopædic apparatus. Serious deformities in old cases may be greatly benefited by tenotomy or myotomy, followed by the use of suitable apparatus. Epilepsy is to be treated as when it depends on other causes.

MENTAL DEFECTS.

DEFICIENCY, IDIOCY, IMBECILITY.

All grades of mental defects are seen in children. While the terms above used characterise the chief clinical types, it should be remembered that these shade into each other by almost imperceptible degrees. They may be the result either of arrested development or of disease or injury of the brain.

The backward child does not belong in this group, although often placed here by parents or teachers. Such children may present many mental peculiarities, but differ from the normal standard chiefly in the slowness with which the mental functions are developed, the most noticeable of these being speech. It is backward children and those who present the milder grades of mental defect that are of the greatest clinical interest and importance, for in them the mental condition often depends upon some physical cause which time and proper treatment may remove. Common causes are defective sight, or hearing, severe early rickets, prolonged malnutrition, etc.

Following somewhat the classification of Ireland, the mental defects of children may be divided into the following groups:

1. Those depending upon such congenital conditions as porencephalus, arrested development of the brain as a whole, or of some portion, particularly the frontal lobes. An excellent illustration of this class of

cases is seen in Fig. 149. Another variety is known as "Agenesia corticalis (page 796).

2. Those associated with external or internal hydrocephalus.

3. Those associated with microcephalus, either with or without premature ossification of the cranial bones (Figs. 146–148).

4. The paralytic cases, including the varieties which occur in the different forms of cerebral paralysis, the greater part of which are due to meningeal hæmorrhage at birth, and which are clinically associated with spastic diplegia or paraplegia; a smaller number are associated with acquired cerebral paralysis, most frequently following meningeal hæmorrhage.

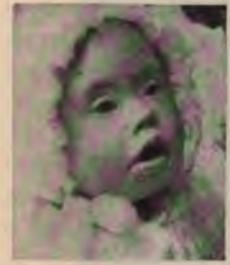

Fig. 143.

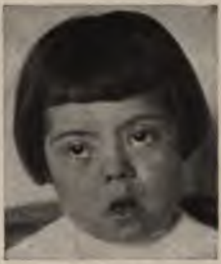

Fig. 144.

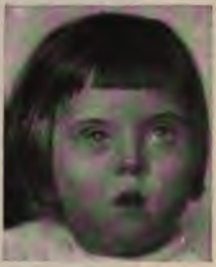

Fig. 145.

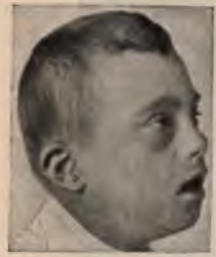

Fig. 146.

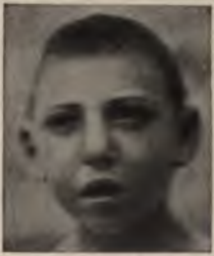

Fig. 147.

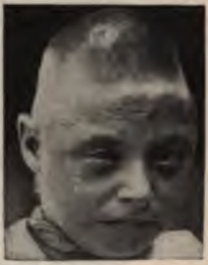

Fig. 148.

Various types of mental defects.

Figs. 143–145.—Mongolian type.

Fig. 143.—Six months old; died at twenty-two months; could not hold up the head, or understand anything.

Fig. 144.—Boy six and a half years old; did not walk or talk till four years old; now quite intelligent, almost normal.

Fig. 145.—Girl four years old; mental development like that of a normal child of two and a half years; walks very awkwardly.

Fig. 146.—Boy twelve years old; microcephalic; walked at about four years; can read and write; development like that of a normal child of eight years.

Fig. 147.—Microcephalic, seven years old; understands most of what is said; cannot talk intelligibly.

Fig. 148.—Girl of eight years; imbecile; cannot walk without help.

Note that the expression in 144, 145, and 146 is not due to adenoids; 144 and 146 have had them removed.

5. Those of inflammatory origin. They follow cerebro-spinal meningitis and acute poliencephalitis.

6. Those associated with epilepsy, in which the condition is a result of changes in the brain produced by the repetition of the epileptic seizures.

7. Mongolian idiocy.—This is a form characterised by a peculiar Chinese type of skull and face, with marked backwardness of physical and mental development (Figs. 143–145). The head is somewhat flattened from before backward; the nose rather broad and flat; but the

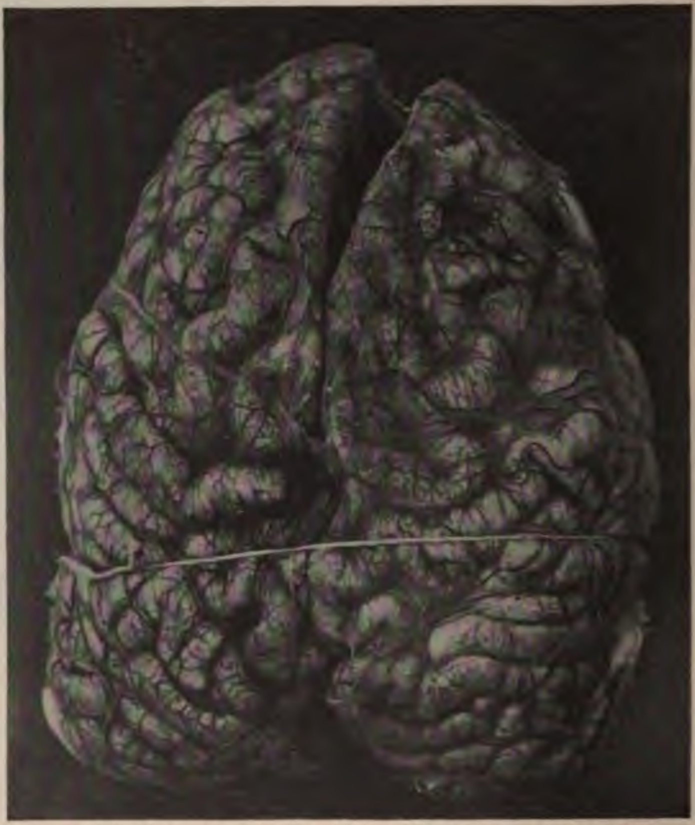

FIG. 149.—Arrested development of the frontal lobes of the brain, particularly of the right side from an idiotic child twelve months old.*

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* A microscopical examination by Dr. Martha Wollstein showed the cortex in the affected region to be only one-third the normal thickness; the cortical layers were ill-defined; there was a striking absence of the characteristic nerve cells, both the large and small pyramidal cells being few in number. There was no growth of connective tissue. The white substance was normal, as were also the dura and pia.

most striking thing is the narrow palpebral fissures which have a downward inclination toward the nose. These patients almost always have the mouth open; and the facial expression like that due to large adenoids may lead to the suspicion that this is the only condition present. The mouth breathing is, however, due rather to the peculiar conformation at the base of the skull, and the anterior projection of the bodies of the upper cervical vertebræ. The Mongolian type is seen in all degrees of severity. In early infancy these children may present no striking peculiarities except in facial expression, and a general backwardness of physical development. Dentition is delayed; they may not sit alone until the age of eighteen months or two years, and frequently do not walk or talk intelligently until they are four or five years old. In the milder forms they are often regarded simply as very backward children. In the more severe forms the mental defect may be great. Their resistance is feeble and many die in early childhood. Little is known of the etiology of this condition. Cases occur in all classes of society, and when other children in the family are quite normal.

8. Amaurotic family idiocy. This name, proposed by Sachs,* indicates the prominent features of the malady, which is not a very rare one. The first symptoms are usually noticed between the third and sixth months in apparently healthy infants. It is then discovered that the infant, who before this has seemed to see well, no longer notices objects; the expression becomes stupid; the infant does not hold up its head and never learns to sit. There is relaxation of the voluntary muscles, especially those of the trunk. The characteristic features of the disease are revealed by the ophthalmoscope. There is a milky blue or white area, with bright cherry-red centre, occupying the place of the macula lutea, and atrophy of the optic disc. The ocular changes are symmetrical. The voluntary muscles show more or less the reaction of degeneration. The disease is progressive, and usually fatal within a year; but occasionally the blind, helpless child may live for two or even six years. Whether the disease is a developmental degeneration or an inflammation is not yet determined. The brain shows defective development, with degeneration and chromatolysis of the nerve cells, sclerosis, and thickening of the membranes. Nearly all of the reported cases have been in Hebrews. The prognosis is at present hopeless.

9. Both sporadic cretinism and chondro-dystrophy have many symptoms suggesting mental defects, but they do not strictly belong in this category. They are considered separately later.

In addition to the etiological factors belonging to the different conditions above described, the influence of heredity is to be considered; there may be hereditary nervous diseases, alcoholism, syphilis, or some other vice of constitution. Intermarriage among blood relations is one of the causes most frequently assigned; but after an exhaustive study of the question, Huth reaches the conclusion that this view is not supported by the facts.

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* New York Medical Journal, July, 1896; also Keating's Cyclo., Supplement, 1899.

Diagnosis.—Certain types of mental defect may easily be recognised after the age of three or four years, especially the more marked forms where they are due to the graver cerebral lesions,—hydrocephalus, microcephalus, various cerebral palsies, amaurotic idiocy, etc. In the milder forms and in infancy, however, this is not so easy a matter; it is often impossible without a considerable period of observation to distinguish a backward or peculiar child from one who has some serious mental defect.

To appreciate the abnormal, one must be familiar with the mental and physical development of healthy children. A normal infant of average muscular development can usually support the head steadily before five months old, often at three months; it can usually sit erect at seven or eight months, and stand with assistance at twelve or thirteen months. Toys are held and usually handled with facility at five or six months. The recognition of the nurse or mother comes at about the same time. Usually the first distinct words are pronounced about the end of the first year, and at two years most children put words together in short sentences. Variations of a few months from the averages above mentioned can not be considered abnormal.

To determine whether an abnormal mental state is simply the result of poor general nutrition, or is dependent upon actual disease or imperfect development of the brain, is frequently a matter of the greatest difficulty. The backward infant is usually distinguished chiefly by the things which he does not do; while with those who are deficient not only are the proper signs of development wanting, but many new and peculiar symptoms may be observed. The backward child may not sit alone until he is twelve or fifteen months old, and may not walk until he is two and a half years old, but the cerebral development is in most cases proportionate to the physical condition. Speech may be so delayed that the first words do not come until two years, and short sentences not until three years old, and yet in understanding what is said to and done for him, the child may seem bright and his development steady and progressive, although slow.

All children whose development is delayed should be examined for local signs of cerebral disease; the symptoms mentioned under the various heads of early hydrocephalus, meningeal hæmorrhage, and cretinism should be sought. Sight and hearing should be tested, and the eyes, if possible, examined with an ophthalmoscope; the co-ordination of the hands should be tested in various ways; the reflexes examined, and general rigidity or slight paralysis noted, also the muscular power in the trunk, neck, and extremities. Many children who are mentally deficient do not show any disturbances of nutrition during the first year. The growth of the body in height and weight may be quite normal; although this is rarely true of the muscular power. Some of them show marked signs of backwardness in physical development, and in nearly all there are some other striking symptoms. Among the most frequently noticed are drooling, an open mouth, a protruding tongue, a fixed aimless stare, the production of some inarticulate sounds, which are usually peculiar to the child and may be repeated many times a day. Occasionally there are sharp screams without any evident cause, also irregular aimless movements of the hands. Objects are not properly held, and if grasped, they are soon dropped by an infant of twelve or fourteen months as by a normal one of three or four months. The child does not recognise its bottle or its nurse. Nystagmus is often present; and there may be ill-defined attacks of a convulsive nature, or typical convulsions. The infant is not attracted by bright colours or toys, and, in short, seems dull and unresponsive to every mental impression.

An accurate diagnosis usually carries with it the data for a definite prognosis. Few misfortunes which can befall a family are worse than to have a mentally defective child, and the physician's opinion is sought early and eagerly as to the probable outlook for all children who are suspected to be in any way abnormal. The possibilities of error in the early years are great, and much needless suffering is often caused to parents by an erroneous opinion. It is the experience of all who see many of these children, that some who were regarded at the age of three or four years as seriously defective, have in the end turned out to be entirely normal. One should therefore always put the best possible interpretation upon the facts. The amount of improvement which takes place in many of these cases is most surprising. The above statement applies of course chiefly to children in whom there are no evidences of gross cerebral lesions. The deviations from what is normal are many and wide, and careful observation for a long period is necessary before a child is pronounced idiotic or even feeble-minded.

Most cases of idiocy exhibit to a greater or less degree the stigmata of degeneration. In an examination of 517 idiots by Howe, there was found blindness in 21; deafness in 12; some defect of the nose or mouth, such as hare-lip, high palatal arch, or cleft palate, in 23 cases; and some deformity of the hands or feet in 54 cases; while in 96 there was paralysis of one or more limbs.

Treatment.—The problem is essentially an educational one, and for such education special teachers and often special schools are indispensable. With such advantages it is surprising to see what can be accomplished with many children who have a severe grade of mental defect. To furnish a proper means for educating these children is a duty of the State, and up to the present time very inadequate provision has been made for them. Except in the mild forms, defective children are better trained and educated in institutions than in the home, and parents should be urged to place them in institutions wherever practicable as soon as they have passed the age or development of infancy.

CHONDRO-DYSTROPHY.

Synonyms: Achondroplasia—Congenital or fœtal rickets.

This rather rare condition is the cause of some of the most marked examples of dwarfism known. It was recognised as an abnormality by the early Egyptians and has figured in art in various ways since that date. Paintings show that many of the old court jesters were of this type. Because of their striking appearance, these dwarfs have always excited much curiosity and interest.

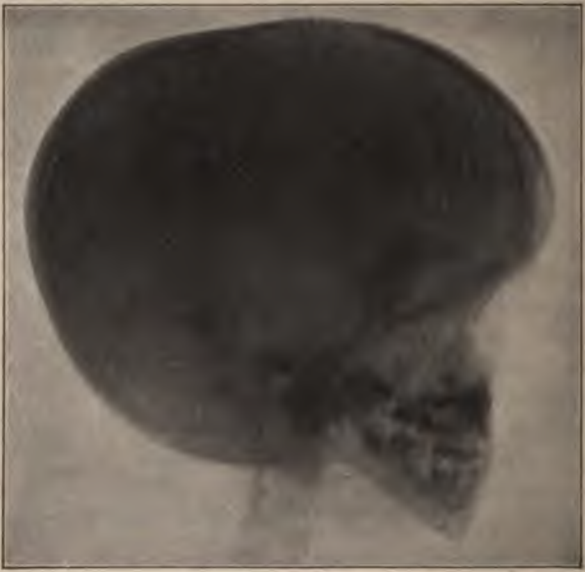

FIG. 150.—Skull in chondro-dystrophy, showing frontal prominence and prognathism. Girl six years old.

The causes of chondro-dystrophy are unknown; only in rare cases has any hereditary connection been traced. The pathological process begins in fœtal life and consists in a disturbance of the normal ossification of primary cartilage. It affects endochondral ossification only, never intra-membranous ossification. The flat bones and the vertebræ therefore escape while the bones of the extremities suffer most. The disease does not affect bones which are cartilaginous or almost entirely so through the greater part of intra-uterine life. One of the most striking changes in the skull is the synostosis or early ossification of the tribasilar bone; this is formed of two parts of the sphenoid and the sphenoidal process of the occipital bone. Normally this ossification does not take place until adult life; in children with chondro-dystrophy it often begins *in utero*. This prevents a normal expansion at the base of the skull, and the brain as it grows is thus crowded upward and forward causing the great prominence of the forehead (Fig. 150). The upper jaw appears very prominent on account of the depression at the root of the nose.

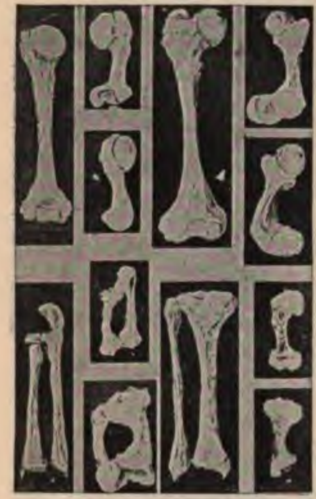

FIG. 151.—Normally developed long bones of a fœtus compared with those of chondro-dystrophy. (Spillmann.)

In the long bones, there is a marked interference with the normal row-formation of the proliferating cartilage cells, which may be seen in all degrees. In some cases a periosteal lamella pushes its way between the epiphysis and the diaphysis, still further restricting the growth of the long bones. As bone formation beneath the periosteum goes on normally, the bones in this condition are thick as well as short.

Symptoms.—The majority of children suffering from this condition are either born dead or die shortly after birth. Those who survive are delicate during infancy, but afterward may become strong and healthy. The most striking thing about their appearance is the very short legs and arms as compared with the length of the body. At birth the arms in many cases do not reach to the waist line, and the length of the body may be less than the circumference of the head. The epiphyses appear somewhat enlarged, the abdomen is prominent, the skin of the extremities is in deep folds, the soft parts seeming to be much too abundant for the shortened bones (Fig. 152). In infancy these children are often quite fat. The facial expression is characteristic. There is usually a deep depression and flattening at the base of the nose with a very marked prominence of the forehead. The head may not only seem large, but by measurement may be one or even two inches

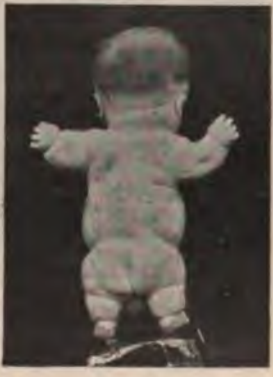

FIG. 152.—Chondro-dystrophy —infantile figure. (Marie.)

above the normal average. An erroneous diagnosis of hydrocephalus is often made in the early stage. Dentition is slightly later than normal, but not more so than is seen in moderate rickets. Marked relaxation of the ligaments and rather feeble muscular power often delay walking until the third or fourth year. If the head is large the fontanel may not close till the fourth or fifth year. The appearance of the fingers is quite characteristic, causing the so-called "trident hand." The fingers are very short and of nearly equal length and an angular separation is seen at the second joint (Fig. 153).

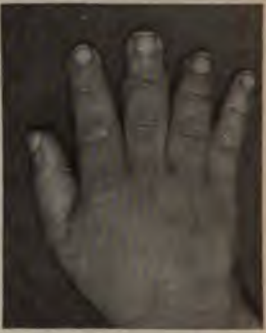

FIG. 153.—Characteristic hand of chondro-dystrophy. (Marie.)

Although not normal in their mental development, these children are far from being feeble-minded. They are often several years behind the normal in speech and in most intellectual efforts. The average patient is able to read and do many ordinary things, but throughout life always remains somewhat peculiar, and on critical examination is found to be subnormal in his mental growth. These dwarfs are good-natured, often amusing, easily controlled, and frequently live to a great age. With advancing years the figure assumes a very peculiar and characteristic appearance. The prominent hips with the marked lordosis, shortened extremities, and late bowing of the legs, present a striking picture (Fig. 154). The maximum height attained is often not more than three and a half or four feet. Although while young of feeble muscular power, later in life they often become very muscular. When adult life is reached the sexual powers are normal; if the women become pregnant, Cæsarian section is almost always required on account of deformity of the pelvis.

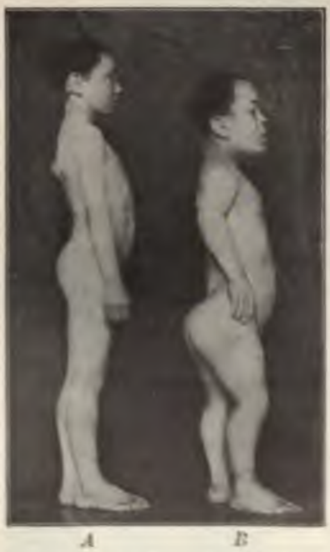

FIG. 154.—*A*. Normally developed boy, age eight years. *B*. Typical chondro-dystrophy, age eighteen years. (Marie.)

In infancy, chondro-dystrophy is often confounded with rickets, hydrocephalus and cretinism; but its features are so characteristic that the mistake can hardly be made if the child is carefully examined. No known treatment has any influence upon the condition. The use of the thyroid extract is entirely without effect.

SPORADIC CRETINISM.

Synonyms: Cretinoid idiocy, myxœdematous idiocy, idiocy with pachydermatous cachexia.

Since the early description of this disease by Fagge, in 1871 and 1874, numerous cases have been published in England, on the continent of Europe, and in America, showing that sporadic cretinism is not confined to any country. While the condition is relatively a rare one, since it has been generally recognised it is found to be much more common than was formerly supposed.

Etiology.—It is now well established that this condition depends upon the absence of the internal secretion of the thyroid gland. In a series of sixteen autopsies collected by Fletcher Beach, the thyroid gland was absent in fourteen and the seat of bronchocele in two. The symptoms closely resemble the myxœdema of adults which follows the removal of the thyroid. Regarding the causes which destroy the thyroid gland or abolish its functions little is as yet known. In most cases it is probably a congenital condition. In some instances it has followed acute disease. In a certain number of cases sporadic cretinism is associated with goître. As a rule, only one case occurs in a family, the other members of which present nothing abnormal in mental or physical development.

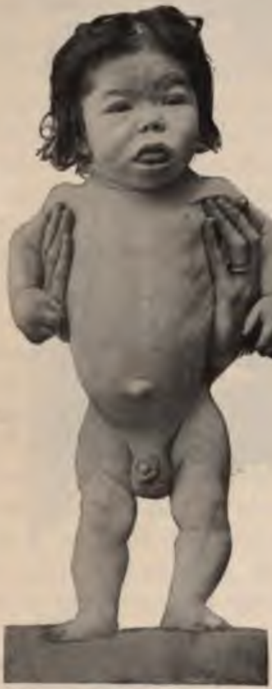
FIG. 155.—A typical cretin; two and a half years old; a patient in the Babies' Hospital.

Symptoms.—The symptoms of cretinism in most cases make their appearance during the first year, but are sometimes so slight as not to be noticed until children are two or three years old, and exceptionally not until the seventh or eighth year. The general appearance of the cretin is striking, and so characteristic than when once seen the disease can hardly fail to be recognised (Figs. 155 and 156). The body is greatly dwarfed, and children of fifteen years are often only two and a half or three feet in height. All

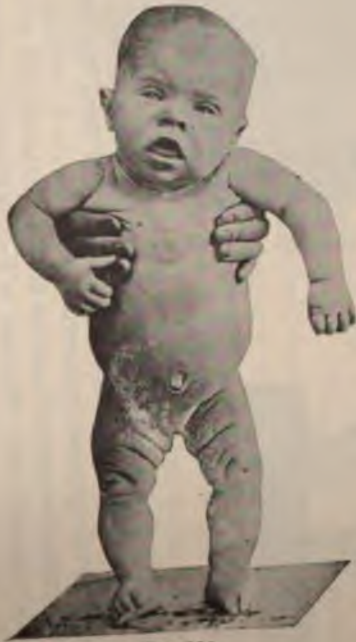

Fig. 156.

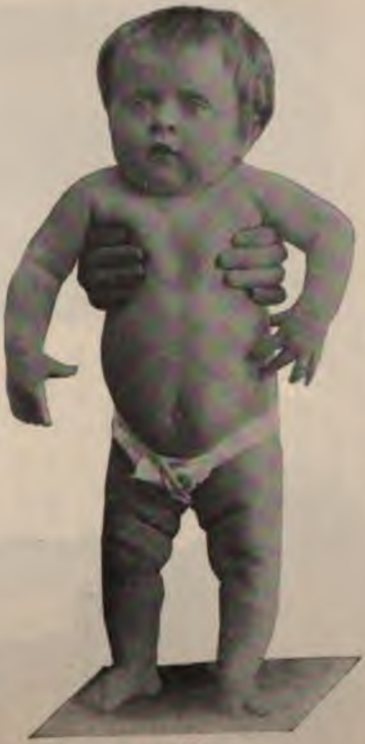

Fig. 157.

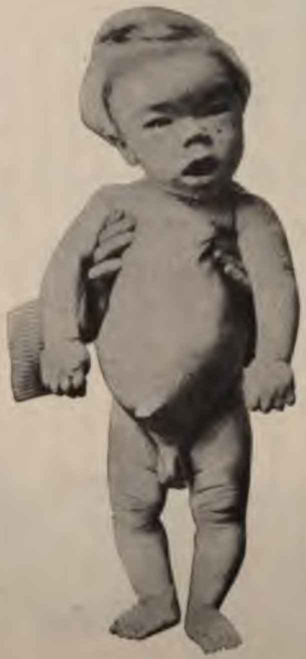

Fig. 158.

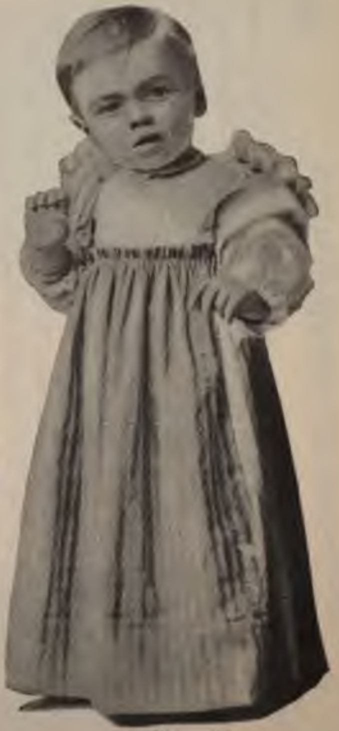

Fig. 159.

Fig. 156, Dr. J P. West's case of cretinism, seventeen months old, before treatment. Fig. 157, after six months' treatment with thyroid extract. Fig. 158, Dr. J. W. Coyner's case, twenty-three months old, before treatment. Fig. 159, after eleven months' treatment with thyroid extract.

the extremities, the fingers and the toes, are short and thick. The subcutaneous tissue seems very thick and boggy, but does not pit upon pressure like ordinary œdema. The facies is extremely characteristic: The head seems large for the body; the fontanel is open until the eighth or tenth year, and it may not be closed even in adults; the forehead is low and the base of the nose is broad, so that the eyes are wide apart; the lips are thick, the mouth half open, and the tongue usually protrudes slightly; the cheeks are baggy, the hair coarse, straight, and generally light coloured. The teeth appear very late—in one of my cases none were present at two years—and are apt to decay early.

Fatty tumours are quite constant in older children, although they are often wanting in infantile cases. They are seen in the supra-clavicular region, just behind the sterno-mastoid muscle, sometimes in the axilla, or between the scapulæ, and sometimes in other parts of the body. In distribution they are apt to be symmetrical, and are usually about half the size of a hen's egg. The neck is short and thick. In rare cases there may be a slight depression corresponding to the location of the thyroid gland. The chest is not deformed. The abdomen is large, pendulous, and resembles that of rickets. The skin is dry, perspiration scanty, and eczema is common. The voice is hoarse and rough. Patients often do not walk until they are five or six years old, and then they waddle in a clumsy way. All the movements of the body are slow and lethargic, and everything indicates mental and physical torpor. The rectal temperature is usually subnormal. I had once an opportunity to observe an attack of acute broncho-pneumonia in one of these cretins two years old. The symptoms and physical signs were typical, but during the greater part of the disease the rectal temperature fluctuated between 95° and 98.5° F. Only once was a temperature above 99° F. recorded. On account of their low temperature and torpid condition these patients are very sensitive to cold. The mental condition is always impaired, and they are often idiotic. Speech is acquired late, and in some cases not at all. Cretins are dull, placid, and good-natured, rarely troublesome or excitable; and when fifteen or eighteen years old they appear like children of two or three years. There is an absence of development of the sexual organs, and almost invariably they suffer from chronic constipation.

Diagnosis.—The diagnosis is usually easy, although the early cases are sometimes miscalled rickets. The low temperature, the facial expression, the torpor, and the fatty tumours are enough to differentiate the two diseases.

Prognosis and Treatment.—There is no tendency to spontaneous improvement. Many of these cases die in childhood, but a few live to adult life. Until within the last few years they have been considered hopeless. The improvement which followed the use of the thyroid extract in cases of adult myxœdema led to a trial of this remedy in sporadic cretinism. A sufficient number of cases have now been recorded to establish the fact that the thyroid extract is a specific remedy for this disease. In many cases the improvement is truly remarkable (Figs. 156–159). After a few months' treatment the entire appearance of the child is in most cases changed. The idiotic expression of the features is lost; the thickening of the skin and subcutaneous tissues disappears; there is a marked increase in height, and in the circumference of the head; muscular power is rapidly developed, so that many soon become able to walk; and progress is seen in dentition, and in some older girls in the establishment of menstruation. Intellectual progress is much slower than physical changes; however, nearly all the children become brighter and more intelligent, and most of them learn to talk.

The ultimate results vary with the grade of the affection and the time when treatment is begun. I have under observation several cretins who have been treated from five to eight years. Although many of these children are very intelligent and able to attend school, they are without exception somewhat below other children of their ages in mental and physical development. There seems to be no reason why complete recovery might not occur if the thyroid were begun in early infancy and faithfully continued.

If the thyroid is omitted relapses occur in a few months, even in cases well advanced toward recovery.

The preparation most used in America is Parke, Davis & Co.'s desiccated extract, prepared from the thyroid gland of the sheep. Of this half a grain may be given once or twice a day at first; after the child becomes somewhat accustomed to the drug the daily dose may be gradually increased to four or five grains. Some disturbances are often seen at the beginning of the treatment—perspiration, marked irritability, and sometimes a rise in temperature—but these soon pass off.*

INSANITY.

Insanity is so special a subject, that all that will be attempted here will be to mention the most frequent varieties seen in early life, with the important etiological factors which operate at this period. For a full discussion of the subject the reader is referred to works upon insanity, and to Sachs, in whose book † will be found quite a full bibliography of this aspect of the subject.

Insanity is distinguished from idiocy in that it affects a mind previously sound; however, the two conditions may be associated. Undoubted cases of mental disease have been observed before the seventh year, but they are extremely rare. From this time up to puberty, however, nearly all the varieties seen in adult life occasionally occur, but they are very infrequent even at this period. The form which insanity in childhood most frequently assumes is mania.

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* See Osler, American Journal of the Medical Sciences, 1897, cxiv, No. 4, and Bramwell's Monograph on Cretinism.

† Nervous Diseases of Children, New York, 1895. See also Mills, in American Text-Book of Diseases of Children, Philadelphia, 1898.

Etiology.—Insanity is sometimes seen as a sequel of one of the infectious diseases, more often typhoid fever than any other, although it may follow measles, scarlet fever, diphtheria, or variola. Another cause is masturbation, although its effect is much more frequently seen after puberty than before. Hereditary syphilis is sometimes the cause of dementia, which comes on about the fourth or fifth year, or even later. Alcoholism, epilepsy, insanity, or other nervous diseases in the parents are important causes. Prolonged or continuous mental strain, the result of overwork in school, is a cause of considerable importance, especially in girls about the time of puberty. As exciting causes may also be mentioned various reflex conditions, such as intestinal worms, phimosis, delay in the establishment of menstruation, and abnormal conditions of the nose and throat; these, however, can not have much influence except where the predisposition is a strong one. Insanity may be associated with or may follow hysteria, chorea, or epilepsy. It has sometimes followed injury to the brain, acute meningitis, and occasionally other forms of brain disease.

Symptoms.—Certain forms of insanity are practically never seen in children, such as paranoia or primary delusional insanity, acute dementia, paretic dementia, periodic or circular insanity, and cataleptic insanity.

Mania is one of the most frequent forms, and is the most common variety of post-febrile insanity. Its symptoms may be quite intense, but are usually of short duration, lasting but a few days or weeks. In rare cases it may continue for months, and it may even be permanent.

Melancholia is not uncommon. It is seen as a result of prolonged mental strain in school, it may be due to fear of punishment, and sometimes may follow masturbation. It is usually associated with some very marked disturbance of the general health. It shows itself, as in the adult, by fits of depression, self-mutilation, and even by suicidal tendencies.

Epileptic insanity may follow epilepsy in children who were previously mentally sound, where it may take the form of true epileptic dementia, or there may be attacks of mania which occur in the place of an epileptic seizure or follow such a seizure. Transitory attacks of fury or frenzy coming on without apparent cause should always suggest the possibility of epilepsy.

Other forms which insanity assumes in early life are: transitory psychoses, such as delirium, night-terrors, attacks of sobbing or weeping, sometimes from fright; moral insanity, as shown by perversion of the moral sense from injury or disease, and by various vicious tendencies; morbid impulses, which may be homicidal or sexual, or a disposition to thieving, lying, pyromania, etc.; morbid fears, of which there may be an almost endless variety. These are sometimes associated with a low state of physical health; this, however, is usually not the case.

Prognosis.—On the whole, insanity in childhood has a better prognosis than in the adult. In most of the cases of mania, melancholia, the various transitory psychoses, or the choreic and hysterical forms, recovery occurs with proper treatment. The outlook for the other varieties is much worse, especially in those in which there is a strong hereditary tendency to mental disease.

The treatment is to be conducted along the same general lines as in adults.

THE STIGMATA OF DEGENERATION.

These marks are of much importance in relation to the different forms of nervous disease in children, especially epilepsy, idiocy, and insanity. They are of great value in determining existing nervous disease, or as showing latent neuropathic tendencies.

The physician should be familiar with these various signs in order that he may connect them with each other and refer them to their proper source, and at the same time, by appreciating their significance, be able to advise parents with regard to the care, education, mode of life, and occupation of children, in whom to a greater or less degree these signs may be present. These stigmata are not of equal importance as marks of degeneration. Some of them, such as facial asymmetry and most of the deformities of the palate, are always to be so regarded; the speech defects are often so, while many of the others may or may not be, according to their association. The stigmata are divided into anatomical, physiological, and psychical. The following is the classification given by Peterson:*

Anatomical Stigmata.—Cranial anomalies: Facial asymmetry; deformities of the palate; anomalies of the teeth, tongue, lips, or nose.

Anomalies of the eye: Flecks on the iris; strabismus; chromatic asymmetry of the iris; narrow palpebral fissure; albinism; congenital cataract; pigmentary retinitis.

Anomalies of the ear.

Anomalies of the limbs: Polydactyly; syndactyly; ectrodactyly; symelus; phocomelus; excessive length of the arms.

Anomalies of the trunk: Herniæ; malformation of the breasts and thorax; dwarfishness; giantism; infantilism; femininism; masculinism; spina bifida.

Anomalies of the genital organs.

Anomalies of the skin: Polysarcia; hypertrichosis; absence of hair; premature grayness.

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* Deformities of the Hard Palate in Degenerates, by Frederick Peterson, M. D., International Dental Journal, December, 1895.

Physiological Stigmata.—Anomalies of motor function: Walking late; tics; tremors; nystagmus; epilepsy.

Anomalies of sensory function: Deaf-mutism; neuralgia; migraine; hyperæsthesia; anæsthesia; blindness; myopia; hypermetropia; astigmatism; Daltonism; hemeralopia; concentric limitation of the visual field.

Anomalies of speech: Mutism; defective speech; stuttering; stammering.

Anomalies of genito-urinary function: Enuresis; sexual irritability; impotence; sterility.

Anomalies of the instinct or appetite: Merycism; uncontrollable appetites for food, liquor, drugs, etc.

Diminished resistance to external influences and diseases.

Retardation of puberty.

Psychical Stigmata.—Insanity; idiocy; imbecility; feeble-mindedness; eccentricity; moral delinquency; sexual perversion.

DEAF-MUTISM.

Excluding the cases in which idiocy is present, which are not considered in this chapter, deaf-mutism may be due either to congenital or acquired conditions; the larger proportion of the cases belong in the latter class. When congenital, deaf-mutism may result from ostitis, or periostitis of the temporal bone, encroaching upon the cavity of the middle ear, from ankylosis of the ossicles, from absence of the internal ear or any of its parts. There may also be colloid degeneration of the labyrinth. It may result from atrophy of the auditory nerve, and it may be due to a lesion of the brain. These congenital conditions are often hereditary. Acquired deaf-mutism is most frequently the result of scarlet fever, and is due to otitis. The second important cause is cerebro-spinal meningitis, where it may be due to a lesion of the brain, the auditory nerve, or the ear. It occasionally follows mumps, diphtheria, measles, and other infectious diseases. It may result from repeated attacks of acute otitis associated with adenoid growths or chronic rhino-pharyngitis.

The younger the child at the time the deafness occurs the sooner the power of speech is lost. In most of the infectious diseases, if the attack occurs before the fifth year speech is lost. According to Love,* total deafness is rare among deaf-mutes; hearing for speech is present to a useful degree in about twenty-five per cent of the cases, while hearing by cranial conduction exists in nearly all cases. Deaf-mutism should be suspected if a child not idiotic shows at the end of two years no signs of beginning to talk. A careful distinction should be made between deaf-mutism and idiocy resulting either from congenital conditions or acquired disease.

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* Deaf-Mutism, by James K. Love. Macmillan & Co., 1896.

It is necessary that this condition be recognised as early as possible, in order that the child may have the advantages of proper training during its early years. The physician should insist upon the child being sent to an institution where it may be taught to speak as early as the third, and certainly by the fourth year.

The treatment is mainly prophylactic. The most important relates to the care of the ears in scarlet fever, and the removal of adenoid vegetations of the pharynx and other causes which produce attacks of acute or chronic otitis. For the condition itself education is the only thing to be considered.

CHAPTER IV.

DISEASES OF THE SPINAL CORD.

MALFORMATIONS.

MALFORMATIONS of the cord are very frequently associated with those of the brain, and bear a certain degree of resemblance to them. (1) The cord may be absent (amyelia); this condition may exist alone or with absence of the brain. (2) The lack of development may be only partial (atelomyelia), as where some of the tracts are wanting. The most important one is defective development of the lateral tracts, which may be a cause of spastic paraplegia (Charcot). (3) There may be a malposition of some of the gray matter (heterotopia). (4) There may be a double cord (diplomyelia); the division is generally incomplete, and is attributed to an abnormal development of the central canal; it is usually associated with other deformities. All of these malformations are extremely rare and of very little practical interest.

There remains to be mentioned the only one which is really important—spina bifida.

Spina Bifida.—This is a malformation of the vertebral canal with a protrusion of some part of its contents in the form of a fluid tumour. The tumour is elastic, compressible, usually increased by crying, and sometimes by pressure upon the anterior fontanel. The contained fluid is clear serum, resembling in all respects the cerebro-spinal fluid. It is one of the most frequent congenital deformities.

According to Humphrey, spina bifida is due to an early failure in development,—in most cases before the cord is segmentated from the epiblastic layer from which it is developed. Hence it remains adherent to the epiblastic covering, and the structures which should be formed between the cord and the skin are undeveloped. For this reason we have in the wall of the sac a fusion of the elements of the cord, nerves, meninges, vertebral arches, muscles, and integument. If the error in development occurs later, the cord and nerves may be attached to the sac, but not intimately fused with it; in still other cases the cord does not enter the sac at all. The malformation may occur before the central canal is closed; or, if closed, it may reopen from the accumulation of fluid. It is probable that the accumulation of fluid first occurs, and that this prevents the union of the parts of the vertebral arches.

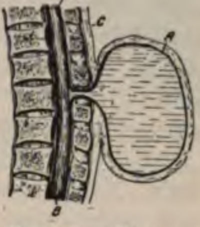

Fig. 160. — Meningocele (partially diagrammatic). *A*, the membranes; *B*, the spinal cord; *C*, the integument. The accumulation of fluid is behind the cord, which does not enter the sac.

Although the tumour is generally associated with a bifid spine, this is not necessarily the case. The protrusion may take place through the intervertebral notch or foramen, or there may be a fissure of the bodies of the vertebræ, and an anterior tumour projecting into the cavity of the thorax, abdomen, or pelvis,—spina bifida occulta. The principal anatomical varieties are meningocele, meningo-myelocele, and syringomyelocele.*

Meningocele.—In this form there is a protrusion of the membranes only (Fig. 160). The accumulation of fluid is either in the arachnoid cavity or the subarachnoid space posterior to the cord. The opening of communication between the tumour and the spinal canal is small in this variety, usually being about one twelfth to one sixth of an inch in diameter. There may, however, be no communication. The skin is usually fully developed (Fig. 161). The tumour is frequently globular, sometimes pedunculated, and may attain a very large size, being as much as five or six inches in diameter. This is because spontaneous rupture is not likely to occur, and the tumour does not become infected except by operative interference. With such tumours patients may live to adult life. This variety is most frequently seen in the cervical region. It has the best chance of natural recovery, and in it operation gives the best results.

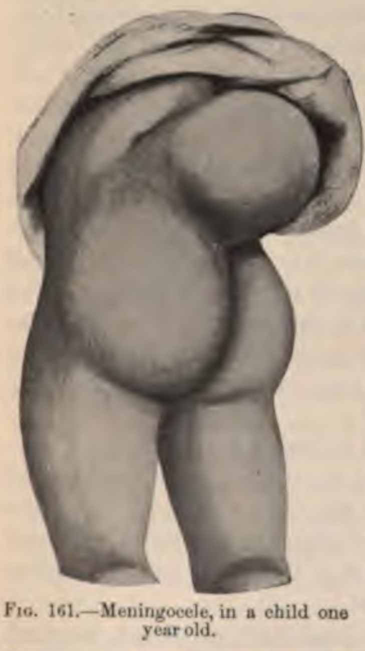

Fig. 161.—Meningocele, in a child one year old.

Meningo-myelocele.—This is by far the most frequent variety of spina bifida, occurring in thirty-five of the fifty-seven cases reported by Demme. It is the form usually seen in the sacro-lumbar region.

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* See Report of London Clinical Society, 1885: and Humphrey, Lancet, March 28, 1885.

The accumulation of fluid takes place in the anterior subarachnoid space, less frequently in the anterior arachnoid cavity (Fig. 162). In this form the cord is contained in the sac, and usually forms a part of its wall. The tumour is smaller than the meningocele, the usual size being that of a mandarin orange. It is sessile, never pedunculated. As a rule it is only partly covered by skin, but has a central area, elliptical in shape, where there is only a thin, translucent membrane. This surface, which is known as the central cicatrix, is sometimes covered with granulations, and frequently ulcerates. The tumour often has a vertical furrow or a central umbilication, corresponding to the attachment of the cord on its inner surface. The usual relation of the parts is for the cord to run horizontally across the upper part of the tumour to the central cicatrix, with which it becomes blended, and from which again the nerves arise. These re-enter the canal at the lower part of the tumour, and are distributed below as usual. In other cases the cord joins the wall of the sac soon after its entrance, and its attenuated fibres are found spread out all over the sac, coming together again below and entering the spinal canal.

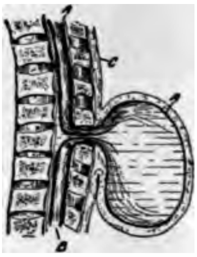

FIG. 162.—Meningo-myelocele (partially diagrammatic). *A*, the membranes; *B*, the cord; *C*, the integument. The accumulation of fluid is in front of the cord, the filaments of which are spread out, forming a part of the wall of the sac.

The following case, upon which I recently made an autopsy, is a good example of the common variety: The child died on the third day after birth from rupture of the sac. The tumour occupied the sacral region. The first sacral vertebra was normal, and beneath this the cord passed, terminating in the cauda equina soon after entering the sac, and continued back to the central cicatrix. Here nerve filaments blended with the other tissues in an indefinite structure, from which again, with tolerable distinctness, they could be seen to pass over the wall of the sac and return to the canal. The afferent and efferent nerves and the part of the membranes they carried with them formed several septa, making a smaller separate sac within the larger one. The large sac was clearly a dilatation of the anterior subarachnoid space, and communicated freely with the same space in the cord above.

Syringo-myelocele.—In this variety the accumulation of fluid is in the central canal of the cord, the lining of the sac being here the attenuated and atrophied cord elements. This is the rarest form of tumour, but the one most frequently associated with hydrocephalus, and consequently having the worst prognosis. It is usually found in the dorsal or dorso-lumbar region, rarely in the lumbo-sacral (Fig. 163).

With spina bifida other deformities are frequently associated, the most common being club-foot, hydrocephalus, more rarely encephalocele or cerebral meningocele, and hare-lip. If hydrocephalus exists, there is in most cases a dilatation of the central canal of the cord and a direct communication between the tumour and the lateral ventricles of the brain. Pressure upon the anterior fontanel causes an increase in the size of the tumour, and conversely. Club-foot is usually double, most frequently talipes equino-varus. In a number of cases there is a history of some deformity in other members of the family. I once saw two successive children in the same family with spina bifida.

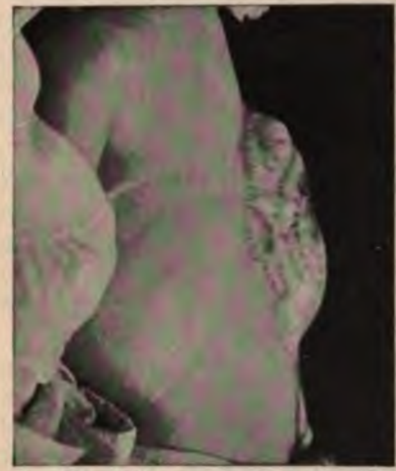
FIG. 163.—Syringo-myelocele of the mid-dorsal region, in a child four months old, who also had hydrocephalus.

Symptoms.—The tumour is present at birth, and is most frequently situated just above the sacrum. Paralysis is frequent in myelocele and syringo-myelocele, but is not seen in meningocele; its degree and its location depend upon the situation of the tumour and the extent to which the cord is involved. It is rare in cervical tumours, and most marked in those situated in the lumbo-sacral region. In the worst cases there is complete paraplegia, with paralysis of the bladder and rectum. If the tumour is sacro-lumbar or sacral, only the cauda equina is likely to be involved, and this but partially, so that the paralysis of the extremities is incomplete, and the bladder and rectum may escape.

In Fig. 164 is shown a very remarkable case of sacral spina bifida in a boy of five years, who came under observation for incontinence of fæces. The tumour was a little more to the left than to the right side, and had been overlooked. It had evidently pressed upon the lower branches of the sacral plexus, so as to involve the sphincter and the gluteal muscles of the left side. The atrophy was very marked, as shown in the illustration.

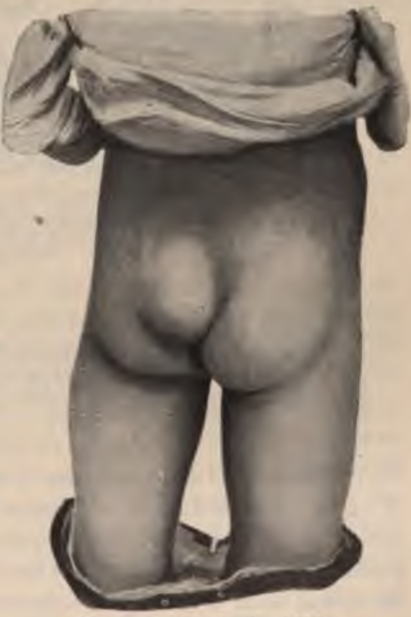
FIG. 164.—Sacral spina bifida.

The natural course of spina bifida

is to increase steadily in size; and if the tumour is covered by skin, its growth may be almost unlimited. It has been known to attain a circumference of twenty-two inches. If the integument is wanting, and the sac wall is very thin, rupture is pretty certain to take place, either spontaneously or by some accident, in the course of the first few months; death then results from convulsions owing to the rapid draining away of the cerebro-spinal fluid, or from secondary infection. In a large number of cases death is due to marasmus dependent upon the associated conditions. Infection of the tumour may take place without rupture, the germs passing through the wall of the sac. If the opening communicating with the spinal canal is small, this infection may excite an inflammation limited to the wall of the sac, and result in a cure of the spina bifida, usually with

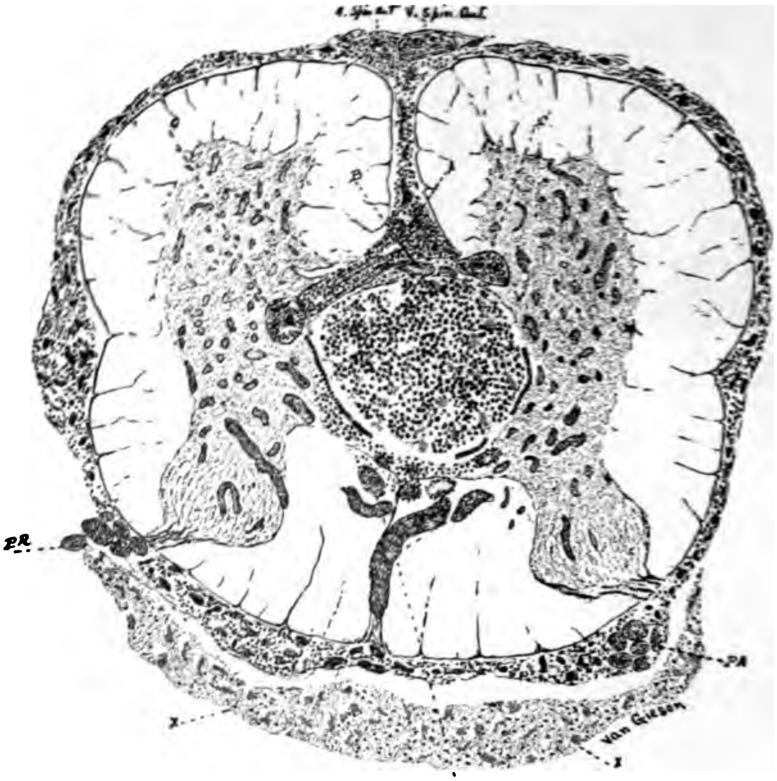

FIG. 165.—Spina bifida, with dilatation of the central canal of the cord, and spinal meningitis. The central canal is filled with round cells, among which are many cocci. *XX* is the pellicle of fibrin upon the posterior surface of the pia mater, also containing many cocci. The pia is everywhere infiltrated with cells, even to the bottom of the anterior fissure. The gray matter of the cord is much congested. *PR* is the posterior nerve root. The section is from the dorsal region of the cord.

sloughing. I have now under observation a girl ten years old in whom this occurred in infancy. The site of the former tumour is marked by a large dense cicatrix, and there still remains partial paralysis of the legs. If the opening into the spinal canal is large, inflammation of the sac is usually followed by spinal meningitis, which may extend upward and involve also the meninges of the brain. In a case published by Van Gieson and myself,* in which there was dilatation of the central canal of the cord and hydrocephalus, bacteria penetrated the wall of the sac and travelled up the central canal of the cord (Fig. 165), finally exciting a suppurative inflammation in the ventricles of the brain, in addition to a spinal meningitis. Sections of the wall of the sac and of the cord at various levels showed the same cocci. The child died at the age of three weeks.

Prognosis.—This depends chiefly upon the anatomical variety and the existence of complications. Simple meningocele, when covered by integument, gives the best prognosis, and complete recovery may occur. In meningo-myelocele, if complete paralysis exists, the prognosis is bad ; and if there is hydrocephalus, the case is hopeless. In quite a number of cases in which cure has followed operation, hydrocephalus has subsequently developed. Of fifty-seven cases reported by Demme, twenty-five were operated upon, with seven recoveries and fifteen deaths, while three were unimproved; of the thirty-two cases not operated upon, twenty-eight died within the first month, and not one lived over two years—the causes of death being marasmus, rupture of the sac, and meningitis.

Diagnosis.—It is usually easy to recognize spina bifida, but it is often difficult to distinguish between the different varieties. The absence of a palpable fissure in the spine, perfect translucency, and a pedunculated tumour, all point strongly to meningocele. Paralysis of the sphincters and lower extremities, umbilication of the centre of the tumour, a sessile tumour, a palpable bony fissure, and a large central cicatrix, point to meningo-myelocele. The coexistence of hydrocephalus points to syringo-myelocele.

Treatment.—In all cases the tumour should be protected from pressure, and care taken where it is not covered by integument, that the surface is kept absolutely clean and aseptic. It should be covered with some antiseptic powder and surrounded by a large pad of absorbent cotton, or a rubber ring-cushion. Complete paraplegia with involvement of the bladder and rectum, hydrocephalus, or extreme marasmus—all contra-indicate operative interference. If these are absent, operation should be considered. The time of operation will depend somewhat upon the nature of the tumour. If it is covered by integument and growing slowly, it is well to wait until the child is at least six months old. In other cases delay is dangerous, because of the liability to spontaneous or accidental rupture.

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* Journal of Nervous and Mental Diseases, December, 1890.

Nothing is to be expected from simple aspiration and compression. The methods of treatment which have been successfully employed are ligation, aspiration and injection, and excision of the sac. Ligation is admissible only where there is a pedunculated tumour; and even for these cases some surgeons prefer the clamp. The treatment by aspiration and injection has been widely used in Europe, but is not so highly esteemed in America. The tumour having been aspirated and about one half of its contents evacuated, there is injected, without removing the needle, a drachm of Morton's fluid (iodine, gr. x; iodide of potassium, gr. xxx; glycerin, ℥j). If the tumour is pedunculated, pressure should be made at its neck to prevent the entrance of fluid into the canal. In all cases the child should be kept in a recumbent position for several hours. The operation is not entirely free from danger, as in some cases it has been followed by convulsions and death in a few hours. Considerable inflammatory reaction usually occurs, lasting from two to four days. After this period there is, in a favourable case, a subsidence of the swelling, with a gradual contraction and finally obliteration of the tumour. The mortality of cases treated by this method is from forty to fifty per cent.* My own experience includes four cases, with two recoveries.

The dangers of this operation and the uncertainty as to its results have led many surgeons to discard it altogether in favour of excision, which with the technique of modern surgery is almost devoid of risk. For a description of this and the various plastic operations that have been proposed in connection with complete or partial excision of the sac, the reader is referred to works upon operative surgery. In operating, it should not be forgotten that in the great proportion of the cases (ninety-five per cent, according to the Clinical Society's Report, which, however, refers only to fatal cases) some part of the cord is in the sac. The cord is often present in tumours situated below the third lumbar vertebra, owing to its attachment to the sac.

Although recovery may follow operation, in a very large number of cases it is incomplete; some degree of paralysis, with atrophy, contractures, and deformities, remaining because of the implication of cord elements in the sac. In a considerable proportion of cases hydrocephalus subsequently develops, as after similar operations upon cerebral meningocele.

SPINAL MENINGITIS.

In acute meningitis usually only the pia mater is involved. This rarely occurs alone, unless it is due to traumatism. It is most frequently associated with inflammation of the pia of the brain, and may occur either with the simple or the tuberculous variety. A certain amount of acute inflammation of the pia mater accompanies most of the cases of acute myelitis.

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* Report of the London Clinical Society.

Chronic spinal meningitis in children usually involves the dura only. Inflammation of the external layer (external pachymeningitis) is usually secondary to caries of the vertebræ. This is considered in the article on Compression-Myelitis.

Symptoms.—The symptoms of inflammation of the spinal membranes, no matter with what pathological condition it may be associated, are due to irritation of, or pressure upon, the cord or nerve roots. Those which are most common are: pain in the back, which is increased by movement, and usually by pressure upon the spinous processes; radiating pains following the course of the spinal nerves, felt in the extremities or in the trunk; rigidity of the spinal column due to spasm of the spinal muscles, or rigidity of the muscles of the extremities; and hyperæsthesia along the spine, which may be quite acute. When pressure upon the cord is added, there is paralysis or paresis, sometimes muscular atrophy and anæsthesia. Any of the above symptoms may be acute or chronic, according to the nature of the primary disease.

The diagnosis between spinal meningitis and myelitis is often not easy, for except in acute cases the two processes are usually associated; and in a given case it may be difficult to decide whether the lesion of the cord or of the membranes is the more important one. In meningitis, pain, tenderness, spasm, and irritative symptoms are generally more prominent, while loss of power and anæsthesia are usually partial. In myelitis the pain, tenderness, and other irritative symptoms are less marked, while paralysis and anæsthesia may be complete.

Treatment.—This is first of the disease with which it is associated; in addition, counter-irritation by means of the Paquelin cautery, rest in bed, and in severe cases even immobilization of the spine by a mechanical support. Iodide of potassium is often useful.

MYELITIS.

Myelitis is a rare disease in children, with the exception of two varieties which are discussed under separate heads, viz., compression-myelitis and acute poliomyelitis. Otherwise myelitis usually results from injury, but it may occur as a complication of any of the acute infectious diseases, especially typhoid or scarlet fever, and diphtheria, and even as a primary disease, where it is attributed to exposure or cold, but where it is probably infectious. Chronic myelitis may be due to hereditary syphilis.

Myelitis usually occurs in children over ten years of age. In situation, it may be transverse, diffuse, or disseminated; the process may be acute, subacute, or chronic. The lesions and the symptoms are essentially the same as when the disease occurs in the adult.

Symptoms.—Myelitis usually comes on rather gradually, with only local symptoms; but the onset may be quite acute, with severe general symptoms,—fever, pain, prostration and localized or general convulsions. The local symptoms vary with the seat and the extent of the disease.

In transverse myelitis loss of power and anæsthesia are present below the level of the lesion; either of these may be partial or complete. At the level of the lesion there is a zone of hyperæsthesia and "girdle-pains." All the reflexes below the seat of the lesion are exaggerated. Those at the level of the lesion are lost. There may be loss of control of the sphincters, bed-sores, degenerative changes in the paralyzed muscles, contractures, and vaso-motor disturbances. The paralyzed muscles may be rigid or flaccid according to the seat and extent of the lesion.

When transverse myelitis is situated in the cervical region there are paralysis and anæsthesia of the arms, legs, and trunk. All the reflexes are exaggerated, and there is general rigidity of the paralyzed muscles. There are incontinence of fæces and retention of urine, followed by incontinence from overflow. The pupils are frequently contracted, and there may be optic neuritis. Atrophy, when present, usually affects the muscles of the arms, and indicates that the cord to a considerable extent is involved. There is great danger to life, owing to paralysis of the muscles of respiration.

When the seat of disease is the dorsal region, the symptoms are similar to those above described, with the exception that the arms escape, and that the eye-symptoms are usually wanting. This is the most favourable seat of the disease.

When the disease is situated in the lumbar region, in addition to paraplegia and anæsthesia of the legs, there is, from the beginning, incontinence of urine and fæces. The knee reflexes are lost; the muscles atrophy, and usually give the reaction of degeneration. Bed-sores are frequent.

In diffuse myelitis the symptoms are a combination of the above groups. If a large part of the cord is involved, there are general paralysis and anæsthesia, loss of reflexes, marked trophic disturbances, bed-sores, etc.

The course of myelitis is slow, and it usually progresses steadily from bad to worse. Death is due to exhaustion or complications—cystitis, bed-sores, or hypostatic pneumonia—or to some intercurrent disease. In a small proportion of the cases there may be partial recovery, but very rarely is this complete. The diagnosis is to be made from spinal meningitis, tumours, and hæmorrhage.

Treatment.—The treatment of the early stage consists in the use of ice to the spine, or counter-irritation by means of dry cups, mustard, or the Paquelin cautery. Later, the iodide of potassium should be given in all cases; improvement may follow its use, even when there is no suspicion of syphilis, but large doses are required, and for a long period. Electricity is contra-indicated except in chronic cases, and then but little improvement is likely to result from its use. In these patients the most important thing is careful attention to cleanliness and to posture, in order to prevent bed-sores, cystitis, and pneumonia.

COMPRESSION-MYELITIS.

Synonyms: Pressure-paralysis of the spinal cord; Pott's paraplegia.

Compression-myelitis is sometimes traumatic, but usually follows caries of the spine. It most frequently complicates this disease when the cervical or upper dorsal vertebræ are involved, rarely when the lower half of the spinal column is affected. This difference is probably due to the smaller size of the spinal canal in its upper portion. According to Gibney,* paraplegia is seen in fifty per cent of the cases of caries of the upper half of the spine. Essentially the same condition, so far as the cord is concerned, may result from tumours of the spinal cord, or from anything else causing pachymeningitis. These, however, are exceedingly rare in childhood.

Lesions.—In spinal caries there occurs as a result of tuberculous disease a softening of the bodies of the vertebræ, which fall together from the pressure due to the superincumbent weight of the body. This causes a backward projection known as the kyphosis, or angular deformity. The spinal canal is encroached upon by the remains of the vertebral bodies whose ligamentous attachments have been loosened, and also by inflammatory products the result of periostitis, and localized inflammation of the dura mater, chiefly of the external layer, but which sometimes affects the internal layer also. All these conditions lead to the production of a mass of inflammatory material, often containing tuberculous deposits, which is chiefly in front of the cord, but may surround it. The compression takes place slowly in most of the cases, from the gradual progress of the lesions mentioned. In a small number of cases there may be a sudden pressure from the slipping backward of one of the vertebral bodies.

In recent cases the cord at the seat of compression is a little smaller than normal. It is usually involved to the extent of from half an inch to two inches. Paraplegia may have existed where the changes found in the cord are very slight, and sometimes where no changes are visible to the naked eye. In more protracted and more severe cases, the cord is much smaller at the point of disease, and under the microscope shows the changes of interstitial myelitis (Gowers) with meningitis. In old cases there are degeneration of the nerve elements, atrophy, and sometimes disappearance of the ganglion cells, with more or less destruction of the nerve fibres; sometimes all distinction between the gray and white substance is lost. In addition to these marked changes at the point of pressure, there may be ascending or descending degeneration, as from other focal lesions.

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* Journal of Mental and Nervous Diseases, April, 1897.

There is usually inflammation of the nerve roots, which have also suffered compression. It is in many cases surprising to see to what degree the cord may be compressed and still preserve its functions.

Symptoms.—In caries of the cervical region the symptoms of compression-myelitis not infrequently precede the deformity, and, in fact, the other objective symptoms of bone disease. The earliest symptoms of caries usually arise from irritation of the nerve roots, and consist of acute pains not often referred to the spine, but radiating to the different regions to which these nerves are distributed. They are felt in the neck, in the chest, in the epigastrium, and sometimes in the loins. Such symptoms indicate the presence of pachymeningitis, and may be present whatever the location of the vertebral caries. Accompanying these pains, there is noticed a gradual weakness in the lower extremities, and sometimes also in the arms, according to the location of the disease. This may steadily increase for several weeks until there is complete paralysis. Other symptoms are then commonly present. There is usually some degree of anæsthesia, but in many cases there is none, and there may be numbness, tingling, formication, and pain. The sphincters are not often involved. When the disease is in the upper half of the cord, there are rigidity of the extremities and great exaggeration of all the reflexes, with marked ankle-clonus. In the rare cases in which the lumbar enlargement is involved, there may be loss of reflexes, paralysis of the sphincters and bed-sores.

The distribution of the paralysis will depend upon the point of compression. If this is in the cervical region, all four extremities will be paralyzed; if in the dorsal region, only the legs. In rare cases the paralysis is unilateral, and if there is no spinal deformity the condition may be a most puzzling one. According to the extent of the secondary lesions in the cord, there may occur muscular atrophy and contractures. With disease in the upper cervical region, death may result from sudden pressure upon the cord, owing to a dislocation of the odontoid process, which happened in one of Gibney's cases; or there may be vomiting, pupillary symptoms, irritation of the phrenic nerve causing hiccough, or pressure causing paralysis of the diaphragm.

Course and Prognosis.—These depend much upon the treatment of the case. In many cases of paralysis occurring early in caries, complete recovery takes place in the course of a few weeks, sometimes in a few days, after the application of a proper mechanical support. This may be true even where the paralysis has continued for three or four months. In the cases which have been long neglected, or those in which the paralysis develops while proper mechanical treatment is being carried out, the chances of improvement, or at least of rapid improvement, are not nearly so good. Gibney gives the following statistics of one hundred and thirty-three cases under his personal observation: thirty-one proved fatal; nine dying from myelitis, fourteen from other diseases subsequent to recovery from the paralysis, and six from tuberculosis before complete recovery; seventy-four recovered from the paraplegia; twenty-seven were recorded as improved or still under treatment. Relapses occurred in about fifteen per cent of the cases; nearly all of these, however, subsequently recovered. The usual duration of the disease is from three months to two years. Complete recovery has often taken place in cases that have persisted for four or five years. No case should be considered hopeless no matter how long the symptoms have lasted, unless there is marked atrophy with loss of electrical reactions, and contractures have taken place.

Diagnosis.—This is rarely difficult. Spinal caries should be suspected in every case where the symptoms point to transverse myelitis coming on without definite cause. The gradual onset, the radiating pains, the stiffness of the spine in walking, the gradual loss of power, the increased reflexes and ankle-clonus—all are usually present and characteristic. They are sufficient to warrant the diagnosis of spinal caries, even when no deformity exists. When there is deformity, the symptoms are unmistakable.

Treatment.—The most important indications are the removal of pressure and the fixation of the spine by a proper mechanical support. The best results are secured by the recumbent position, the child being fixed upon a frame, continuous traction being made upon the head. Other measures to be advised are the Paquelin cautery, and the internal use of potassium iodide. From his very extensive experience, Gibney has more confidence in this drug than in all else except mechanical treatment. Large doses are required, often from sixty to ninety grains being given daily for months. From personal observation of many of Gibney's cases I can bear testimony both to the beneficial effect of the iodide, and to the ease with which it is generally borne by children in the doses mentioned. Very often patients gained steadily in weight while taking the drug, and acne was the exception. The iodide should always be largely diluted. In all cases patients should be carefully watched, kept scrupulously clean, and the position changed frequently to prevent the formation of bed-sores. Electricity is contra-indicated. When the paralysis develops rapidly or occurs suddenly, relief may sometimes be obtained by the operation of laminectomy; but little is to be expected from this in the slow cases.

ACUTE POLIOMYELITIS.

Synonyms: Infantile spinal paralysis, acute atrophic paralysis.

This disease is characterized by an acute onset, generally with febrile symptoms, by an early and usually extensive loss of power, and by a considerable degree of spontaneous improvement except in certain groups of muscles which remain permanently paralyzed, and undergo a very rapid and marked atrophy. A chronic form of the disease is described in adults, but this is rarely, if ever, seen in children.

Acute poliomyelitis is the most frequent cause of paralysis in early life and it is often designated simply as *infantile paralysis.*

Etiology.—In 566 * cases the age at which the paralysis developed was as follows:

During the first year	20 per cent.
" " second year	38 "
" " third year	22 "
" " fourth, and fifth years	15 "
After " fifth year	5 "

From this table it will be seen that the great proportion of cases develop before the fifth year, and that eighty per cent of them begin during the first three years, the most frequent period being the second year.

Boys are rather more frequently affected than girls. In the series referred to, fifty-five per cent were males and forty-five per cent were females. Hereditary influences seem to have but little effect in the production of this disease. It is rare to find several cases in the same family, or to trace any relation to nervous antecedents. The onset of the great proportion of the cases is in summer. Of Sinkler's cases, eighty per cent began during the five warm months. This fact is decidedly against the theory so often advanced, that the disease results from exposure to cold. There are, however, a few cases in which the connection between exposure and the disease seems to be a close one. On account of the time of onset—most frequently in the second year—the disease is often ascribed to dentition. In my series this was given as the cause in one fifth of the cases. The connection is at most merely a coincidence. Traumatism is sometimes given as a cause, but the proportion of cases in which the paralysis can be fairly attributed to injury is very small, yet there are a few in which a definite injury of considerable severity has immediately preceded the onset. In about twelve per cent of the cases above mentioned the paralysis came on as a sequel to some other acute disease; this list includes nearly all the diseases of infancy, those most frequently noted being diarrhœa, scarlet fever, and measles; but in the great proportion of the cases the patient was in good health at the time of the attack.

The essential cause of the disease is as yet unknown. On account of the close relation of the lesion to the distribution of the blood-vessels, many recent writers believe poliomyelitis to be of infectious origin, the cord changes being the result of infectious embolism or thrombosis. The occasional occurrence of small epidemics strengthens this opinion.

Lesions.—Infantile spinal paralysis is due to an acute inflammation of the gray matter of the anterior portion of the spinal cord. The late changes which occur in the cord as a result of this process have for many years been well established; but the early changes are even yet a matter of dispute, owing to the lack of opportunities of examining the cord during the stage of acute inflammation.

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* These statistics and those which follow in this article are derived from the following sources: Sinkler, in Keating's Cyclopædia, vol. iv, 355 cases; Galbraith, American Journal of Obstetrics, 1894, 75 cases; the remaining 146 are personal cases and others taken from the records of the Hospital for Ruptured and Crippled, New York.

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In paralysis of the trunk, the diaphragm and other respiratory muscles are very rarely affected. In combinations of an upper and a lower extremity, the limbs are more frequently affected upon opposite sides than upon the same side. The sphincters almost invariably escape.

Course of the disease.—The rapid development of the paralysis is followed by a period of from one to four weeks' duration in which but little change is seen in the affected muscles. This is followed by spontaneous improvement, which, according to Gowers, begins in the muscles last affected, and generally reaches its limit in about three months. After this time but little spontaneous improvement is to be looked for, and the residual paralysis is likely to be permanent. By the end of two months marked atrophy is present in the paralyzed muscles. The affected limb is distinctly smaller than its fellow, this being quite apparent even in the paralysis, and six from tuberculosis before complete recovery; seventy-four recovered from the paraplegia; twenty-seven were recorded as improved or still under treatment. Relapses occurred in about fifteen per cent of the cases; nearly all of these, however, subsequently recovered. The usual duration of the disease is from three months to two years. Complete recovery has often taken place in cases that have persisted for four or five years. No case should be considered hopeless no matter how long the symptoms have lasted, unless there is marked atrophy with loss of electrical reactions, and contractures have taken place.

Diagnosis.—This is rarely difficult. Spinal caries should be suspected in every case where the symptoms point to transverse myelitis coming on without definite cause. The gradual onset, the radiating pains, the stiffness of the spine in walking, the gradual loss of power, the increased reflexes and ankle-clonus—all are usually present and characteristic. They are sufficient to warrant the diagnosis of spinal caries, even when no deformity exists. When there is deformity, the symptoms are unmistakable.

Treatment.—The most important indications are the removal of pressure and the fixation of the spine by a proper mechanical support. The best results are secured by the recumbent position, the child being fixed upon a frame, continuous traction being made upon the head. Other measures to be advised are the Paquelin cautery, and the internal use of potassium iodide. From his very extensive experience, Gibney has more confidence in this drug than in all else except mechanical treatment. Large doses are required, often from sixty to ninety grains being given daily for months. From personal observation of many of Gibney's cases I can bear testimony both to the beneficial effect of the iodide, and to the ease with which it is generally borne by children in the doses mentioned. Very often patients gained steadily in weight while taking the drug, and acne was the exception. The iodide should always be largely diluted. In all cases patients should be carefully watched, kept scrupulously clean, and the position changed frequently to prevent the formation of bed-sores. Electricity is contra-indicated. When the paralysis develops rapidly or occurs suddenly, relief may sometimes be obtained by the operation of laminectomy; but little is to be expected from this in the slow cases.

ACUTE POLIOMYELITIS.

Synonyms: Infantile spinal paralysis, acute atrophic paralysis.

This disease is characterized by an acute onset, generally with febrile symptoms, by an early and usually extensive loss of power, and by a considerable degree of spontaneous improvement except in certain groups of muscles which remain permanently paralyzed, and undergo a very rapid and marked atrophy. A chronic form of the disease is described in adults, but this is rarely, if ever, seen in children.

Acute poliomyelitis is the most frequent cause of paralysis in early life and it is often designated simply as *infantile paralysis.*

Etiology.—In 566 * cases the age at which the paralysis developed was as follows:

During the first year	20	per cent.
" " second year	38	"
" " third year	22	"
" " fourth, and fifth years	15	"
After " fifth year	5	"

From this table it will be seen that the great proportion of cases develop before the fifth year, and that eighty per cent of them begin during the first three years, the most frequent period being the second year.

Boys are rather more frequently affected than girls. In the series referred to, fifty-five per cent were males and forty-five per cent were females. Hereditary influences seem to have but little effect in the production of this disease. It is rare to find several cases in the same family, or to trace any relation to nervous antecedents. The onset of the great proportion of the cases is in summer. Of Sinkler's cases, eighty per cent began during the five warm months. This fact is decidedly against the theory so often advanced, that the disease results from exposure to cold. There are, however, a few cases in which the connection between exposure and the disease seems to be a close one. On account of the time of onset—most frequently in the second year—the disease is often ascribed to dentition. In my series this was given as the cause in one fifth of the cases. The connection is at most merely a coincidence. Traumatism is sometimes given as a cause, but the proportion of cases in which the paralysis can be fairly attributed to injury is very small, yet there are a few in which a definite injury of considerable severity has immediately preceded the onset. In about twelve per cent of the cases above mentioned the paralysis came on as a sequel to some other acute disease; this list includes nearly all the diseases of infancy, those most frequently noted being diarrhœa, scarlet fever, and measles; but in the great proportion of the cases the patient was in good health at the time of the attack.

The essential cause of the disease is as yet unknown. On account of the close relation of the lesion to the distribution of the blood-vessels, many recent writers believe poliomyelitis to be of infectious origin, the cord changes being the result of infectious embolism or thrombosis. The occasional occurrence of small epidemics strengthens this opinion.

Lesions.—Infantile spinal paralysis is due to an acute inflammation of the gray matter of the anterior portion of the spinal cord. The late changes which occur in the cord as a result of this process have for many years been well established; but the early changes are even yet a matter of dispute, owing to the lack of opportunities of examining the cord during the stage of acute inflammation.

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Electrical reactions.—Very early in the disease the atrophied muscles begin to lose their power to respond to faradism. In the muscular groups which are to be permanently paralyzed, the faradic response may be lost in a week. The muscles in which recovery is to take place often preserve a certain degree of contractility, although this is less than normal, and improves later. The response to the galvanic current may be increased for a few months, and then slowly fail as the muscular fibres themselves degenerate, and at the end of two or three years it may disappear altogether. The reaction of degeneration is present with great uniformity in the atrophied muscles, but in them alone.

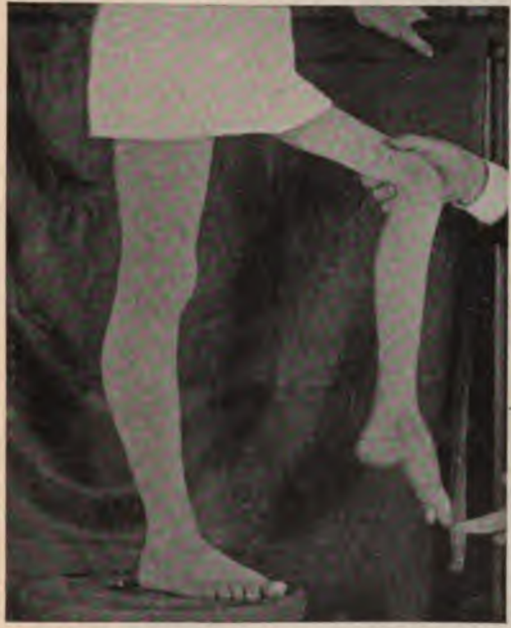

FIG. 166.—An old case of infantile spinal paralysis of the entire left lower extremity, showing extreme atrophy of the thigh and leg, and a very characteristic deformity of the foot.

Residual paralysis and deformity.—Only one lower extremity is involved in half the cases, and the paralysis is usually incomplete and confined to certain groups of muscles. The extensors both of the thigh and of the leg are nearly always involved to a greater degree than the flexors, and in very many cases only the extensor groups are paralyzed. The muscles most frequently affected are the anterior tibial group, and next the peroneal group. The most frequent deformity resulting from this paralysis is talipes valgus, and next to this talipes varus, both of these being usually associated with a certain amount of equinus. In very rare cases there is talipes calcaneus. Most children with paralysis of only one lower extremity are able to walk alone, or with the assistance of a steel brace.

Paralysis of both lower extremities is the next in frequency. This also is rarely complete. In forty-three cases of my series there was originally complete paraplegia, but it was permanent in only three. The extent of recovery varies much in different cases. Usually one leg recovers to a much greater degree than the other. Most of these patients are able to walk with the assistance of braces, a few only by the aid of crutches. Some walk while they are young, but are unable to do so when fully grown, because the disproportion between the size of the body and the limbs is then much greater.

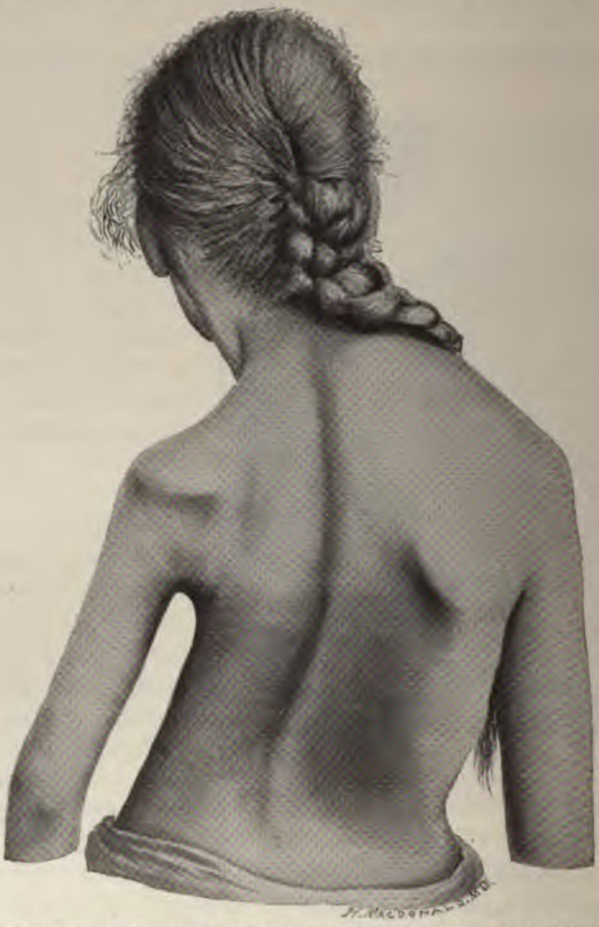

FIG. 167.—An old case of infantile spinal paralysis of the left arm and shoulder muscles, with resulting lateral curvature. The spinal deformity is increased by the fact that the patient had also suffered from empyema of the left side.

Paralysis of one upper extremity rarely occurs alone, but is associated with paralysis of one or both lower extremities. Complete paralysis of an arm is rarely, if ever, seen. The muscular groups affected may be the small muscles of the hand, the muscles of the forearm,—especially the extensors,—or the shoulder group. Of single muscles, the one most frequently involved is the deltoid; this may result in subluxation of the shoulder. From paralysis of the muscles of the trunk or shoulder of one side, lateral curvature may develop (Fig. 167). If the serratus magnus is affected the scapula stands out prominently, giving rise to the so-called "angel-wing" deformity.

Diagnosis.—The general symptoms of the onset have nothing characteristic about them, and no diagnosis can be made until the paralysis has taken place. The acute onset, the rapid wasting, the spontaneous improvement in certain groups of muscles, the absence of sensory symptoms, and finally the reaction of degeneration,—all constitute a type which it is difficult to confound with any other disease.

At the onset this paralysis may resemble that resulting from acute transverse myelitis. In the latter, however, we get anæsthesia, exaggerated knee-jerk, ankle-clonus, generally involvement of the sphincters, a tendency to bed-sores, slight wasting, and no reaction of degeneration. It is, besides, extremely rare.

Multiple neuritis is in most cases easily distinguished from poliomyelitis by its gradual onset, by the presence of pain and other sensory symptoms as well as loss of power, and by the fact that spontaneous recovery generally occurs within two or three months. Besides, there is usually a history of antecedent diphtheria. But multiple neuritis sometimes begins suddenly with febrile symptoms, and paralysis may occur early, precisely as it does in poliomyelitis. Furthermore, in some cases of neuritis, the sensory symptoms are not marked, and they may have entirely disappeared before the patient is seen. In such cases the diagnosis from poliomyelitis may be difficult or even impossible except by the course of the disease; for atrophy is common to both conditions, and even the electrical reactions may be identical. There is no doubt that some cases formerly reported as examples of poliomyelitis terminating in complete recovery were really cases of multiple neuritis.

The diagnosis from acute cerebral palsy is chiefly difficult when the spinal paralysis has been hemiplegic or diplegic in type, or when after cerebral hemiplegia the leg or the arm has recovered so completely that the case resembles monoplegia. In cerebral palsies there is usually rigidity; there is no reaction of degeneration; other cerebral symptoms are commonly present, or there is a history of an onset with cerebral symptoms; and the atrophy is less marked. The most diagnostic point is the electrical reactions.

Acute poliomyelitis may be mistaken for other than nervous diseases.

In the early stage it may be confounded with the pseudo-paralysis of scurvy. I have several times seen the mistake made of diagnosticating paralysis where scurvy was present. In scurvy, however, there is seen excessive tenderness and hyperæsthesia, pain upon motion, especially about the knees, spongy gums, and sometimes ecchymoses about the joints. The muscular weakness of rickets is sometimes mistaken for infantile paralysis. However, in rickets the symptoms are always bilateral, the electrical reactions are normal, and other signs of rickets are present. In all doubtful cases the chief reliance for the diagnosis of paralysis should be placed upon the character of the electrical reactions. The lameness resulting from paralysis may resemble somewhat that due to hip-disease; but with a careful examination there can rarely be any difficulty in making the differential diagnosis.

Prognosis.—Poliomyelitis is accompanied by little, if any, danger to life. It is possible that death may take place during the stage of acute inflammation, but this is certainly extremely rare. The most important question in early prognosis is whether there will be any permanent paralysis, and, if so, what will be its extent. The important symptoms for prognosis are the amount of wasting and the condition of the electrical reactions. Muscles which in ten days have lost completely their faradic contractility are almost certain to waste rapidly and severely. The best indication of coming improvement is the return of faradic contractility. If this is completely lost for six months, recovery is doubtful; if for one year, improvement in these muscles is not to be expected. If faradic contractility has never been lost, very great and early improvement in the paralyzed muscles may be confidently predicted. After three months but little spontaneous improvement is to be looked for, and after two years none at all. Complete recovery is possible only with a lesion of very limited extent; and while it may occur, it is so infrequent that it should not be expected.

Treatment.—Unfortunately, most of the cases do not come under observation during the acute stage, or the nature of the disease is overlooked until the paralysis has occurred. In the early stage the indications are, to induce free perspiration by hot baths, to keep the patient in a prone or lateral position, and to use counter-irritation to the spine by means of dry cups, mustard, or the Paquelin cautery, or an ice-bag may be placed along the spine. The natural course of the disease is to be kept in mind, for the tendency is to overestimate the effect upon the paralysis of the drugs used in the early stage. On theoretical grounds, ergot is indicated, but it is doubtful whether any drugs have much effect.

After all acute symptoms have subsided, or at the end of two or three weeks, electricity may be used, but its curative effects have been very greatly overestimated. The object in using electricity is to keep up the nutrition of the muscles until the cord has recovered, which it is almost certain to do to a considerable degree. But no amount of electrization can preserve muscles whose ganglion cells have completely disappeared. These continue to waste and lose their faradic contractility, no matter how early electricity is begun nor how faithfully it is continued. Faradism may be used for such groups as respond to it; otherwise galvanism should be employed. The beneficial results from electricity are to be obtained in the first year, chiefly in the first six months. Too much can not be said against the routine use of electricity in cases which have been paralyzed three or four years, with the vain hope that some good may be done, even though there is no response to either current. Strychnine may be used in conjunction with electricity after all symptoms of central irritation have subsided, but there is still great diversity of opinion regarding its effect.

Friction and massage are of undoubted value in improving the circulation and the nutrition of a limb, and should be continued regularly twice a day for a long period.

Mechanical Treatment.—The first use of mechanical appliances is the prevention of deformity. All cases of paralysis should be carefully watched, and braces applied as soon as any tendency to deformity from muscular contraction shows itself. This is much easier than to overcome deformities which have been allowed to develop, and quite as important for the patient. The second use of apparatus is to furnish support to the limb in order to enable the child to walk. By such means many get about with tolerable comfort, for whom locomotion without apparatus is impossible except with crutches. The third purpose of apparatus is, to overcome existing deformities in neglected cases.* Braces are generally used in conjunction with myotomy or tenotomy of the various shortened tendons, excision of portions of elongated tendons, and the production of artificial anchylosis in cases of "flail joints." By these means the orthopædic surgeon is able to give a great deal of relief to these unfortunate and sometimes helpless patients.

On the whole, the treatment is extremely unsatisfactory, and the result depends upon the severity and extent of the original disease, rather than upon the particular line of treatment adopted or the time at which it is begun.

TUMOURS OF THE SPINAL CORD.

Tumours of the cord are exceedingly rare in childhood, and almost unknown in infancy. The most common varieties seen in early life are glioma, sarcoma, and tuberculous tumours. Eisenschitz has reported a case of tuberculous tumour in the dorsal region occurring in a child of three and a half years. There was a similar growth in the cerebellum. The symptoms were essentially those of compression-myelitis.

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* See Gibney, New York Medical Journal, April 3, 1886, On the Limitation of Therapeutics in Infantile Paralysis.

In my service at the Babies' Hospital I have had a case of glioma of the cord in a child only one year old, which was in many respects unique. The early symptoms were gradual paralysis of the upper extremities, to which were added later, stiffness of the neck, and finally immobility of the head—the position being that of typical cervical caries. During the sixteen days of observation there was high fever, from 101° to 104° F. There were no pupillary or vaso-motor symptoms. At the autopsy the cord was found to be the seat of a diffuse gliosis. In the cervical region there was marked enlargement, the cord being fully four times its natural size. A microscopical examination by Dr. C. A. Herter showed that the growth apparently began in the vicinity of the central canal, and that the gliomatous process involved the entire length of the cord.*

A somewhat similar case has been reported by Miura in a boy of eight years.

The diagnosis of tumours of the spinal cord in infancy is practically impossible. In later childhood they are most apt to be mistaken for Pott's disease, but the symptoms are the same as those seen in tumours of adult life.

SYRINGO-MYELIA.

Syringo-myelia, although a rare disease, is sometimes seen in early life. The term is applied to a condition in which there is a cavity in the cord the result of a pathological process, in contradistinction to the cases in which a cavity is the result of a malformation, or *hydromyelus*, although it is not infrequent for the two conditions to be associated. The pathological process which precedes the cavity formation is now thought to be, in most cases at least, an infiltration of the substance of the cord with gliomatous cells. The process is somewhat similar to that just described in the case of tumour of the spinal cord, with the exception that where it results in cavity formation it is slower. The infiltration in these cases usually begins near the central canal. It is followed by a degeneration and breaking down of the infiltrated areas, beginning at the centre. As the cavity forms it extends, and usually first invades the gray matter of the commissure, later the posterior gray horns, the posterior columns, or the anterior horns. The resulting cavity is usually irregular in shape, and may be very small, or may extend through a large part of the length of the cord. It is most frequently situated in the lower cervical and upper dorsal regions. It is filled with fluid, and surrounded by gliomatous tissue.

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* For a full report of this case by Dr. Herter and myself, see American Journal of the Medical Sciences, April, 1895. See also Kohts, Beitrag zur Diagnostik der Rückenmarkstumoren im Kindesalter, Dresden, 1886.

According to Starr, the essential symptoms are of three kinds: (1) There is progressive muscular atrophy, with paralysis of some or all the muscles of one limb, usually extending to the opposite limb and to the trunk, sometimes accompanied by the reaction of degeneration; (2) vasomotor and trophic disturbances in the affected limb, such as cyanosis, coldness, bullous eruptions, ulceration, abscesses, atrophy, and sometimes fragility of the bones and diminution of perspiration; (3) sensory disturbances, which are probably the most characteristic symptoms of the disease,—there is loss of the sense of pain and of temperature in the atrophied part, while the sense of touch and of location may be preserved. The extent and distribution of these symptoms will of course depend upon the position of the disease.

The course of syringo-myelia is essentially chronic, the duration being usually several years; and although spontaneous arrest sometimes occurs the disease is in most cases steadily progressive.

The cause is unknown, and it is not influenced by any form of treatment.

FRIEDREICH'S ATAXIA.

This is a chronic disease of the spinal cord and medulla, which begins most frequently in childhood or about puberty. The lesion affects first the posterior columns, afterward the crossed pyramidal tracts, the direct cerebellar tracts in the lateral columns, and Clarke's vesicular columns in the gray matter of the cord. There is probably some disease of the medulla, the pons, and possibly of the cerebellum and the posterior nerve-roots. In advanced cases other parts of the cord may be involved. The disease is seen in certain families, often affecting several members in succession at about the same age. It occurs particularly in families where alcoholism, insanity, and other nervous diseases are frequent.

Bramwell, in his monograph upon this disease, gives the following as the characteristic symptoms: There is ataxia, first of the lower extremities, but gradually extending to the upper extremities and the face. Early in the disease there is some weakness in the legs, especially in the anterior group of muscles. In the late stages this is marked and accompanied by atrophy. The gait is peculiar, like that of ordinary ataxic patients, the difficulty in walking being due to the ataxia and not to the paresis. After a time there is produced a characteristic deformity of the foot,—it is shortened, as if from pressure against the toes and the heel, the instep is high, and the extensor tendon of the great toe stands out prominently. This deformity is seen quite early in the disease. There is often lateral curvature of the spine. The knee-jerk is absent. Unprovoked and uncontrollable laughter is quite a characteristic symptom of the disease. The patient is unable to stand with his eyes closed. There are palpitation, occipital headache, and sometimes vertigo. In the later stages speech is slow and difficult, and the patient talks like one intoxicated. The expression of the face is vacant, and often nystagmus is present. There may be choreic movements. The symptoms steadily progress until the patient may be helpless, although the general health may remain good for years.

The disease is distinguished from locomotor ataxia by the absence of the "lightning pains," and of the bladder, rectal, or genital symptoms, the pupillary changes, the optic-nerve atrophy, and the trophic changes in the bones and joints. It is distinguished from cerebral tumour by the absence of headache, vomiting, and optic neuritis, and by its longer course. The progress of the disease is slow but steady. It may last from twenty to thirty years. It is incurable.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS).

This rare disease is occasionally seen in early life. In regard to its etiology but little is definitely known, the usual causes assigned being the same as those of myelitis.

It is characterized by a paralysis—sometimes preceded by general symptoms of *malaise*, fever, etc.—which begins in the legs and spreads rapidly to the muscles of the trunk and upper extremities; finally it may involve the neck, diaphragm, and muscles of articulation. The paralysis develops quite rapidly, often attaining its height in from twenty-four to forty-eight hours, sometimes even proving fatal within this time. In other cases it comes on gradually, and may be two or three weeks in reaching its maximum. There is dyspnœa from involvement of the muscles of respiration. The paralyzed muscles are flaccid. There is hyperæsthesia, followed by partial or complete anæsthesia and loss of reflexes. There are no changes in the electrical reactions, no atrophy, no bed-sores, and usually no involvement of the sphincters. Occasionally the arms may be affected before the legs, and even the bulbar symptoms may be the first noticed. Death is the most frequent termination, and in fatal cases the disease lasts from two days to a week. If recovery takes place, it is after two or three months of illness.

The pathology of the disease is as yet unknown. The indications for treatment are the same as in acute myelitis, for in the beginning the two diseases can not usually be distinguished from each other.

THE MUSCULAR ATROPHIES.

These cases may be broadly divided into two groups, following in the main the classification of Sachs:* (1) Those dependent upon disease of the spinal cord,—the spinal atrophies; (2) those which are primarily diseases of the muscles themselves,—the idiopathic atrophies.

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* New York Medical Journal, December 15, 1888.

In the group of atrophies of spinal origin belong (1) the "hand type" of Aran and Duchenne, which has been shown to be dependent upon a lesion of the spinal cord; (2) the "peroneal type" of Charcot, Marie, and Tooth, which as yet lacks positive pathological proof of its spinal origin, although its etiology, symptoms, and course leave but little doubt that it belongs in the same category with the hand type.

In the second (idiopathic) group are included (1) muscular pseudo-hypertrophy, and (2) the so-called "juvenile atrophy" of Erb, which is a much less frequent condition. These two varieties have the following features in common: There is progressive wasting, beginning early in childhood, and associated at some period with hypertrophy of certain muscles. There are no fibrillary contractions, no reaction of degeneration, and no lesions in the cord. From a pathological point of view these diseases might be more properly considered elsewhere, but they are so closely associated clinically with the spinal atrophies that it has seemed better to describe them in this connection.

Progressive Muscular Atrophy of the Hand Type.—This disease is characterized by a very slow but progressive wasting, which usually begins in the muscles of the ball of the thumb of one or both hands. Then the palmar group of muscles belonging to the little finger are affected, and later the interossei. When the wasting has reached a certain degree, there is produced a peculiar and characteristic deformity of the hand known as *main en griffe*, or "claw-hand." Following these muscles, those of the forearm may be affected. At this point the disease is sometimes arrested, or the atrophy may extend to the muscles of the arm and shoulder, especially the deltoid, and finally to those of the back. Exceptionally, the atrophy begins in the muscles of the shoulder group or even in those of the leg. The wasting takes place very slowly, the muscles disappearing fibre by fibre, but the degree which may be reached is often extreme. The only other characteristic symptoms are fibrillary contractions in the muscles which are soon to atrophy. The patient is not conscious of them, but they are visible. The faradic contractility is preserved just in proportion to the amount of muscle remaining. If the atrophy is complete, it is entirely lost.

The course of the disease is a very chronic one, covering many years. It is incurable. In rare cases the process may extend to the muscles of the tongue, affecting deglutition and articulation, and death may occur from interference with respiration; otherwise the disease does not tend to shorten life.

In this form of atrophy heredity is an important etiological factor. The disease may occur in children, but very often does not begin until after puberty. The lesion consists in an atrophy of the ganglion cells of the anterior horns of the spinal cord, followed by secondary degeneration of the anterior nerve-roots.

Progressive Muscular Atrophy of the Peroneal Type.—This is much less frequent than the variety just described. In this form, the first to waste are the anterior muscles of the leg, especially the extensor longus hallucis and extensor communis digitorum, afterward the peroneal group. The small muscles of the foot are next affected, and the disease may then go on to involve the muscles of the calf. At this point it may be arrested permanently, or for several years, after which the thigh muscles may waste like those of the leg. After many years the hands are in some cases involved as in the type previously described, and even the muscles of the forearm. As a rule, the supinator longus, the muscles of the shoulder, neck, trunk, and face, escape altogether. The atrophy is generally symmetrical, but not invariably so. The cutaneous reflexes are usually present. There is no pain. The reaction of degeneration is present in some of the muscles, and fibrillary contractions are frequent, but not always seen.

In this variety also the influence of heredity may often be traced. It is said that boys usually inherit the disease through the mother. Like the previous type, it begins late in childhood or not until after puberty.

As stated above, positive proof that this disease is due to a central lesion in the cord is as yet lacking. Analogy, however, leads to the belief that it depends upon changes in the ganglion cells of the anterior horns in the lumbar region, similar to those found in the cervical region in the hand type. The course of the disease is very chronic, and it, too, is incurable. The resulting deformity resembles that seen after poliomyelitis, and may require the same mechanical treatment, with similar operations for relieving contractions.

Muscular Pseudo-Hypertrophy (Pseudo-Hypertrophic Paralysis).—This is the most frequent and best-known variety of the idiopathic atrophies. It is a disease of certain families, often three or four children being affected, the boys much more frequently than the girls. The symptoms as a rule come on early in childhood, nearly always before the tenth year. The earlier symptoms relate to a general weakness of the lower extremities, which is accompanied by a marked increase in the size of certain muscular groups, usually those of the calves, but sometimes more of the thighs or the gluteal regions. Children walk late and unsteadily, and fall very easily. They have special difficulty in rising from the floor and in mounting stairs. The method of rising is quite characteristic: the patient lifts his body until he touches the floor only with the hands and feet; then he proceeds to "climb up himself" by putting first one hand upon the knee, and then the other, gradually moving his hands higher and higher up the thighs until the erect position is attained. This is seen in most of the cases, but not in all.

The size attained by the calves is sometimes very great. Gowers mentions a case in which a boy of twelve had calves measuring fourteen and a half inches in circumference. The enlargement may affect almost any muscular group of the lower extremity. In the upper extremity, the infra-spinatus is most frequently enlarged, next the supra-spinatus and the deltoid. The pectorals and latissimus dorsi are never enlarged, but are generally markedly wasted. Most of these patients exhibit while standing a marked degree of lumbar lordosis, due to the weakness of the extensors of the hip. This is well shown in Fig. 168. The patient may be so weak upon his legs that the slightest touch will cause him to fall, even with his apparently immense muscular development. The small muscles are generally weaker than those which are enlarged.

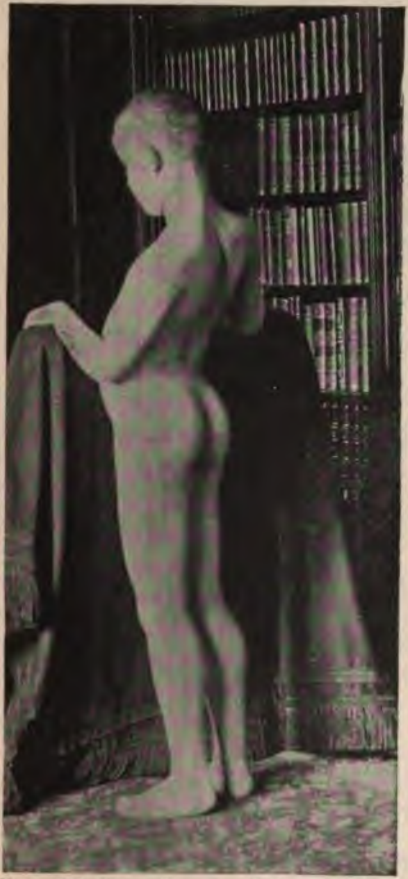

FIG. 168.—Muscular pseudo-hypertrophy, showing to a moderate degree the large calves and gluteal regions with a marked lordosis. (From a photograph by Dr. M. A. Starr.)

Later in the disease marked atrophy occurs with a corresponding weakness of all the affected groups, and the patient may be unable to walk or even stand. With the exception of the use of his hands, he may be absolutely helpless. The knee-jerk is at first normal, but gradually diminishes until it is finally lost. The electrical reactions are normal until marked wasting occurs, when there is a lessened response to faradism and galvanism, but never the reaction of degeneration. There are no fibrillary contractions, and no sensory disturbances. The progress of the disease is generally slow, and sometimes irregular. It is often more rapid in early childhood, and slower after puberty.

The lesions are confined to the muscles. At autopsy they appear yellow, and microscopically there is found very marked atrophy of the muscle fibres, which in places have been almost entirely replaced by fat; there may be no trace of muscle left, the structure resembling adipose tissue. In other places there is an accumulation of fat between the atrophied muscle fibres, and a very great increase of the interstitial tissue.

The prognosis is grave, most patients dying before adult life is reached. The diagnosis is generally easy from the apparent hypertrophy and actual weakness of the muscular groups. The disease is incurable.

The Juvenile Form of Muscular Atrophy.—This is much less frequent than the form just described, but, like it, begins in childhood or early youth. It is characterized by progressive wasting of certain muscular groups, especially those about the shoulders and pelvis, and hypertrophy of others. Of the shoulder and upper extremity, the muscles affected are the pectorals, the trapezius, the latissimus dorsi, the serrati, the rhomboidei, the muscles of the upper arm, and the subscapularis. The deltoid, infraspinatus and supra-spinatus for a long time escape, and may be hypertrophied. The hand and forearm are not involved. In the lower extremity, the muscles of the pelvis, thighs, and gluteal regions are affected, while those of the leg and foot escape. With this atrophy there may be associated a true or pseudo-hypertrophy of certain muscular groups. In this disease there are no fibrillary contractions, no reaction of degeneration, and no sensory disturbances. The course and result of this form are essentially the same as in the preceding variety. It is now generally regarded as closely allied to it in its pathology, the most important difference being that of localization.

There has been described, chiefly by Landouzy and Déjerine, another form of atrophy known as the *infantile facial type*. In this, wasting begins in the muscles of the face; the lips are thickened, but all the rest of the facial muscles are markedly atrophied, giving a peculiar expression to the mouth known as "the tapir mouth." Later, the atrophy extends to the shoulders and arm, but does not involve the supra-spinatus or infraspinatus, or the flexors of the hand and forearm. This is sometimes described as beginning in the shoulders, or even in the legs. The description therefore corresponds to the juvenile form of Erb, with the addition of facial symptoms, and it is probably a variety of the same disease.

CHAPTER V.

DISEASES OF THE PERIPHERAL NERVES.

MULTIPLE NEURITIS.

Under the term multiple neuritis are included those cases in which several nerves are involved in an inflammatory process, which may at times be general. In its distribution multiple neuritis is usually symmetrical, but it is not necessarily so.

Etiology.—The chief cause of multiple neuritis in children is diphtheria, although it is occasionally seen after other infectious diseases, especially malaria, typhoid or scarlet fever, and measles. In diphtheria the inflammation is due to the direct action of the toxines upon the nerve structures, since it can be induced in animals by injecting toxines into the circulation. There is little doubt that in all infectious diseases the inflammation is excited in a similar way. The metallic poisons, lead and arsenic, are rarely the cause of multiple neuritis in early life, and the same is true of alcohol, although a marked case from this cause has recently come under my observation in a child only three years old.* Lastly, there are cases in which the cause assigned is simply exposure to cold,—those classed as rheumatic.

Lesions.—Almost any nerves in the body may be affected, although the distribution varies somewhat with the cause of the disease. The musculo-spiral and the anterior tibial nerves are most frequently involved, but the inflammation may affect any of the spinal nerves, including the phrenic, and occasionally the cranial nerves, especially the pneumogastric, hypoglossal, oculomotor, and abducens. Several nerves in different parts of the body are usually affected, the lesion being in most cases symmetrical.

The affected nerve is sometimes red and swollen, owing to acute congestion and œdema or a sero-fibrinous exudation. In other cases the changes are almost entirely degenerative. The microscope shows the changes sometimes to be chiefly interstitial and sometimes chiefly parenchymatous. There is an exudation of cells into the sheath, between the sheath and the nerve fibres, and even between the nerve fibres themselves. The myeline breaks up into granules, and in places may completely disappear.

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* This case was in many respects a remarkable one. The boy completely emptied a decanter containing twelve ounces of whisky, but almost immediately vomited the greater part of it. He soon after showed the symptoms of alcoholic intoxication, and in a few hours became comatose, in which condition he continued for twelve hours. After this he gradually lost power in his legs, and at the end of a week was unable to walk at all. He had convulsions, and after this there developed the usual symptoms of meningitis at the convexity, with which he was admitted to the Babies' Hospital, December 13, 1895, three weeks after drinking the whisky. The child was then unconscious and there was present incomplete paralysis, affecting all four extremities, with anæsthesia of the arms. The active inflammatory symptoms continued for six weeks longer, during which time there were repeated convulsions, continuous stupor, fever, gradually increasing deformities, marked atrophy, loss of reflexes, and great diminution in the faradic contractility of all the paralyzed muscles; in the thighs, left leg, and abdominal muscles there were no responses to a strong current, but there was nowhere the reaction of degeneration. The child was at death's door for three or four weeks. Three months after the attack the first signs of improvement were observed in the cerebral symptoms. Shortly afterward he began to use his hands, and at the end of six weeks he was walking alone and talking freely. The improvement was very rapid, and eight weeks from the date of the first change for the better, and five months from the time of taking the whisky, he was as well as ever. The diagnosis was multiple alcoholic neuritis, with a convexity meningitis. (Fig. 169 is from a photograph taken while the symptoms were at their height.)

The late changes are those of subacute or chronic degeneration of the nerve fibres.*

With these changes in the nerves there are associated, in some cases, inflammatory and degenerative changes in the ganglion cells of the spinal cord, although they are much less severe than are the lesions in the nerves. However, they were once regarded as the explanation of some of these cases, particularly of diphtheritic paralysis.

Symptoms.—The onset of multiple neuritis is in most cases a gradual one, it being usually from two to four weeks before the paralysis reaches its height. Very exceptionally the onset may be abrupt, with fever, and marked paralysis in a few days. It is characteristic of this disease that both motor and sensory symptoms are present, and that they are the same in their distribution. The symptoms are usually symmetrical. There is first noticed a general weakness in the affected muscles, which slowly increases to complete paralysis. As the extensor groups of the hands and feet are apt to be affected, there are wrist-drop and foot-drop (Fig. 169). The paralysis may begin in the feet and hands, and gradually extend until it involves not only the four extremities, but even the muscles of the trunk and the neck, although this is rare. The child may then be absolutely helpless, unable to sit up, or even to support his head. In such cases the head seems loosely attached to the body, and rolls about on the shoulders like a ball. Weakness of the spinal muscles leads to deformities (Fig. 170), which I have seen mistaken for Pott's dis-

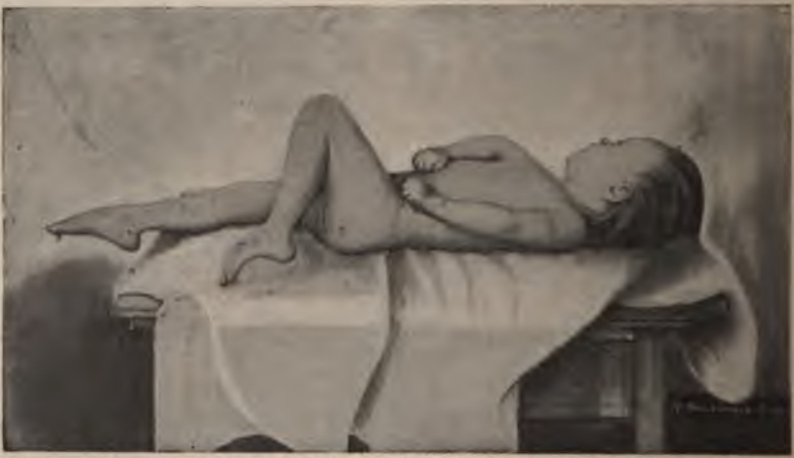

FIG. 169.—Alcoholic neuritis, showing characteristic dropping of the feet. This position of the lower extremities was maintained for over a month. Boy three years old.

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* For a full description of the lesions, consult Starr's Middleton-Goldsmith Lectures, New York Medical Record, 1887.

ease, even by experienced observers. In most of the muscular groups the paralysis is incomplete. The symptoms which relate to the phrenic and the cranial nerves will be described with Diphtheritic Paralysis, for they are rarely seen in any other form. It is characteristic of multiple neuritis that the bladder and rectum escape.

The sensory symptoms are marked only in the early stage of the disease, while the paralysis is increasing; they improve so much more rapidly than the motor symptoms, that they may be altogether wanting at the time that the paralysis is at its height. In some cases they are so slight as to be overlooked. There is usually pain along the course of the affected nerves, which is sharp and neuralgic in character, and generally associated with acute tenderness of the nerve trunks and of the muscles. Often there is a general hyperæsthesia in the early part of the attack, followed by partial anæsthesia. The sensations of touch, pain, temperature, and the muscular sense are all about equally affected.

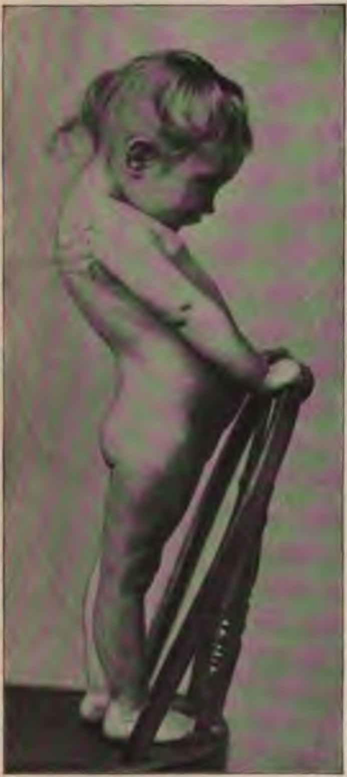

FIG. 170.—Multiple neuritis after diphtheria in a child four years old. The position of the head and spine are due to partial paralysis of the trunk and neck. The legs were also affected.

Ataxia is not uncommon, and may be a more striking symptom than the loss of power. All the reflexes are diminished or lost, especially the knee-jerk, as the legs are usually most affected. Sometimes, particularly after diphtheria, there is loss of the knee-jerk, when there is no other symptom of neuritis. In the severe cases muscular tremor is frequent.

Atrophy is a prominent symptom of neuritis, and it is evident early in the disease, often being quite as rapid as in poliomyelitis. The electrical reactions are altered,—every grade of reduction in the responses being seen, from a slight diminution in the reaction to faradism to the complete reaction of degeneration. Vaso-motor symptoms, such as œdema of the affected parts, glossiness of the skin, etc., are often present. Deformities from muscular contraction occur early; they may be severe, and in some cases, permanent.

Course and Prognosis.—The usual course of the disease is for the symptoms gradually to increase for three or four weeks and then improve, sometimes rapidly, but more often slowly, the case usually going on to complete recovery in the course of a few months. Exceptionally the paralysis may be permanent. The sensory symptoms always disappear before the motor ones. Multiple neuritis may prove fatal, from paralysis of the heart or the muscles of respiration, or death may be due to asphyxia from the entrance of food or foreign bodies into the air passages, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. Death sometimes follows from complications, especially pneumonia. The electrical reactions are of much prognostic value in regard to the persistence of the paralysis. If the reaction of degeneration is present the paralysis is certain to last many months, and some muscles are sure to be permanently affected. Where there is simply a diminution in the faradic responses, even though accompanied by marked atrophy, complete recovery may be expected, although it is often slow.

Diagnosis.—The diagnostic features of multiple neuritis are the combination of motor and sensory symptoms with the same distribution, the occurrence of atrophy, and the diminution in the electrical responses, even the reaction of degeneration. The gradual onset and the wide-spread distribution of the paralysis are also characteristic. If all four extremities are paralyzed, it is altogether the probable disease; and if to this is added paralysis of the neck and spinal muscles, the diagnosis is almost certain. The facts that the paralysis is often incomplete, and that it involves parts distant from each other, are also important. Neuritis may be mistaken for poliomyelitis, for Landry's paralysis, or for Pott's paraplegia; an important diagnostic point from the last mentioned is the condition of the reflexes,—being greatly exaggerated in Pott's paraplegia, while they are diminished or lost in multiple neuritis.

Treatment.—As this disease tends in the great majority of cases to spontaneous recovery, it is difficult to estimate the value of any method of treatment. Causes, such as lead, arsenic, alcohol, and malaria, are to be sought and removed as the first step. During the acute stage the pain may be so severe as to require relief, which is best accomplished by the application of heat. In using counter-irritation care is necessary, and such active measures as cauterization should not be employed, for troublesome ulceration may follow. After the acute stage has passed, or at the end of three or four weeks, electricity should be begun, faradism being used if the muscles respond to a moderate current, otherwise galvanism. This should be continued daily until recovery. Strychnine is much used in these cases, but it is doubtful whether it has any specific influence, although as a tonic it is valuable. Other tonics, such as iron, quinine, and most of all cod-liver oil, should be given in every case. Massage is also beneficial. The special treatment of cardiac and respiratory paralysis will be discussed in the following article.

DIPHTHERITIC PARALYSIS.

This is not only the most frequent variety of multiple neuritis, but it has some peculiarities which make a separate consideration of it desirable.

Frequency.—According to the statistics of various observers, paralysis, including all varieties, occurs after diphtheria in from 5 to 15 per cent of the cases. Sanné gives 11 per cent in 2,448 cases; Lennox Browne, 14 per cent in 1,000 cases; the Report of the Collective Investigation by the American Pædiatric Society, 9.7 per cent of 3,384 cases which were treated by antitoxine.

It is difficult to state to what degree the frequency of paralytic sequelæ after diphtheria is affected by the antitoxine treatment; but the figures above given would indicate that the protective power of the serum over the nervous tissues is not so great as over others, and that unless administered very early it may have little or no influence.

Being one of the direct effects of the diphtheria toxine, neuritis is much more likely to follow severe than mild cases; however, its occurrence after some very mild attacks shows how great is the susceptibility of the nervous tissues to the action of this poison. Sometimes the throat symptoms have been entirely overlooked, and the development of paralysis has been the first thing to arouse a suspicion of previous diphtheria.

Time of Occurrence.—During the second week, and sometimes even during the latter part of the first week, the early paralysis occurs, affecting the palate, and in some cases the heart. The most frequent and most characteristic paralysis—that affecting the throat, eyes, extremities, heart, or respiration—begins at a later period, usually from one to three weeks after the throat has cleared off, and sometimes even later than this.

Extent and Distribution of the Paralysis.—Ross * gives the following statistics of 171 collected cases of diphtheritic paralysis: Palate affected in 128; eyes in 77, in 54 of which the muscles of accommodation were involved; lower extremities in 113; upper extremities in 60; trunk or neck in 58; muscles of respiration in 33. I do not think this represents the actual frequency of the different varieties so truly as do the American Pædiatric Society's figures, which give the forms of paralysis noted in a series of cases collected for another purpose. In 328 cases of paralysis, the variety was mentioned in 189: in 124 the throat was affected; in 22 the extremities; in 11 the eyes; in 5 the muscles of respiration; in 32 the heart; in 1 the neck only; in 8 the paralysis was "general."

Symptoms.—In the great majority of cases the throat is affected, and usually the paralysis is first noticed there. It may involve the palate alone, or the muscles of the pharynx or larynx in addition. The muscles of the extremities or of the eye are often next attacked. In severe cases there may also be involved the muscles of the trunk and neck, and sometimes the diaphragm. Cardiac paralysis not infrequently occurs where no other parts have been previously affected, but in nearly all the other forms, the throat symptoms have preceded. It is this which distinguishes diphtheritic paralysis from other forms of multiple neuritis. Whatever the extent or situation of the paralysis, the knee-jerk is nearly always lost. The symptoms in the extremities and the trunk do not differ from those of multiple neuritis from other causes. The throat paralysis shows itself by a nasal voice and by regurgitation of fluids through the nose, sometimes by difficulty in swallowing or the entrance of food into the larynx, owing to anæsthesia of the epiglottis and paralysis of the muscles of deglutition. There may be difficulty in protruding the tongue or in articulation. Paralysis of the vocal cords may cause hoarseness, aphonia, or attacks of spasmodic dyspnœa. Facial paralysis is very rare. On the part of the eye there is most frequently seen inability to read, owing to paralysis of the muscles of accommodation; there may be dilatation of the pupils, rarely strabismus or ptosis.

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* The Medical Chronicle, December, 1890.

Next to that of the throat, paralysis of the muscles of respiration and the heart are the most characteristic forms of diphtheritic neuritis. Respiratory paralysis may be due to involvement of the phrenic or the intercostal nerves, more frequently the former. Extensive paralysis of other parts—the throat, extremities, or trunk—usually precedes. The first warning is generally in the form of occasional attacks of dyspnœa, sometimes accompanied by cough. Gradually these attacks increase in frequency and severity. The voice is reduced to a whisper. As the diaphragm is usually affected, the breathing is entirely thoracic. The respiratory movements are rapid, but irregular, shallow, and ineffectual. There is cyanosis, also great subjective as well as objective dyspnœa. The anxiety, distress, and apprehension of the patient are sometimes terrible. There is a constant dread of impending suffocation, and the respiratory movements are continued only by the patient's constant efforts, otherwise they may cease altogether. After a few hours these severe symptoms may subside, to return after a short respite. There may be several such attacks during two or three days, in each of which death seems imminent. Unfortunately, this is the most frequent termination. Of thirty-three such cases collected by Ross, only eight recovered. Associated with these respiratory symptoms others may be present, indicating that the pneumogastric is involved. There may be attacks of abdominal pain, vomiting, and disturbance of the heart's action,—usually an irregular or intermittent pulse, which may be either unnaturally slow or very rapid. In many cases the heart continues to beat normally, even though the respiration is so much disturbed.

The premonitory symptoms of cardiac paralysis are an irregular or intermittent pulse, often slow, but becoming very rapid from even the slightest exertion. It is always weak and compressible. The first sound of the heart is feeble and may be reduplicated. As the symptoms increase there are marked pallor, coldness of the extremities, great restlessness, anxiety, precordial distress, and perhaps orthopnœa. Within twenty-four hours from the beginning of such symptoms death usually occurs. In other cases it may come suddenly without any warning, or with a warning so slight as to be overlooked. At such times it often follows some muscular exertion, such as getting out of bed, walking across the room, or so slight an effort as sitting up suddenly in bed. Fits of temper or other excitement have at times produced it. It is by no means certain that sudden heart paralysis is always due to a lesion of its nerves. A not less important cause is toxic myocarditis. In the cases where death occurs suddenly without premonition after some muscular effort, it is in all probability the heart muscle which is most at fault. However, in many cases the two conditions are associated.

Death from diphtheritic paralysis is usually due either to cardiac or respiratory paralysis. Of one hundred and seventy-one cases of all varieties collected by Ross, forty-five were fatal.

Treatment.—Cases of paralysis of the trunk or extremities are to be managed like others of multiple neuritis. In severe forms of throat paralysis feeding by a stomach tube should always be employed, on account of the danger of the entrance of food into the air passages. It must in most cases be continued for several days. The tube may be passed either through the mouth or the nose.

The great mortality attending paralysis of the heart and respiration shows how unsuccessful is treatment in most of the cases; still, no doubt there are instances where life may be saved by judicious treatment. In cases of threatened heart paralysis, the drug most to be depended upon is morphine, hypodermically; this should be used every two or three hours in sufficient doses to keep the patient under its influence while threatening symptoms are present. In some cases it may be advantageously combined with strychnine. The patient should be kept absolutely quiet, not even being allowed to turn in bed. In respiratory paralysis the general reliance is upon strychnine used hypodermically in full doses, and faradisation of the respiratory muscles, particularly the diaphragm; it may be used in the attacks of respiratory failure and continued while they last. Large doses of diphtheria antitoxine have in some instances appeared to benefit these cases and should be tried. In the great majority, however, the damage already done is so great that no improvement follows.

FACIAL PARALYSIS.

Peripheral paralysis of the face occurring as a result of injury inflicted during delivery has already been described (page 110). There remain to be considered here cases which arise from causes that operate at a later period. The facial nerve may be affected in any one of three situations,—after its exit from the cranium, in the bony canal, and within the cranium.

In the first situation, the principal cause of neuritis is exposure to cold (the "rheumatic" cases), but it occasionally occurs as a complication of mumps and disease of the lymph glands of this region. The nerve is affected just after it has escaped from the stylo-mastoid foramen, and all the branches given off beyond its exit are involved. There is paralysis of the muscles of the forehead, those about the eye, the cheek, nose, and mouth. The affected side of the face is smooth, there is inability to wrinkle the forehead, contract the eyebrows, close the eye completely, raise the nostril, whistle, or blow. The mouth is drawn to the healthy side (Fig. 171). If the paralysis is complete, there may be difficulty in drinking or in articulation. In partial paralysis the symptoms may not be noticeable while the face is at rest. There are no sensory symptoms. The electrical reactions resemble those of other forms of neuritis; there is diminution in the response to the faradic current, which is more or less marked according to the severity of the lesion, and there may be the reaction of degeneration.

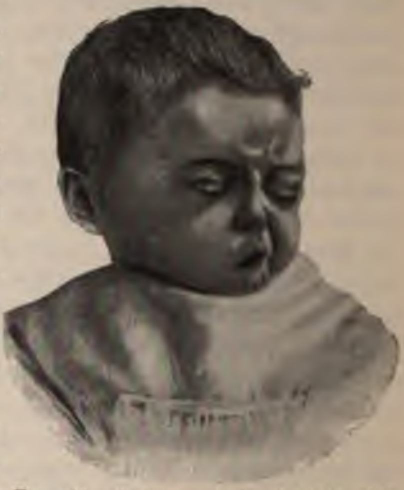


FIG. 171.—Facial paralysis of right side from middle-ear disease in a child two and a half years old.

In the bony canal, the facial nerve is usually inflamed as a result of disease of the ear. In children this is much more frequent than from the causes just mentioned. While it is possible for it to occur in acute cases, it generally accompanies chronic otitis, especially where there is caries of the petrous bone. In addition to the paralysis there is present or there is a history of a discharge from the ear, and generally there is some deafness upon the side affected. The facial symptoms are usually the same as in the cases first described. However, when the nerve is affected between the stapedius and the geniculate ganglion, there is a disturbance of the sense of taste, and of the secretion of the saliva.

At the base of the brain the trunk of the nerve may be involved in cerebral tumour, basilar meningitis, and in fracture of the skull. In any of these conditions the auditory nerve also is likely to be affected.

Prognosis.—The result is greatly modified by the causes in the different cases. In those which are due to cold, spontaneous recovery usually occurs in the course of a few weeks or months. In those depending upon disease of the ear, the outlook is not so favourable, and though there may be improvement, it is not rare for some paralysis to be permanent. In the third group of cases, facial paralysis is only one of the symptoms, and the result depends entirely upon the nature of the cause.

Diagnosis.—Facial paralysis is easily recognised. It is important to separate the peripheral paralysis from that due to a lesion above the pons, as in cases of ordinary hemiplegia. In the latter group only the lower half of the face is affected, the muscles of the forehead and those about the eye escaping, and the electrical reactions are unchanged.

Treatment.—This is essentially the same as in other cases of neuritis. In cases due to ear disease the primary lesion should receive appropriate treatment.

SECTION VIII.

DISEASES OF THE BLOOD, LYMPH NODES, SPLEEN, BONES, AND JOINTS.

CHAPTER I.

DISEASES OF THE BLOOD.

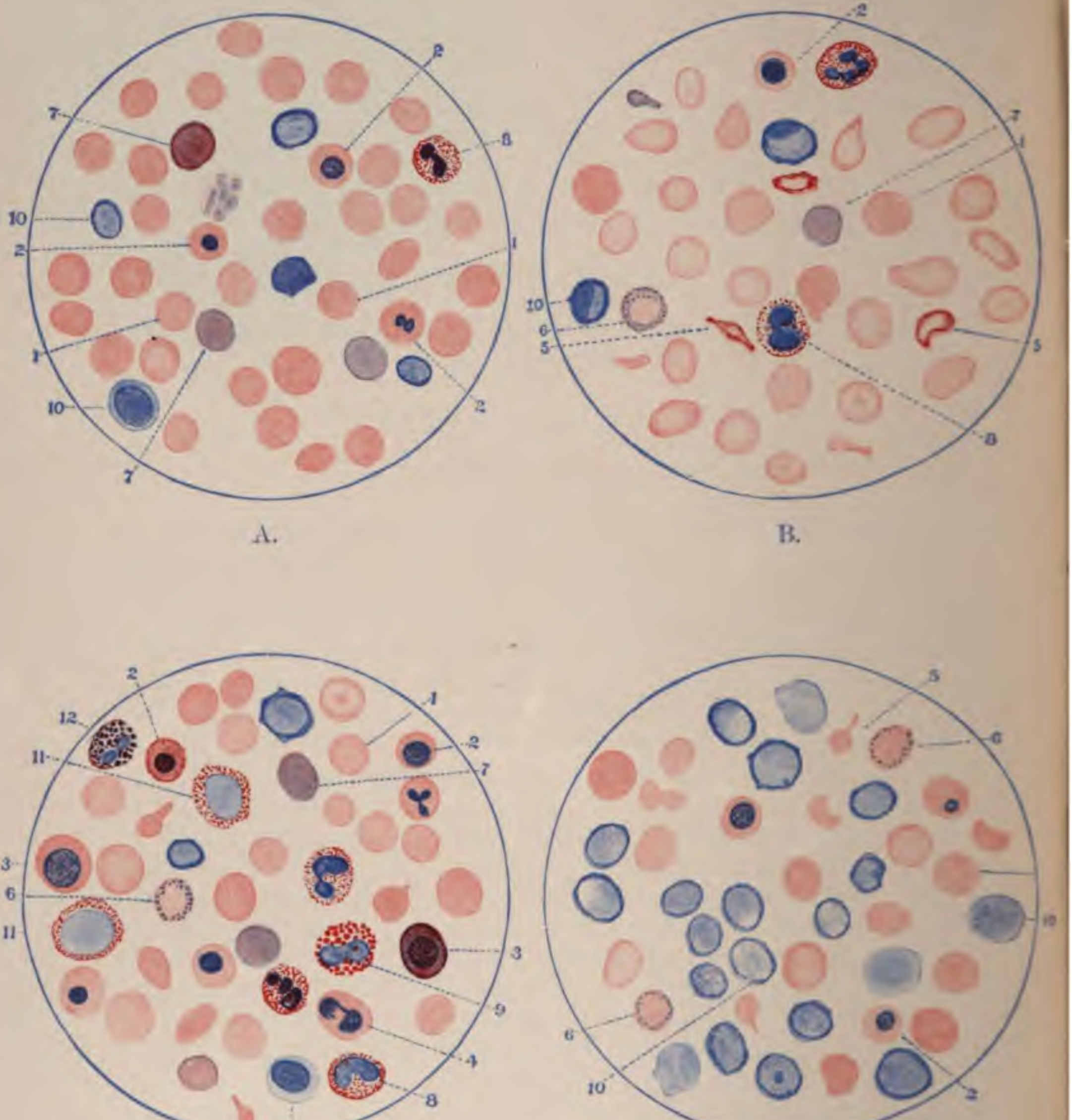
In general, the blood in infancy and childhood, as compared with that of adult life, is thinner and contains a larger proportion of water; it is also poorer in solids and has a lower specific gravity.

Specific Gravity.—This has no constant relation to the number of white or red corpuscles, but varies with the amount of hæmoglobin. The highest specific gravity is seen in the blood of the newly born. During the first two weeks of life it sinks rapidly to its lowest point, where it remains until about the end of the second year; after this time it rises gradually until about puberty. The average specific gravity during childhood is 1·050 to 1·055.

Hæmoglobin.—The percentage of hæmoglobin is highest in the blood of the newly born, and falls rapidly during the first few days after birth. Throughout childhood it is considerably lower than in adult life. The hæmoglobin is lowest between the third month and the second year; after the second year it gradually increases up to puberty. The usual range in young children, as measured by the adult standard, is between 65 and 85 per cent, 65 per cent being a low limit in healthy children.

Red Corpuscles.—The number of red corpuscles is highest in the newly born. At this time it is from 4,350,000 to 6,500,000 in each cubic millimetre. In infancy it is from 4,000,000 to 5,500,000; in later childhood, from 4,000,000 to 4,500,000 (Hayem). In size a much greater variation is seen in the red cells of the newly born than in those of older children and adults. In the blood of the fœtus there are present nucleated red corpuscles or normoblasts (Plate XV, A). These diminish in number toward the end of pregnancy. They are always found in the blood of premature infants, but in infants born at term they are seen only in small numbers and disappear after a few days. In later infancy their presence is always pathological.

PLATE XV.



A.

B.

C.

D.

Drawn by Dr. F. C. Wood.

A. Blood of an Eight-Months' Fœtus.

B. Simple Anæmia.

C. von Jaksch's Anæmia.

D. Acute Lymphatic Leukæmia.

1. Red cells, normal.
2. Red cells, normoblasts.
3. Red cells, megaloblasts.
4. Red cells, showing mitosis.
5. Red cells, poikilocytes.
6. Red cells, granular degeneration.
7. Red cells, polychromatophilia.
8. White cells, polynuclear neutrophiles.
9. White cells, eosinophiles.
10. White cells, lymphocytes.
11. White cells, myelocytes.
12. White cells, mast cells.

Normal White Cells.—According to Ehrlich, the following varieties are found in health:

1. *Lymphocytes.* These are small cells about the size of a red blood cell. The protoplasm is small in amount, forming merely a narrow rim about the nucleus; it stains with basic dyes rather more deeply than does the nucleus. The nucleus is relatively large, is centrally situated, and shows at times one or two nucleoli. The protoplasm may have a reticular structure. These cells form in adults from 22 to 25 per cent of the white corpuscles, but in children they are often as high as 50 or 60 per cent. **(Plate XV, B, 10).**

2. *Large mononuclear leucocytes and transitional forms.* These cells are two or three times the size of ordinary red cells (Plate XV, D, 10). The oval nucleus is not so centrally situated as in the lymphocytes, and stains feebly but rather darker than the protoplasm, which is feebly stained by basic dyes. The protoplasm is homogeneous and relatively large in amount.

The transitional forms occasionally contain a few feebly staining neutrophilic granules; their nuclei are bent or curved and stain more deeply.

3. *Polynuclear neutrophiles.* These are smaller than the large leucocytes (Plate XV, B and C, 8). The nucleus consists of three to four parts, usually connected by narrower portions, and stains darkly. The protoplasm stains with acid dyes and shows a great number of granules which stain only with neutral dyes. In adults these cells form about 70 per cent of the white cells; but in children they are less numerous, the increase in the lymphocytes being at the expense of the neutrophiles.

4. *Eosinophiles.* These are about the same size as the neutrophiles (Plate XV, C, 9); they have deeply staining nuclei, usually divided into two parts. The protoplasm has many large granules that stain deeply with acid dyes, and often a narrow outer layer staining more deeply than the rest. They form from 2 to 4 per cent of the total number of white cells.

5. *Mast cells.* They are only occasionally found, their proportion being about 0.5 per cent of the white cells; they are mononuclear or polynuclear cells whose granules stain only with basic dyes, not at all with tri-acid; often they are metachromatic (Plate XV, C, 12).

Pathological White Cells.—Of these there are two principal forms:

1. *Myelocytes.* They have neutrophilic granules and a single rounded nucleus (Plate XV, C, 11). Ehrlich's myelocytes differ from those of Cornil in that the cells as a whole are smaller, the nuclei are more centrally situated and stain more intensely.

2. *Mononuclear eosinophiles.* These resemble the polynuclear eosinophiles, except for the round undivided nucleus. Pathologically, the leucocytes may undergo acute or chronic degeneration, with swelling and fragmentation, nuclear changes, hydropic degeneration, etc.

The number of leucocytes in the blood of the newly born, according to Rieder, is at birth from 14,200 to 27,400; from the second to the fourth day, from 8,700 to 12,400; after the fourth day, from 12,400 to 14,800. The variations in infancy are from 9,000 to 14,000, and in later childhood from 6,000 to 12,000.

LEUCOCYTOSIS.

By leucocytosis is meant an increase in the white corpuscles of the blood. This may relate to all or any of the varieties; although it is chiefly of the polynuclear neutrophiles, there is seen in children a greater tendency than in adults to an increase in the lymphocytes.

It is customary to distinguish between physiological leucocytosis, such as that which follows a full meal, exercise, cold baths, or that which occurs in the newly-born infant, and pathological leucocytosis which occurs principally in inflammatory and toxic conditions, but may be seen also in malignant disease and after serious hæmorrhage.

Digestive leucocytosis, that which occurs after feeding, is especially pronounced in children, the increase frequently amounting to 33 per cent of the total number of leucocytes present. Leucocytosis of the newly born has already been mentioned.

Leucocytosis is present in a great variety of pathological conditions. In many of them its significance is not yet fully understood; further study of it has not fulfilled the expectations of those who had hoped to obtain from it exact information regarding many pathological processes.

The form of leucocytosis which is chiefly important in children is the inflammatory. This is most marked in acute pneumonia, diphtheria, and in inflammations attended by the formation of pus. It is also frequently present in pertussis, scarlet fever, erysipelas, acute rheumatism, septic and cerebro-spinal meningitis, and in severe forms of rickets. Of the purulent inflammations, it is especially important in appendicitis, peritonitis, empyema, pyæmia, septicæmia, osteo-myelitis, and all acute abscesses. In the conditions above mentioned the increase is chiefly or exclusively in the polynuclear neutrophiles.

There are other conditions, especially hereditary syphilis, scurvy, and certain diseases of the spleen, in which the proportion of the lymphocytes may be increased; but under these circumstances the other white cells are generally diminished.

The eosinophiles are principally increased in leukæmia; but an increase may also be present with intestinal parasites, especially tapeworm (Buckler), and in some forms of chronic skin disease. As a rule, leucocytosis is absent in typhoid fever, measles, malaria, influenza, and in tuberculous inflammations. D'Orlandi found it wanting in twenty cases of gastro-enteritis in infants.

Leucocytosis may be regarded as the reaction of the organism to the toxins in the blood elaborated by the bacteria concerned in the inflammation or infection, or to the bacteria themselves. It thus depends upon two factors: the severity of the infection, and the amount of resistance of the individual, the latter being the more important. A severe infection with a high degree of resistance produces the most marked leucocytosis, while with very feeble resistance and the same infection the leucocytosis would be slight or possibly absent.

The degree of leucocytosis is also influenced by the nature of the inflammatory process, it being less marked in serous inflammations, more pronounced in suppurative processes. In inflammations it is usually greatest during the active stage of exudation.

The Diagnostic Value of Leucocytosis.—The following are the principal diseases in which a leucocyte count may be of clinical assistance:

Appendicitis.—A marked leucocytosis may assist in distinguishing suppurative from catarrhal appendicitis, and usually points to the existence of an abscess.

Pneumonia.—A marked leucocytosis is a characteristic feature of this disease; the exceptions are very mild cases or very severe infection with little or no reaction. The increase begins shortly after the onset and continues during the stage of exudation, generally reaching its maximum shortly before the crisis, when it declines rapidly. The usual number of white cells in an average case of pneumonia in a young child is from 15,000 to 30,000, but it is not rare for the count to run up to 40,000 or even 50,000. I have seen it over 100,000. The absence of leucocytosis in a strong child who is acutely ill is always strong presumptive evidence against pneumonia. A well-marked leucocytosis is of much value in differentiating pneumonia from typhoid fever, tuberculosis, influenza, and bronchitis.

Empyema.—A rapid increase in the leucocytes in the active stage of a pneumonia or after defervescence, in the absence of physical signs pointing to an extension of the pneumonic process, almost invariably indicates empyema.

Typhoid Fever.—Leucocytosis is regularly absent in typhoid; its presence in an undoubted case indicates complications.

Pertussis.—Leucocytosis is of considerable value in the diagnosis of this disease; it is considered in that connection.

Meningitis.—As a rule, leucocytosis is present in acute simple and cerebro-spinal meningitis; in tuberculous meningitis it is not constant, and if present is generally less marked than in the other forms.

Tuberculosis.—Leucocytosis is regularly absent in unmixed tuberculous infections.

In surgical diseases the presence of leucocytosis is considered a reliable guide as to the existence of acute suppuration, although not always as to its degree. An increasing leucocytosis is usually an indication for operative interference in cases where operation is admissible. This applies particularly to appendicitis.

The Prognostic Value of Leucocytosis.—As the leucocyte count depends largely upon the resistance of the individual, it is generally true that in the diseases usually accompanied by leucocytosis a high count is a favourable sign. This is generally the case in pneumonia, unless the attack is a very mild one. On the other hand, in a severe attack a low count is very unfavourable. The following case may be cited in illustration: A delicate child, twelve months old, on the eleventh day of a severe lobar pneumonia had 24,500 leucocytes. Two days afterward a critical fall in the temperature occurred and resolution followed. The same child two weeks later was attacked with pneumonia in the opposite lung. On the second day the leucocyte count was 18,000; on the fourth day, 9,900; on the sixth day, 7,300. Death occurred the following night.

The value of the leucocyte count in diphtheria and its bearing upon prognosis are discussed under that disease.

SIMPLE ANÆMIA.

This consists in an impoverishment of the blood, especially the red cells, and a corresponding diminution in the specific gravity and in the amount of hæmoglobin. It is essentially a secondary anæmia, and occurs apart from disease of the blood-making organs. Infancy and childhood are themselves strong predisposing causes of anæmia, on account of the great demands made upon the blood in the rapid growth of the body.

Etiology.—The causes of anæmia embrace a wide range of pathological conditions. A child born of a delicate mother or of one suffering from tuberculosis or syphilis may show marked anæmia at birth. It may follow any severe hæmorrhage or occur in any of the blood dyscrasiæ, purpura, scurvy, etc.; also, loss of albumin from the blood as in prolonged suppuration, chronic nephritis, large serous effusions, many forms of diarrhœa and in malignant disease. Anæmia is often of toxic origin, sometimes being due to mineral poisons—lead, mercury or potassium chlorate; more frequently it arises from auto-intoxication, the result of absorption of the products of intestinal putrefaction. Certain of the specific infections, notably diphtheria, malaria, tuberculosis and rheumatism, produce a marked degree of anæmia, as one of their effects; also some of the intestinal parasites, particularly varieties of the tapeworm.

Much more frequent in young children than any of the above are the anæmias due to improper feeding and unhygienic surroundings. How important these causes are and how severe a grade of anæmia may be produced by them, is not usually appreciated. The physician is often led to suspect some serious organic or constitutional disease where none exists and to overlook such common conditions and obvious causes as those mentioned. Anæmia is seen where lactation is unduly prolonged. It is a frequent result of the long-continued use of milk or infant foods as the sole diet, given, as these often are, throughout the second or third year, for the reason that the child will take no solid food, because he is allowed to have the bottle. Lack of fresh air, confinement to overheated rooms and the crowding of young children in hospitals and institutions are common and important causes of anæmia.

Symptoms.—Anæmic children usually exhibit many symptoms of malnutrition. Their tissues are flabby; they are generally below average weight and suffer from digestive disturbances and chronic constipation. The associated nervous symptoms are many: headaches, indefinite pains, insomnia or disturbed sleep, general irritability and a high degree of nervousness, often ending in chorea. There is easy fatigue, shortness of breath on exertion, and sometimes fainting attacks. The peripheral circulation is poor; hands and feet, often cold. The pulse may be slightly irregular. Anæmic murmurs are heard over the base of the heart or the large vessels, and may be so loud even in infancy as to be mistaken for organic disease. A venous hum is sometimes heard in the neck. Epistaxis is not uncommon. The urine is scanty, sometimes pale, and frequently contains an excess of uric acid. There may be enuresis. Œdema is rare in older children, but in severe anæmias of infancy it is often marked. In a certain number of cases, even of moderate severity, the spleen is much enlarged. Pallor of the skin and mucous membranes is present in most cases, but is not an accurate guide as to the degree of anæmia. This can only be determined by an examination of the blood.

The Blood.—There is a reduction of the number of red cells and to a still greater degree in the hæmoglobin. In a case of moderate severity the red cells are from 4,000,000 to 4,500,000, and the hæmoglobin from 50 to 60 per cent. In severe cases the red cells may fall to 2,000,000 or 2,500,000 or even lower, and the hæmoglobin to 20 or 30 per cent. These figures are not uncommon. The lowest I have seen is a reduction of the hæmoglobin to 15 per cent and of the red cells to 1,400,000. The red cells are pale. There is usually poikilocytosis; and, especially in infancy, a few normoblasts and megalocytes may be found (Plate XV, B).

There is generally a slight leucocytosis. The differential count of the white cells shows an increase in the lymphocytes, chiefly the small variety; the polynuclear cells are relatively reduced in number.

Prognosis.—The course and termination of anæmia depend upon its cause. If this is one that can be removed, as in cases depending upon improper feeding and surroundings, very rapid improvement often takes place and prompt recovery. In the most severe cases death may occur, rarely from the anæmia, usually from some complicating disease.

In making a prognosis in a given case the general symptoms and the cause of the anæmia are much more important than the examination of the blood. If the digestive organs are in good condition and good surroundings can be secured, often, though the hæmoglobin and red cells are very greatly reduced, the prognosis is good. But in unfavourable surroundings and with a greatly disordered digestion, the outlook is much more serious.

Typical blood examinations of a moderate and of a severe case of secondary anæmia in a young child are as follows:

SEVERE ANÆMIA.		MODERATE ANÆMIA.	
Hæmoglobin	20 per cent.	Hæmoglobin	50 per cent.
Red blood cells	2,500,000	Red blood cells	4,000,000
White cells	12,000	White cells	10,000
Polynuclear	30 per cent.	Polynuclear	40 per cent.
Small mononuclear	45 per cent.	Small mononuclear	25 per cent.
Large mononuclear	25 per cent.	Large mononuclear	20 per cent.
Other forms	5 per cent.	Other forms	5 per cent.

The treatment of all the forms of anæmia will be considered together at the close of the chapter.

CHLOROSIS.

Chlorosis is a primary or essential anæmia which usually occurs in young girls about the time of puberty. It is characterized by a peculiar greenish-yellow tint of the skin, and is not accompanied by emaciation. The changes in the blood consist in a very great reduction in the hæmoglobin without a corresponding diminution in the red corpuscles.

Etiology.—The exact cause of chlorosis is not yet understood. The disease rarely occurs in males; it is usually seen in girls between the fourteenth and seventeenth years, and more often in blondes than in brunettes. Heredity appears to be a factor in some cases. Other causes are occupations deleterious to health, such as employment in factories or confinement in ill-ventilated rooms; insufficient food or clothing; psychical disturbances, like grief, care, or fright; excessive mental or physical strain; and disorders of menstruation—although the latter are perhaps more frequently a result than a cause of the disease. Virchow first called attention to the fact that chlorosis might depend upon a congenital narrowing of the aorta, sometimes associated with a small heart. It is difficult to reconcile this etiology with the rapid recovery under appropriate treatment which is seen in most of the cases. Andrew Clark has advanced the view that the chief cause of chlorosis is constipation and the resulting absorption of toxic materials from the intestine.

Lesions.—Chlorosis is rarely fatal. In the few fatal cases the lesions noted have been dilatation of the right heart with hypertrophy of the left ventricle, a small aorta, small uterus and ovaries, and occasionally round ulcer of the stomach. Under the microscope there may be found a very marked degree of fatty degeneration of the heart muscle, and sometimes of the inner coat of the blood-vessels.

Symptoms.—The general symptoms of chlorosis are very much like those of simple anæmia. There are observed shortness of breath upon exercise, palpitation, syncope, attacks of vertigo, disturbances of digestion, amenorrhœa, and almost invariably constipation. The appetite is capricious, it being a peculiarity of these patients to crave all sorts of indigestible articles. Instead of the usual pallor of anæmia, the skin has a yellowish-green tint, from which the term "green-sickness" has arisen. Occasionally patches of pigmentation are seen. Anæmic cardiac murmurs may be heard in various situations, most frequently a systolic murmur at the base of the heart, and usually loudest over the pulmonic area. There may be a venous hum in the neck. In some marked cases there is evidence of slight cardiac dilatation, especially of the right heart, and there may be hypertrophy of the left ventricle. The pulse is weak and soft, œdema of the feet is frequent, and sometimes there is slight albuminuria. In some cases there is fever. Nervous disturbances, such as vague, indefinite pains, attacks of migraine, supra-orbital neuralgia, various hysterical manifestations, and chorea, are common. Ulcer of the stomach is sometimes seen as a complication.

The blood.—The specific gravity is reduced in proportion to the loss of hæmoglobin. The characteristic feature of chlorosis is a loss of hæmoglobin which is out of proportion to the reduction in the red cells. The hæmoglobin in an ordinary case is frequently as low as 35 or 40 per cent, while the red cells may be 3,500,000 to 4,000,000, or even higher.

Morphologically the cells are pale with a wide central clear area. Poikilocytosis may be present, but is not marked; rarely normoblasts may be found. The presence of megalocytes is disputed. The leucocytes are usually unchanged in number and proportion, but the lymphocytes may be relatively increased.

Prognosis.—The course of the disease is essentially a chronic one, often lasting for a year. Relapses are quite frequent. Except when dependent upon congenital malformations of the heart and blood-vessels, these cases regularly recover when proper treatment can be carried out. A small number prove fatal by the development of tuberculosis or the occurrence of gastric ulcer.

Diagnosis.—The diagnosis is in most cases easily made from the etiology, the functional derangement of the heart, the colour of the skin, and a positive diagnosis always by an examination of the blood.

PSEUDO-LEUKÆMIC ANÆMIA OF INFANCY.

This form of anæmia was first described by Von Jaksch in 1889, and is by him believed to be peculiar to infants and young children. It is characterized by marked leucocytosis, marked reduction in the number of red cells and in the hæmoglobin, a great enlargement of the spleen, and sometimes a moderate enlargement of the liver and the lymphatic glands. This disease is not to be confounded with the pseudo-leukæmia of adults, or Hodgkin's disease, which is purely a disease of the lymphatic glands with secondary anæmia, but without any leucocytosis.

The existence of pseudo-leukæmic anæmia as a distinct disease is denied by several authorities on diseases of the blood, who maintain that all such cases are to be classed as secondary anæmia, pernicious anæmia, or leukæmia.

Etiology.—Of the cases thus far recorded the majority have been between the ages of seven and twelve months. Of twenty cases collected by Monti and Berggrün, sixteen showed evidences of rickets and one was syphilitic. The exact cause of the disease is still unknown, and its essential nature is a matter of some doubt. Monti believes that it may develop from the more severe cases of anæmia which are accompanied by leucocytosis, as he has observed this condition before the development of pseudo-leukæmia and during its subsidence.

Lesions.—The most characteristic change is found in the spleen, which is very much enlarged, often forming an abdominal tumour of considerable size. It is firm, hard, and there may be evidences of perisplenitis. The microscope shows a simple hyperplasia. Enlargement of the liver is less constant, it being normal in more than half the cases. There is no relation between the size of the spleen and that of the liver. The hepatic cells are unchanged. Enlargement of the lymph glands has been noted in about half the reported cases, the swelling affecting the cervical, axillary, or inguinal glands; but it is rarely great. Changes in the bone-marrow have been described by Luzet, these being usually most marked about the epiphyses.

Symptoms.—*The blood.*—The number of reported cases is as yet too small to make positive statements possible upon all points. The main features noted thus far are the following (Plate XV, C):

The specific gravity is lowered, the usual range being between 1·035 and 1·044. The reduction of the hæmoglobin is very great; in many of the cases it has been as low as 30 per cent, and in a few below 25 per cent.

The red cells are always diminished; in 6 of 20 cases they were below 1,600,000 (Monti and Berggrün). There is also great inequality in their size and shape. Nucleated red cells are found in considerable numbers; as a rule, these are chiefly normoblasts, but when the anæmia becomes more severe, it is usually the megaloblasts that predominate. The leucocytes vary from 20,000 to 50,000. They may show an increase in the mononuclear or in the polynuclear forms. The eosinophiles are usually increased, but not to the extent to suggest leukæmia. All varieties of cell degeneration are found.

The general symptoms of the disease develop slowly and with the usual signs of anæmia. In some cases the infants continue to be plump and well nourished. Pallor is usually very marked. Enlargement of the spleen is so great that it can hardly be overlooked if the abdomen is examined. The glandular enlargements are not marked, and in many cases are wanting altogether.

The course of the disease is essentially chronic. Cases have been seen in which pseudo-leukæmia developed from an ordinary severe simple anæmia in the course of a few weeks. The symptoms and blood changes generally come on slowly in the course of weeks or months, and sometimes remain nearly stationary for as long a period as several months, and then slowly improve. In other cases they grow gradually worse. In the cases going on to recovery, there is noticed improvement in the general symptoms coincident with a diminution in the size of the spleen, a reduction in the number of leucocytes, an increase in the red cells, the hæmoglobin, and the specific gravity, and a gradual disappearance of the nucleated red cells.

Prognosis.—In Monti's list of twenty cases four proved fatal; one recovered, in which the proportion of leucocytes to the red cells had been 1 to 12. The prognosis should be guarded, for, although improvement may take place, many patients die from intercurrent disease.

PERNICIOUS ANÆMIA.

This is the most severe form of anæmia known. Its cause and essential nature are as yet very imperfectly understood. It is characterized by quite uniform blood changes and by the general symptoms of a very marked anæmia, and it tends to go on from bad to worse, terminating fatally in the great proportion of cases.

Etiology.—Pernicious anæmia is a rare disease in childhood, and especially rare in infancy. In the cases which have been observed in early life the following etiological factors have been noted: It has been associated with hereditary syphilis and with severe rickets, especially when accompanied by a marked enlargement of the spleen. It has followed other diseases, especially grave disturbances of nutrition. Sometimes simple anæmia, when severe and of long standing, has gradually developed into the pernicious type. In a few instances parasites, particularly tapeworms, have been the cause. Pernicious anæmia has in some instances occurred in patients where no cause whatever could be assigned.

Many theories have been advanced in explanation of pernicious anæmia. The one which at present appears to have most in its favour is that the disease consists in a great destruction of the red blood-cells, particularly in the liver, and that this is brought about through the agency of some poison or poisons taken up from the intestine by the portal circulation. This has been advanced by Hunter and others in explanation of the peculiar deposit of iron found in the hepatic cells.

Lesions.—There is found a very high grade of anæmia in all the internal organs, fatty degeneration of the heart and blood-vessels, and sometimes also of the liver and kidneys, with numerous capillary hæmorrhages in the various organs. The most characteristic post-mortem change, however, according to Hunter, consists in the deposit of iron in the hepatic cells. Its distribution is peculiar and unlike that seen in any other disease.

Symptoms.—*The Blood.*—The specific gravity of the blood in pernicious anæmia is constantly and considerably reduced, and its coagulability is feeblė. The hæmoglobin is always reduced, usually it is as low as from 20 to 30 per cent. The red cells are always much diminished in number and generally to a degree greater than the reduction in the hæmoglobin. Their number is seldom greater than 2,000,000, and frequently less than 1,000,000. Megalocytes are present, often in great numbers, and a preponderance of them is regarded essential to the diagnosis. Microcytes are rare. It is characteristic of pernicious anæmia that owing to the relatively high hæmoglobin the red cells stain well, usually deeper than in normal blood. A striking feature of these cases is the presence of extreme poikilocytosis. Nucleated red cells are also present, megaloblasts in greater numbers than normoblasts. The red cells do not collect to form rouleaux.

The total number of leucocytes is markedly diminished, but the lymphocytes may be relatively increased. An occasional myelocyte may be found.

The general symptoms are those of a most intense anæmia. There is marked pallor of the skin and mucous membranes, with great weakness and prostration. Various anæmic heart murmurs are heard. There is dyspnœa, and usually the urine is scanty and of low specific gravity. There may or may not be emaciation. The late symptoms are hæmorrhages from the nose and other mucous membranes, subcutaneous ecchymoses with dropsy of the feet and ankles, and sometimes of the large serous cavities of the body, but without albuminuria. In many cases fever is present. This may be so high as to lead to the suspicion of some acute infectious process.

The course of the disease is, as a rule, more rapid than in adults, the duration being in most cases several months; it is marked by periods of exacerbation and remission. During the exacerbations all the symptoms are intensified, and as a rule some fever is present. During the remissions marked improvement may take place in all the symptoms and an increase in the hæmoglobin and red cells occur. In general, the progress of the disease is downward and sometimes the loss is very rapid. The only exceptions are the cases in which the disease depends upon some intestinal parasite, where improvement and even recovery may occur.

Treatment of the Different Forms of Anæmia.—In *secondary anæmia* the thing of the first importance is to discover and treat the primary condition upon which the anæmia depends. In infancy, special attention should be given to diet and hygiene, particularly with reference to an abundant supply of fresh air. The whole manner of life of these patients must be carefully studied and managed according to the directions laid down in the chapter upon Malnutrition, with which condition, especially in infancy, a very large number of these cases are associated. The general treatment referred to is often more important than the administration of the preparations of iron, which, however, should never be omitted.

The preparations of iron available for infants are the albuminate, pepto-manganate, ovoferrin, hæmaboloids, bitter wine, sweet wine, saccharated carbonate, malate, and citrate. The dose should be regulated according to the age of the child. Older children may take the same preparations as adults, especially reduced iron and Blaud's pills. Much benefit is seen from combining arsenic with iron, or from alternating the two. Arsenic should be used in conjunction with iron when there is enlargement of the spleen or lymphatic glands. In addition to these remedies, cod-liver oil should be given if the condition of the digestive organs will permit.

In *chlorosis* more decided results are seen from the use of iron than in any other form of anæmia. Blaud's pills are here the favourite method of administration, and are advantageously combined with small doses of nux vomica and aloin to overcome the tendency to constipation. Arsenic is useful in these cases also. Great benefit in chlorosis results from change of air and change of scene, thus removing the patient from all sources of nervous excitement or disturbance. The general condition, diet, and habits of life should also receive careful attention, particularly the condition of the bowels.

Oxygen is a valuable adjuvant in the treatment of all anæmias not yielding to iron alone. It is important that the administration of iron should be continued for several months after the disappearance of all symptoms, on account of the tendency to relapse.

In the *pseudo-leukæmic anæmia* of infants, arsenic is decidedly the most valuable drug, but should be given in combination with iron. Fowler's solution is the best preparation for infants; the dose should rarely be more than one drop, which should be repeated four or five times daily after feeding, and continued for a long time. The general treatment of these patients is the same as in cases of simple anæmia. When rickets is present cod-liver oil and phosphorus should be added.

In *pernicious anæmia,* arsenic offers a much better prospect of improvement than iron. Beginning with small doses, the amount should be gradually increased up to the point of tolerance, very much as in cases of chorea.

In every case of anæmia the most careful attention should be given to the general condition, particularly guarding against exposure to cold and dampness. The feeble circulation of these patients renders them peculiarly susceptible. Caution should also be given against much muscular exercise. With a severe grade of anæmia very active exercise should be prohibited, and many of these patients do best when complete rest in bed, either for the entire time or for a considerable part of each day, is insisted upon. This applies to children of all ages.

LEUKÆMIA.

This is a disease in which the essential feature is a great increase in the number of leucocytes, with a moderate reduction in the number of red corpuscles, and the presence in the blood of cellular forms not found in health.

Etiology.—Leukæmia is a rare disease in childhood, but has been seen even in early infancy. Its greater frequency in males holds good even in childhood. In a small number of cases heredity seems of some importance as an etiological factor. Leukæmia may follow syphilis, rickets, malaria, or even simple anæmia, or it may occur as a primary disease in children previously healthy. In the great majority of cases the cause is unknown.

Lesions.—The essential lesions of leukæmia are found in the spleen, the lymphatic glands, and the bone-marrow. In rare cases the most important changes are in the lymphatic glands, giving rise to the *lymphatic* form of leukæmia. In such cases the changes in the spleen or marrow may be slight or absent. Changes in the spleen and marrow are, however, usually associated, giving rise to what is known as the *splenomyelogenous* form of the disease, which is the most frequent variety. The spleen is usually enormously enlarged, sometimes filling half the abdominal cavity. In the early stage it is soft, vascular, and of a darkred colour; in the late stages it is firm and hard, and usually deeply fissured at its margin. There may be perisplenitis. On section, light-gray patches of lymphoid tissue may be seen scattered throughout the organ, and in some instances there may be wedge-shaped infarctions. The microscope shows thickening of the trabeculæ and deposits of lymphoid tissue, especially about the arteries. In the lymphatic form any of the external glands of the body may be affected, the cervical, axillary, and the inguinal, or the mesenteric, tracheo-bronchial, the tonsils, and even the lymph nodules of the tongue, pharynx, and intestines. The changes in the glands are generally those of a simple hyperplasia. The liver is enlarged in very many of the cases, chiefly from an infiltration with lymphoid tissue, which may be diffuse or may occur in patches. Less frequently similar lymphoid masses are seen in other organs.

Symptoms.—*The blood* (Plate XV, D).—The colour is lighter than normal and its coagulability usually diminished. Generally the red cells are much reduced in number, although not to the extent seen in pernicious anæmia. The most important feature is the great increase in the leucocytes, which vary in form according as the type is spleno-myelogenous or lymphatic. The red cells are usually of normal size and a moderate number of normoblasts is found; the hæmoglobin is diminished.

In the spleno-myelogenous form the white cells may be from 100,000 to 500,000, but, especially under the influence of arsenic, a marked temporary diminution may occur, so that their number may be scarcely above the normal; both Ehrlich's and Cornil's myelocytes are present, and the presence of a large number of these is pathognomonic. The number of polynuclear neutrophiles is greatly increased, although their proportion is diminished. The eosinophiles are very much increased in number, mononuclear forms being present. The number of lymphocytes is increased, but they vary according to the type and stage of the disease; this is true also of the large mononuclear leucocytes. Mast cells are much increased in number, this being the most reliable diagnostic sign.

In the lymphatic form the lymphocytes alone are increased, so that the other white cells are relatively diminished. The increase is usually in the small lymphocytes which form from 80 to 90 per cent of the leucocytes present. Myelocytes and mast cells are either present in small numbers or absent altogether.

The other symptoms of leukæmia in children resemble those in adults, with the difference that, as a rule, the progress of the disease is much more rapid in early life. In most of the cases the early symptoms are latent. A sudden and alarming hæmorrhage is sometimes the first thing to call attention to the serious condition. In other cases there are only the symptoms of general weakness and anæmia. Sometimes the splenic tumour or the enlargement of the lymphatic glands is first noticed. In the early part of the disease, the usual symptoms of anæmia are present—digestive disturbances, shortness of breath, weak and rapid pulse. Hæmorrhages may occur as an early or late symptom; they are most frequently from the nose, but severe hæmorrhages may occur from the stomach, the mouth, the intestines, or there may be ecchymoses upon the skin. The enlargement of the spleen may be sufficiently marked to form an abdominal tumour, so as to attract the attention even of the parents. The swelling of the liver is not so great. The lymphatic glands are enlarged only to a moderate degree, and in many cases this symptom is absent altogether. They are painless, movable, and usually several groups are affected.

The late symptoms are dropsy of the feet or general anasarca, hæmorrhages, diarrhœa, headaches, general weakness, and attacks of fainting. Fever is quite constant in the late stages of the disease, and the temperature may be from 101° to 103° F. The urine may contain albumin and casts. Vision is sometimes disturbed by the formation of leukæmic plaques in the retina. It is rare that there are any symptoms referable to the bones, although expansion and tenderness of the flat bones have been observed.

Course and Prognosis.—The course of leukæmia is chronic, and in most cases slowly progressive, but not always so. The prognosis is very bad, the great proportion of the cases in children proving fatal within a year from the first symptoms, in infancy sometimes in two or three months. There has been described by Epstein and others an acute form of the disease, proving fatal in a few weeks. The usual causes of death are exhaustion, hæmorrhages, and broncho-pneumonia.

Diagnosis.—This, in children, has to be made chiefly from simple anæmia with leucocytosis, and pseudo-leukæmic anæmia. Without a blood examination this is impossible. The chief reliance is to be placed upon the enormous increase in the leucocytes, and especially upon the presence of numerous mast cells and myelocytes.

Treatment.—The general treatment of leukæmia should be the same as that of anæmia. Of the drugs now in use, arsenic has altogether the most testimony in its favour. It must be given in large doses and for a long period. Next to this in value come iron and cod-liver oil. Leukæmia, however, is in most instances very little influenced by treatment. The reported cures must be taken with some allowance, for most of these were published before the time when leukæmia was sharply differentiated from simple anæmia with leucocytosis and from the pseudo-leukæmic anæmia of infancy.

HÆMOPHILIA.

Hæmophilia is an hereditary disease, in which there is a tendency to profuse or even uncontrollable bleeding from slight wounds, or sometimes even spontaneously. In many cases there is associated an inflammation of the joints. Persons so affected are known as "bleeders."

Etiology.—The hereditary tendency of the disease is very strongly marked, and it has often been traced through seven or eight generations. Males are much more frequently affected than females, the proportion being about twelve to one. In the matter of inheritance, the disease is most often transmitted through the mother, who may, however, herself escape. Patients suffering from hæmophilia have nothing else about them that is abnormal. The exact nature of the disease is unknown. It has no connection with either purpura or scurvy. Although generally classed among the diseases of the blood, it has not been established that there are any constant changes either in the blood or in the blood-vessels.

Symptoms.—The first manifestations of hæmophilia are not often seen before the second year. The hæmorrhages of the newly born have no relation to this condition. The discovery of the disease is generally quite accidental. The first hæmorrhage may be traumatic or spontaneous. In traumatic hæmorrhages there may be very severe bleeding after so slight a wound as the drawing of a tooth; sometimes a large hæmatoma forms between the muscles as the result of a moderate contusion.

The following is the relative frequency of spontaneous hæmorrhages in 334 cases collected by Grandidier: bleeding from the nose in 169, mouth in 43, intestines in 36, stomach in 15, urethra in 16, lungs in 17. There may be hæmorrhage from the skin or from any mucous membrane of the body. The attacks of spontaneous hæmorrhage are often periodical, and may be accompanied by arthritic symptoms resembling rheumatism. The severity of the hæmorrhages varies much in the different cases. From a slight wound a patient may bleed until he is exsanguinated, and even until death occurs. Such a result from the first hæmorrhage, however, is rare. In some cases the disposition to bleed is outgrown in later life. Grandidier states that, of 152 boys, over one half died before reaching the seventh year. It is striking that when the disease affects females there is no tendency to excessive bleeding at menstruation or parturition.

Treatment.—The indications at the time of bleeding are, to arrest the hæmorrhage by the use of the ordinary surgical means—compression, styptics, etc. (For epistaxis, see page 488). Little benefit is to be expected from drugs. In convalescence after attacks of hæmorrhage, iron and general tonics should be given. In all patients who are bleeders everything which might by any means excite hæmorrhage should be avoided. Marriage should be discouraged in girls who inherit the disease.

PURPURA.

The term purpura is used to designate a condition in which there is a tendency to spontaneous hæmorrhages beneath the skin, from the various mucous membranes, and in some cases into the internal organs. The term *purpura simplex* is applied to those cases in which the hæmorrhages are limited to the skin; *purpura hæmorrhagica* to those in which there is in addition bleeding from the mucous membranes or visceral hæmorrhages. It is impossible to draw a line sharply between these two classes of cases, as the chief difference between them seems to be one of degree. Purpura is sometimes known as *morbus maculosus* or as *Werlhof's disease.*

Symptomatic Purpura.—This occurs in quite a variety of conditions, the hæmorrhages generally being limited to the skin, but not always so. These cases may be grouped in the following classes:

1. *Infectious.*—This form of purpura is very constantly seen in malignant endocarditis, in the hæmorrhagic forms of the various eruptive fevers—measles, scarlet fever, variola, vaccinia, and typhus—also in epidemic meningitis and occasionally in diphtheria, pyæmia, and septicæmia. The occurrence of hæmorrhages in these cases appears to depend upon an altered condition of the blood, which is a direct result of the infection, and it is a bad prognostic sign.

2. *Cachectic.*—Purpura occurs late in the course of many protracted and exhausting diseases, especially in infancy. It is most frequently met with in broncho-pneumonia, empyema, tuberculosis, ileo-colitis, in both the tuberculous and the simple forms of meningitis, and in malignant disease. It also occurs from apparently similar causes in several of the diseases of the blood, particularly in leukæmia and pernicious anæmia. In most cases of cachectic purpura the hæmorrhagic spots are small, not very abundant, and occur either upon the abdomen or the lower extremities. This form is quite common in hospital practice, and is almost invariably indicative of a fatal result. In cachectic purpura the hæmorrhages are usually limited to the skin. The condition is undoubtedly dependent upon a deterioration in the blood, possibly also upon the condition of the minute blood-vessels.

3. *Toxic.*—Certain drugs, such as phosphorus, quinine, potassium chlorate and sometimes others, may in rare cases produce hæmorrhages when long continued or in large doses. The hæmorrhage of jaundice may also be considered in this group.

4. *Mechanical* hæmorrhages, such as those occurring in pertussis or epilepsy, are sometimes classed with purpura. In convalescence from protracted illness there are sometimes seen, when patients first stand or walk, purpuric spots on the lower extremities. They may occur after confinement of a limb in bandages or splints. In both these cases the cause is partly mechanical and partly due to the weakened condition of the blood-vessels.

5. *Neurotic.*—These cases are occasionally seen in diseases of the spinal cord and sometimes in hysteria in young adults, but very rarely in children.

Primary Purpura.—This occurs in children of all ages, being not uncommon in infancy. Hæmorrhages of the newly born have not generally been included in this class, although there are some reasons why they might well be. The age at which primary purpura is most frequently seen is from two to ten years. The sexes are about equally affected; of Steffen's 56 cases, 27 were males and 29 females. The disease may occur in children who are cachectic, rachitic, or anæmic, and in those whose surroundings are poor, but it has not, like scurvy, any close relation to diet. It may follow any acute disease, being associated most frequently with derangements of the stomach and bowels. Quite frequently the disease develops abruptly, without any assignable cause, in children previously healthy.

Lesions.—The external hæmorrhages may occur upon any part of the body. There are smaller or larger ecchymoses or an infiltration of the tissues with blood, which undergoes gradual absorption with the usual changes. With the hæmorrhages, various forms of inflammation of the skin may be associated, especially erythema and urticaria, with sometimes more or less œdema. Hæmorrhages from the mucous membranes are more frequent, because of the feebler resistance of the tissues. There are seen ecchymoses upon the visible mucous membranes which resemble those upon the skin. At autopsy they are occasionally seen in the trachea or bronchi, but more often in the digestive tract. In the colon, and occasionally in the small intestine, ulcers may be found; but they are rarely if ever seen in the stomach. They may be superficial or deep, and have even been known to cause perforation.

Intracranial hæmorrhages are rare, and are usually meningeal. These may be sufficient to cause severe symptoms. In 1893 a case occurred in the New York Infant Asylum in an infant six months old, with an extensive meningeal hæmorrhage covering a large part of the brain. In Steffen's paper several such cases are mentioned.

Pulmonary hæmorrhages are not frequent. Ecchymoses are found beneath the pericardium; but endocarditis and pericarditis are extremely rare, probably occurring only in the rheumatic cases. The spleen is occasionally enlarged, but by no means uniformly so, and it may be the seat of hæmorrhages.

While hæmaturia is one of the most frequent of the visceral hæmorrhages, severe nephritis is rare. Acute degeneration of the renal epithelium of the tubes is quite common. There may be punctiform hæmorrhages, and occasionally larger ones beneath the capsule or in the mucous membrane of the pelvis of the kidney. The suprarenal capsules may be the seat of extensive and even fatal hæmorrhage. There may be effusions of a sero-sanguineous fluid into any of the large serous cavities, most frequently into the peritonæum. The articular lesions of purpura may be of a rheumatic character, with which purpura occurs as a complication; or there may be hæmorrhages into the tissues about the joint, or even into the joint itself—usually the knee or elbow.

Thus far no constant or essential changes have been demonstrated in the blood, other than those which are due to hæmorrhages—viz., a moderate reduction in the hæmoglobin and the red corpuscles, with occasional irregularities in size and the appearance of nucleated red cells. In the most severe cases there is a moderate degree of leucocytosis.

Pathology.—Why it is that under certain circumstances the blood-vessels will not hold their contents, it is difficult to understand. There have been described by Cassel, Riehl, Wilson, and others, changes in the small blood-vessels, usually a form of endarteritis, but it is not necessary to assume a lesion in the blood-vessels, since we know that diseased blood may pass through even normal vessels. Henoch has suggested the vaso-motor origin of purpura, in which there is first a paralytic distention of the small vessels, followed by stasis, hæmorrhage, or œdema. In certain forms, as in malignant endocarditis, it is well established that the cause is an infectious thrombosis. Although the bacteriological examinations made thus far in purpura are not numerous enough to settle the question positively, there is little doubt that infection is the essential factor in some forms of the disease, particularly in the cases characterized by sudden onset, high temperature, and cerebral symptoms, and which run a rapidly fatal course. At the present time the exact pathology of purpura is unknown. There are, no doubt, now included under this term, several diseases quite distinct from one another.

The clinical types.—1. The ordinary form.—In the mild cases the hæmorrhage is confined to the skin (purpura simplex), or it is accompanied by slight bleeding from the mucous membranes. There is usually some general indisposition of an indefinite character for a day or two before the purpuric spots are noticed; most frequently a disturbance of digestion with vomiting, diarrhœa, and sometimes slight fever. The hæmorrhages appear as small petechiæ, varying in size from a pin's head to a pea; usually first upon the lower extremities. There may be only a few widely scattered spots or the body may be covered. The colour is first a bright red, then purple, gradually fading in the course of a few days. New spots come as the old ones disappear, so that the amount of eruption may not diminish. They do not disappear upon pressure.

The course of these cases is generally favourable, recovery taking place in from one to four weeks under the influence of general tonic treatment. Relapses are, however, very frequent, and such attacks may come at intervals of a few weeks or months for a considerable period. One must be guarded in giving an absolutely favourable prognosis even in cases of such severity, for it occasionally happens that in a patient, who for several days has had symptoms of mild purpura, there suddenly develop those of the most severe type with a rapidly fatal termination.

2. The severe form.—Such cases are characterized by hæmorrhages from the mucous membranes (purpura hæmorrhagica) from the outset. These may even appear before the spots upon the skin. In severe attacks the petechial spots are more likely to appear suddenly, and large ecchymoses, varying in size from a pea to the palm of the hand, are more frequent. There may be bleeding from the nose, gums, mouth, or pharynx, and ecchymoses may be seen upon these mucous membranes, also upon the conjunctivæ. Vomiting of blood and bloody discharges from the bowels are quite frequent symptoms. The urine may contain enough blood to give it a bright-red colour. Less frequently there are seen hæm-

orrhages of the retina or choroid and from the female genitals. In one of my own cases there was almost continuous bleeding from one ear. Cutaneous ecchymoses are increased by slight injuries, such as the pressure from a bandage or from scratching. Epistaxis may be copious enough to necessitate plugging of the nares. The amount of blood vomited is not often large; its source may be the stomach, the mouth, or the pharynx. The blood in the stools is usually dark coloured, but there may be some bright-red blood even when there are no ulcers present. In one of my cases so much blood was lost by the bowels as to produce the symptoms of a very marked cerebral anæmia. In certain cases the gastro-intestinal symptoms are very prominent, and there may be slight icterus. The discharge of blood from the stomach or intestine may be accompanied by very severe attacks of colic and tenesmus. In some of these cases there are pains and slight swelling of the joints. Renal symptoms are generally present. These attacks of pain with purpura and the discharge of blood, may come on paroxysmally every few days for a period of several weeks. They have been ascribed to thrombosis of the intestinal vessels. This is sometimes known as " Henoch's purpura."

Constitutional symptoms are present in most of the severe cases. There is usually fever, from 101° to 103° F., and sufficient prostration to keep the patient in bed. If the amount of blood lost is large, there are the usual symptoms of severe anæmia. The loss of blood may be sufficient to cause death, particularly in infants. Cerebral symptoms may depend upon anæmia or upon meningeal hæmorrhage. They are not frequent in this form of the disease. Œdema, especially of the face and feet, may exist without albuminuria, and albuminuria may be present in cases in which there is no renal hæmorrhage.

In some of the cases beginning with severe general symptoms, and occasionally when the onset is mild, the patients after a few days pass into a typhoid condition with low delirium, great prostration, weak and irregular pulse, dry, cracked tongue, and high temperature. Such cases are almost always fatal. They are not to be confounded with ordinary typhoid fever complicated by purpura.

The course varies much in the different cases. It lasts from one to six weeks, the symptoms slowly subsiding, but often showing a strong tendency to recurrence. The prognosis depends upon the age of the patient, the extent of the hæmorrhage, and the presence or absence of septic symptoms.

3. The hyper-acute form (purpura fulminans).—This is a rare form, especially in young children. Its development is usually sudden with a chill, vomiting, marked prostration, and high temperature. The purpuric spots come out with great rapidity, and in the course of a few hours or a day they may be very extensive. In addition to the ordinary subcutaneous hæmorrhages, bloody vesicles may form upon the skin. In

many cases the hæmorrhages are limited to the skin, the mucous membranes and the viscera escaping altogether. There is no tendency to gangrene. Cerebral symptoms are invariably present and usually prominent; there may be delirium, dulness, stupor, and finally coma. The spleen is apt to be enlarged. The urine is nearly always albuminous. This form of purpura has all the characteristics of a general infectious disease, and it is almost invariably fatal.

4. The gangrenous form.—Sloughing is not common in purpura, but it is most often seen in the mucous membranes. Osler refers to two cases affecting the uvula. I once saw a slough which caused perforation of the soft palate. Wickham Legg reports a case with gangrene of the prepuce. Gangrene of the skin is even less frequent, although cases have been reported even in young children. Charron's case was only three years old, and several others in children are collected in Gimard's monograph upon this subject. The gangrene may involve the skin only, or the subcutaneous tissues and even the muscles. It has been seen upon the upper and lower extremities and even upon the face, and may extend over quite a large surface. In some of the milder forms of purpura, gangrene results from some slight injury, such as a blow, the pressure from a bandage, or in the nose, from the pressure of a tampon. These cases are almost invariably fatal. Those in which the sloughing is confined to small areas of the mucous membrane of the mouth often recover.

5. The rheumatic form.—The term "rheumatic purpura" (peliosis rheumatica) is applied to cases, not so common in children as in older patients, in which subcutaneous hæmorrhages, and sometimes bleeding from the mucous membranes, are associated with painful joint swellings. These are to be regarded as cases of rheumatism complicated by purpura. The joints most frequently affected are the knee and the ankle. The arthritic symptoms are usually less severe than in attacks of acute rheumatism. There may be present erythema exudativum or erythema nodosum or urticaria. Usually there are throat symptoms and fever, and frequently œdema of the face and eyelids with albuminuria. The spleen may be enlarged. The usual duration is from one to three weeks, and although relapses may occur, the cases usually recover.

Joint symptoms, particularly articular pains, are not infrequent in the course of milder attacks of purpura without the febrile symptoms mentioned. In severe cases extravasations of blood have been reported as occurring in the tissues about the joints, and even in the joints themselves, these being cases of true arthritic purpura. It is probable that, in the past, some cases of scurvy have been included in this group.

Diagnosis.—The rapid acute cases may be confounded with the hæmorrhagic forms of the various eruptive fevers. The ordinary subacute or passive forms are chiefly to be differentiated from scurvy. The diagnosis is not difficult and the mistake need not be made if the essential features of scurvy are borne in mind—its dietetic cause, bleeding gums, hyperæsthesia, and deep rather than subcutaneous hæmorrhages which are usually near the joints.

Prognosis.—This depends very much upon the form of the disease. Of 128 cases of all varieties occurring in children in Steffen's collection, there were 40 deaths. In 12 cases of severe primary purpura reported by Gimard, there were 3 deaths and 9 recoveries. Purpura simplex is rarely fatal; cases of purpura hæmorrhagica usually recover unless marked febrile symptoms are present. The forms classed as typhoid, gangrenous, and purpura fulminans are almost invariably fatal. The tendency to relapses exists in all varieties.

Treatment.—The treatment of symptomatic purpura should have reference to the cause of the disease. The mild cases of primary purpura usually recover promptly under a tonic plan of treatment. The more severe cases require confinement in bed, absolute quiet, and care to avoid exposure and even the slightest injury or extra pressure upon any part. Drugs do not seem greatly to influence the course of the disease. Those most frequently employed are supra-renal extract, hydrastis, hamamelis, aromatic sulphuric acid, the vegetable acids, ergot, and gallic acid. Whether or not it is true, as claimed by some, that all hæmorrhagic diseases are related to scurvy, the striking improvement seen in this disease from the use of fresh fruit and vegetables, suggests their employment in purpura. In some cases very decided benefit seems to follow their use in the acute stage, but more particularly in convalescence. For hyperacute and gangrenous cases, little can be done except to treat the symptoms. Surgical means of arresting the hæmorrhage are rarely successful. Iron and arsenic and alcoholic stimulants should be used in all cases during convalescence.

CHAPTER II.

DISEASES OF THE LYMPH NODES (LYMPHATIC GLANDS).

It is characteristic of infancy and childhood that the lymphoid tissues—tonsils, adenoids, external and internal lymph glands, and many smaller lymph nodules throughout the body—are prone to swelling and hyperplasia. While this tendency belongs to all children, in certain individuals it is so marked as to deserve a place as a distinct diathesis. It was formerly classed as one of the manifestations of "scrofula" or "struma"; but the proof that most of the manifestations once called "scrofulous" are really forms of local tuberculosis, makes it undesirable to use that term any longer to designate the condition under discussion.

In robust children, infectious processes of the nose, pharynx, or bronchi, cause acute swelling of the lymph nodes in the neighbourhood, which rapidly subside when the cause is removed. In others, in whom this vulnerability of the lymphoid tissues exists, the hyperplasia in the lymph nodes is out of proportion to the exciting cause and continues after the cause has ceased to operate. Certain children have at birth an excessive development of lymphoid tissue, particularly in the region of the throat in the form of enlarged tonsils, adenoid vegetations of the pharynx, etc.

The influence of heredity in causing this condition is too often seen to be passed over as a coincidence. Frequently the parents, when children, suffered from the same condition, and often every member of a large family of children is affected. They may be in other respects healthy, reared amid good surroundings, and show no evidence of any other constitutional disease. Any disease in the parents in consequence of which children are born with tissues having less than normal resistance, may be regarded in the light of a remote cause.

The condition is seen to perfection in children reared in institutions and in crowded tenements. It is more common in cities than in the country. Anything which produces malnutrition or lowers the general vitality of the tissues may be ranked as a cause. Rickets is often associated; sometimes it is to be reckoned as a cause, and sometimes both conditions depend upon the same causes.

During infancy, the lymphoid structures most frequently affected are those connected with the gastro-enteric and the bronchial mucous membranes; in later childhood it is those which are connected with the pharynx and tonsils.

The degree of enlargement of the lymph nodes which is sometimes found in the different situations has often led to a misinterpretation of them, particularly by those who only seldom see autopsies upon infants or young children. They have often been connected with pathological conditions or clinical symptoms with which they have really nothing to do.

Enlargement of the mesenteric glands and of the solitary follicles of the large and small intestine is very frequently seen in infants who have died from marasmus, and has been regarded as the cause of the wasting; while in reality it was only the consequence of the chronic intestinal indigestion which is an almost constant accompaniment of that condition.

As age advances we usually see retrograde changes in the different groups of glands unless they become the seat of tuberculous infection. Those connected with the digestive tract generally begin to diminish after the second year, and by the fifth or sixth year the enlargement has almost disappeared; while the tonsils, adenoid growths of the pharynx, and enlarged cervical glands are usually stationary after the seventh or eighth year and undergo quite a marked atrophy about the time of puberty. The presence of these enlarged lymph nodes and the catarrhal condition of the mucous membranes with which they are associated, are important in relation to all acute infectious diseases which affect these mucous membranes. They bring about an increased susceptibility to scarlet fever, measles, diphtheria, diarrhœal diseases, and most of all to tuberculosis.

STATUS LYMPHATICUS.

This condition is known also by some writers as "lymphatism"; while in its marked form it is quite distinct from that just described, the two have many points of resemblance, have often been confounded, and in fact, shade into each other. The term "status lymphaticus" is applied to a very definite pathological condition which is associated with clinical manifestations, less constant and not characteristic. The relation between the lesions and the symptoms is little understood, and almost nothing is known of the etiology or pathogenesis. The most striking part of the lesion is the great enlargement of the thymus gland, with which is found a hyperplasia of the lymphoid tissues throughout the body, more marked than is seen in any other condition in childhood. The two most frequent symptoms are convulsions and attacks of asphyxia.

Etiology.—The status lymphaticus is most often seen between the sixth and twelfth months, but may be met with in children of any age. Enlargement of the thymus to a degree sufficient to be regarded as pathological, is not an infrequent condition, being present according to the observations of Bovaird and Nicoll in about 12 per cent of the autopsies in the New York Foundling Hospital. How frequently the condition exists in cases not fatal it is impossible to say; but it certainly is not rare. An association with rickets is often observed, but it is doubtful whether this is anything more than a coincidence.

Lesions.—Since the large thymus is so important a lesion it is desirable to know what may be regarded as normal. The most extensive observations upon this point have been made by Bovaird and Nicoll, who weighed the thymus in 495 consecutive autopsies in children under five years. They found that the weight was greatest at birth, the average being 7.7 grams. After this time the change in weight was very slight for the period of five years, the average for the entire 495 observations being 5.9 grams, which was about the same as the average for each of the years taken separately. Excluding cases in which the organ was so large as to be considered abnormal (10 grams or over) the average weight at birth was 6.5 grams; during infancy and early childhood, 4 grams. The results of these observations do not differ essentially from those of Friedleben, which have been so extensively misquoted. Of 141 observations up to the age of five years, he found the average weight to be 7.4 grams; excluding cases in which the enlargement might be considered pathological, the average was 3 grams. It may therefore be assumed that the average weight of the normal thymus at birth is from 6 to 7 grams; from birth to five years from 3 to 4 grams. Anything over 10 grams may be considered distinctly abnormal.

In the status lymphaticus the thymus is often from five to ten times larger than normal. In the marked cases its weight is from 30 to 40 grams; in the less marked cases from 10 to 20 grams. The appearance of the enlarged thymus is well shown in the accompanying illustration (Fig. 172). A thymus of the size shown weighs about 45 grams, or 1½ ounces. In this instance it was nearly as large as one of the lobes

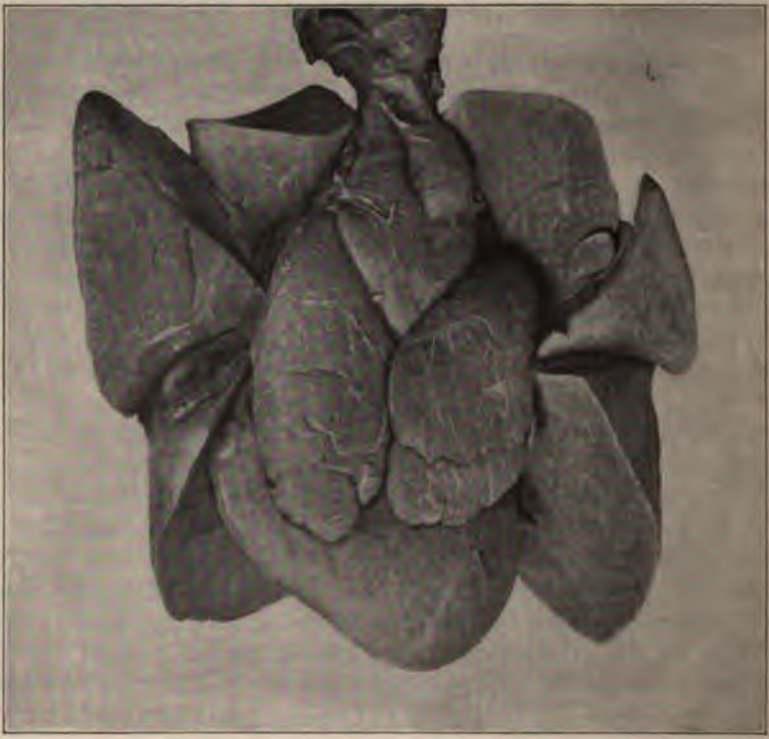


Fig. 172.—Enlarged thymus.

The lungs, heart, and thymus are shown in the picture. The lungs have been turned back showing the two lateral lobes of the thymus overlapping the heart; the central lobe, above, covers the trachea.

History.—Breast fed, male child, nine months old, well developed; ill less than twenty-four hours; dyspnœa, slight cyanosis, with death by asphyxia. T. 103° F.

Autopsy.—Besides the large thymus there were present the general lesions of the status lymphaticus to a marked degree; lungs deeply congested.

of the lung. In general appearance, the enlarged thymus is rather more vascular than normal, but other than hyperplasia shows no constant or essential changes, either by gross or microscopical examination.

The lymph nodes of the tracheo-bronchial region are greatly enlarged, often to the size of a small cherry, and are found in great clusters. Those of the mesenteric region may be still larger. Peyer's patches are very prominent, and the solitary follicles of the small intestine appear like mustard seeds upon the folds of the mucous membrane. Those of the colon are also very prominent. The lymphoid tissues about the pharynx and all the lymph nodes of the body are greatly hypertrophied. The spleen is usually enlarged with prominent follicles. There are no other constant changes. Those present are usually accidental, depending upon the cause of death.

Symptoms.—In very early infancy this is one of the explanations of sudden death occurring after slight causes, and in some cases without any apparent cause.

Death may be attributed to overlying, to asphyxia from food, or to some other condition affecting respiration, or infants are simply found dead in their cribs.

Even in those who live until they are several months, sometimes several years, old, there may be nothing in the child's condition to indicate the presence of the status lymphaticus until something acute occurs. This may be in the nature of a slight accident, a surgical operation of a trivial character, the administration of an anæsthetic, or some acute disease, frequently one affecting the respiratory tract. The symptoms associated with this condition are most frequently of a nervous character, usually attacks of convulsions, or they affect the respiration, causing paroxysms of dyspnœa, cyanosis, and even asphyxia. A frequent history is somewhat as follows: A child previously regarded as healthy, often well nourished and perhaps entirely breast fed, is taken with convulsions followed by high fever, preceding which there may have been some pulmonary symptoms suggesting a commencing broncho-pneumonia. The convulsions recur at short intervals; the temperature remains steadily high; the signs in the lung are few and not proportionate to the other symptoms; and death occurs in from twelve to thirty-six hours often in convulsions.

In other cases convulsions are absent and the prominent symptom is asphyxia, which comes in paroxysms and may be so complete as to lead to the suspicion of laryngeal obstruction. If intubation or tracheotomy is performed, no relief follows. The child may die in the first severe attack, which may be preceded for a few hours by moderate dyspnœa, or may come on almost without warning. It is more frequent, however, for the first attack to be less severe, the child perhaps being resuscitated with some effort, after which he may breathe almost as well as usual. In a few hours the attack of asphyxia is repeated; after several of these, each one growing more severe, death occurs. In these cases the elevation of temperature is usually slight and may be wanting.

Symptoms similar to the above but of less severity and resulting in recovery would suggest this condition, although the diagnosis cannot be established.

The cause of the symptoms is not definitely known. The asphyxia has been ascribed to pressure of the large thymus upon the lungs, the trachea, the pneumogastric nerves, or the auricles of the heart. Pressure would certainly seem to be one factor in the production of the dyspnœa. Further evidence in support of this is obtained by the relief afforded by an operation in which the anterior mediastinum is opened and the thymus raised and fixed to the sternum. This has been done in two or three instances with striking, but not always permanent, benefit.

In other cases, although the thymus may be quite as large as in those just described, the evidences of obstructive dyspnœa are much less and may scarcely be noticed.

There is another group of cases, perhaps the largest of all, in which there are no symptoms distinctly referable to the status lymphaticus, and yet this condition appears to be the factor which determines the fatal outcome of what was apparently an infection or an inflammation of only moderate severity. What is seen here is simply a greatly diminished resistance to disease. In these cases it is only the autopsy which reveals the explanation.

Diagnosis.—The diagnosis of the status lymphaticus is very uncertain. In some cases of marked enlargement it is possible to make out the enlarged thymus by percussion, but this is always difficult on account of its proximity to the lungs and trachea. We may suspect this condition during life; we can hardly do more. Marked enlargement of the tonsils and the adenoid tissue of the pharynx exists so frequently without thymus enlargement, that this can hardly be regarded as suggesting the condition. The hyperplasia of the tracheo-bronchial or mesenteric lymph nodes or of the follicles of the intestine produces no especial symptoms.

Prognosis.—While this condition apparently may exist for an indefinite time without producing any symptoms, it undoubtedly often determines a fatal outcome of what might otherwise have been a mild illness or a trivial accident. It is especially important in connection with acute bronchitis and broncho-pneumonia, with attacks of convulsions, with the shock of slight operations, and with the administration of anæsthetics, particularly chloroform. It is one of the most frequent explanations of unexpected death from slight causes, such as an exploratory puncture or the injection of antitoxine.

At present no known treatment has any influence upon the condition.

*Table showing the Situation and the Drainage-Areas of the Various Groups of Lymph Nodes of the Head and Neck.**

	Name of the group.	Number and situation.	Organs or areas from which they receive lymphatics.
1	Sub-occipital	One or two; at nape of neck.	Scalp, posterior portion.
2	Mastoid.	Four or five small ones; in mastoid region.	Receive efferent vessels from group 1, and through them from part of scalp.
3	Parotid.	Five to ten; on the surface and in the substance of the parotid gland.	Scalp, frontal and parietal portions; orbit, posterior part of nasal fossa, upper jaw, posterior and upper part of pharynx.
4	Submaxillary.	Twelve to fifteen; along base of jaw, beneath cervical fascia.	Mouth, lower lip, gums.
5	Supra-hyoid.	One or two; median line between chin and hyoid bone.	Chin and middle portion of lower lip.
6	Superficial cervical.	Five or more; along external jugular vein, beneath platysma, but superficial to the sterno-mastoid.	Auricle, part of scalp, skin of face and neck, and some efferent vessels from groups 1 and 2.
7	Deep cervical, upper set.	Ten to sixteen; about bifurcation of common carotid and along internal jugular vein. They are just above upper border of thyroid cartilage and on a level with hyoid bone.	Lower part of pharynx, larynx, palate, tonsils and part of tongue, part of nasal fossa, deep muscles of head and neck, and from inside the cranium. Receive also efferent vessels from groups 3 and 4.
8	Deep cervical, lower set.	A chain in the supra-clavicular fossa.	Connect with axillary group by a chain along axillary artery; also with glands of mediastinum and with groups 7 and 9.
9	Sub-hyoid.	A few small glands below hyoid bone and near median line.	Communicate with group 8, and may connect below with chain of bronchial glands.
10	Retro-pharyngeal.	Two small glands in front of spine and upon prevertebral muscles.	Pharynx and part of nasal fossa.

SIMPLE ACUTE ADENITIS.

This is an acute inflammation of the lymph nodes which in infancy frequently terminates in suppuration. A certain amount of inflammation of the lymph nodes occurs in children in all acute processes affecting the mucous membranes, especially when they are severe or prolonged. Those in connection with the various internal organs are considered with the diseases of the organs. Acute inflammation of the external nodes is of sufficient frequency to require separate consideration. While this is probably always secondary to some pathological process in the skin or mucous membranes, the primary condition may be so slight as to be overlooked, and the adenitis may be the more important condition or may even assume the appearance of a primary disease. It is particularly in infants that this is seen, and it depends upon the unusually active absorption and upon the susceptibility of the lymphoid tissues at this age. The cervical glands are frequently affected, and occasionally those of the axillary and inguinal regions.

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* Modified from Treves after Curnow in the Lancet, 1879, vol. i, p. 397.

Etiology.—Acute adenitis occurs in children of all ages in connection with diphtheria, scarlet fever, measles, and influenza. In such cases it is often severe, and after scarlet fever, occasionally terminates in suppuration. With the simple acute catarrhal processes of the pharynx and rhino-pharynx adenitis also occurs, but it is usually mild and rarely ends in suppuration. In infancy, on the other hand, acute adenitis from simple catarrh is not only very common but often severe, and frequently terminates in suppuration. Ulcerative stomatitis, carious teeth, eczema of the scalp or traumatism, may excite adenitis in children of all ages. Axillary adenitis may result from vaccination; inguinal adenitis, from vaginitis.

Of 109 cases of acute adenitis from my records, not including any associated with diphtheria, measles, or scarlet fever, more than three fourths occurred in the first two years, and half of them in the first year of life. This susceptibility of infants is very striking. The disease occurs frequently in those who are in other respects perfectly healthy, and often when the evidences of disease of the mucous membrane are slight. This is true not only of the cases of cervical adenitis, but also of others in which the inguinal glands are involved. The inflammation is excited in most of these cases by the absorption of pyogenic germs, usually staphylococci or streptococci, from the mucous membranes or skin; in some cases, as in diphtheria, probably by the action of toxins.

Lesions.—The changes taking place in the glands are acute congestion, with swelling, œdema, and active hyperplasia of the lymphoid elements. The process may terminate in resolution or in suppuration according to the intensity of the infection and the susceptibility of the tissues. When severe enough to cause suppuration, the adenitis is accompanied by considerable inflammation of the surrounding cellular tissue.

In the series of 109 acute cases to which I have referred, not including the specific infectious diseases, 96 were cervical, 9 were inguinal, and 4 axillary; 62 per cent terminated in suppuration, the latter being nearly all in infancy. Suppurative otitis was present in 16 per cent of the cases. Suppurative retro-pharyngeal adenitis (retro-pharyngeal abscess) was seen in several cases.

In infancy the disease is usually unilateral, or, if bilateral, the glands of one side are more severely affected than those of the other. Suppuration is nearly always of one side, and usually the abscess starts from a single gland.

Symptoms.—The symptoms and course of the adenitis of the specific infectious diseases belong to their clinical history. Suppuration is infrequent, except after scarlet fever. It is very rare after diphtheria, and when present usually signifies mixed infection; I have seen it occur but twice.

The typical cases of acute adenitis are those which occur in infancy. There are present the symptoms of the original disease,—usually catarrh of the nose or rhino-pharynx, mouth, or ear, which may not be very severe, and sometimes is overlooked. The glands most frequently affected are the deep cervical group. The tumour appears just below the angle of the jaw at the anterior border of the sterno-mastoid muscle (Fig. 173). The swelling during the acute catarrh is not rapid or great, but continues after the original process has subsided until it reaches the size of a walnut or even larger. In the most acute cases there is marked inflammation of the periglandular cellular tissue, with pain, tenderness, and extra heat. If suppuration occurs, it is generally evident in the latter part of the second week, but sometimes it may be as late as the third or even the fourth week. In the axillary or inguinal region (Fig. 174) the symptoms of adenitis are essentially the same as in the neck. In the inguinal cases the degree of catarrh of the mucous membrane is often very slight.

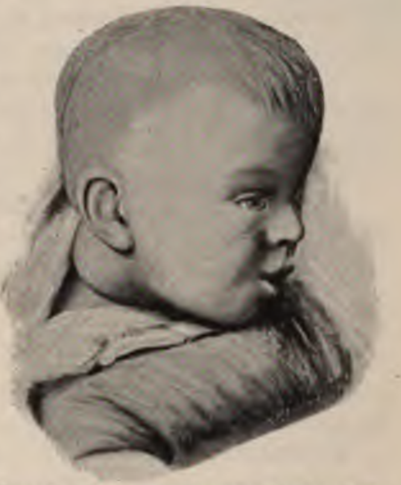
FIG. 173.—Acute suppurative adenitis in an infant one year old, showing the most frequent situation of the tumour in the cervical region.

Most cases run their course with slight fever and few general symptoms; but in young infants the constitutional symptoms are often severe and the physician may be in doubt whether the local process is sufficient to explain them. The temperature may be from 102° to 104° F. for several days, with considerable prostration, which is much increased if there is complicating otitis. After suppuration, if freely opened at the proper time, the abscess heals rapidly and permanently, a sinus being rare. Occasionally infection extends from one gland to another, and a succession of these glandular abscesses occurs.

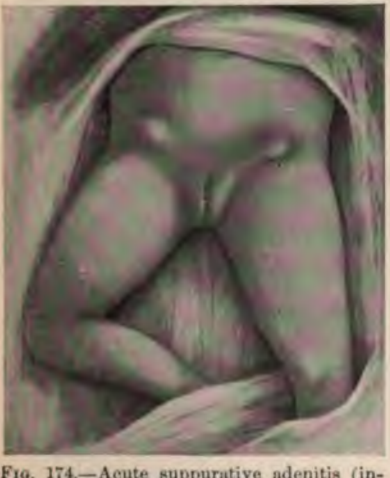
FIG. 174.—Acute suppurative adenitis (inguinal) in an infant three months old.

In the non-suppurative cases the swelling may be even greater than in those which suppurate; but it is less diffuse and apparently limited to the gland. It subsides slowly in the course of from four to eight weeks, often leaving a small tumour which may be apparent for several months. In susceptible children recurrent attacks of acute inflammation may lead to chronic enlargement which may last indefinitely. These glands do not become cheesy, except from subsequent tuberculous infection.

The acute cases in infancy in which suppuration occurs, appear to recover about as promptly and quite as completely as those terminating in resolution, although in the former the constitutional symptoms are more severe.

Diagnosis.—This is usually easy if it is remembered that, with the exception of the specific infectious diseases, and occasionally local causes like eczema of the scalp, carious teeth, etc., acute suppurative adenitis is essentially a disease of infancy. I have often seen it mistaken for mumps when the swelling was severe, but on close examination there is but little resemblance between the conditions. The disease is essentially acute, and has nothing in common with the slow suppuration seen in later childhood from the breaking down of tuberculous glands.

Treatment.—Prophylaxis requires that in all acute catarrhs the mucous membrane should be kept as clean as possible by the use of nasal or pharyngeal sprays, or by syringing with simple solutions like Dobell's or Seiler's, or one of common salt.

In the stage of acute inflammation very hot applications or an icebag may be used for the relief of pain. It is very doubtful whether either of these means has much influence in preventing suppuration. If abscess forms, incision should be deferred until pointing has taken place. If this plan is followed, refilling is rare. A simple free incision with proper aseptic treatment is all that is required. Curetting may be done if there is much broken-down tissue present, but it is not usually necessary. In most of the cases the abscess promptly heals and a perfect cure takes place. In cases which do not suppurate, absorption may be promoted by the internal use of the iodide of potassium in full doses—gr. x daily to an infant of one year. I confess rarely to have seen any benefit from painting with iodine or from inunctions of iodine ointment or the oleate of mercury. If adenitis is secondary to carious teeth, eczema, or ulcerative stomatitis, these conditions should receive appropriate treatment. Such cases do not usually suppurate, but subside rapidly when the primary cause is removed.

SIMPLE CHRONIC ADENITIS.

This consists in a simple hyperplasia of the lymph nodes. There are considered here only the external glands, but those of the cavities of the body are affected in a similar way, in diseases of the mucous membranes with which they are connected.

Simple chronic adenitis is not nearly so frequent as the acute form even in infants and young children, and it is rare after the fifth year. It may follow one or more attacks of acute adenitis, or it may result from subacute or chronic inflammations of the skin or of the various mucous membranes, infection from which causes the acute form. The most frequent subjects are children who have the diathesis described as "lymphatism."

Symptoms.—The glands upon both sides of the neck are usually involved, and more often a group than a single gland. The degree of swelling is not generally great, being much less than in acute adenitis, and usually less than in the tuberculous form. There are no constitutional symptoms. Hypertrophy of the tonsils and adenoid growths of the pharynx are frequently present. There is no tendency to suppuration or caseation. The swelling usually increases slowly for one or two months, then remains stationary for about the same length of time, after which it slowly subsides. A subacute course is more frequent than a very chronic one.

Diagnosis.—These cases are especially to be distinguished from those of tuberculous adenitis. The most important points for differentiation are, that they occur most frequently in children under three years, a period when tuberculous adenitis is not common; some definite exciting cause is usually present; caseation and suppuration do not occur; the glands do not become adherent to the skin or to the deeper tissues; they enlarge much more rapidly than do the non-caseating tuberculous glands; and they are influenced to a much greater degree by constitutional treatment.

Treatment.—Operative measures are not called for in simple adenitis; but as there are some cases in which a positive diagnosis from tuberculous adenitis is impossible, operation is to be considered in all doubtful cases if a thorough trial of other measures for two or three months has been without benefit. Local causes usually found in the pharynx, nose, or mouth should be removed if possible. Often more can be accomplished by removal to a climate in which the child's catarrhal symptoms are relieved than by all else. Little benefit is seen from local applications. The most useful internal remedies are, the syrup of the iodide of iron (twenty drops three times a day to a child of four years), guiaquin (one grain three times a day), and arsenic (two or three drops of Fowler's solution three times a day). Cod-liver oil should be given except during warm weather.

SYPHILITIC ADENITIS.

It is quite rare that a marked degree of glandular enlargement is seen as a symptom of hereditary syphilis; indeed, it is so rare that it is often forgotten that chronic multiple glandular enlargements are ever due to this disease. In the few examples that have come under my observation, this has been a late symptom of hereditary syphilis. The glandular enlargements were cervical and multiple, and the degree of swelling was often marked. They may be associated with disease of the bones or of the mucous membrane of the throat or of the nose, or without signs of such disease. The diagnosis of syphilis rests upon the association of other late manifestations of the disease—keratitis, periostitis, deformities of the teeth—and the prompt improvement under antisyphilitic treatment. In their local appearance they resemble tuberculous glands.

TUBERCULOUS ADENITIS.

Synonym: Scrofula.

Tuberculous disease of the lymph glands of the cavities of the body is discussed elsewhere; only that of the external glands is here considered. This condition presents some striking peculiarities: it is relatively rare in infancy, although a frequent form of tuberculosis in older children; it often exists as the only tuberculous lesion in the body. In the great majority of cases it is the cervical glands which are affected.

Etiology.—The age at which tuberculosis of the cervical lymph glands is usually seen is from three to ten years. In my experience with tuberculosis in infancy, the external glands are rarely involved, while the bronchial glands are almost invariably the seat of infection.

Local conditions favouring infection are adenoid growths of the pharynx, chronic pharyngitis, and hypertrophied tonsils; less frequently chronic otitis, chronic conjunctivitis, and pathological processes of the skin or the mouth, such as eczema of the face or scalp, ulcerative stomatitis, carious teeth, etc. That the pharynx is the most frequent seat of primary infection, is shown by the fact that the deep cervical glands are generally first affected. The question often arises whether the process is at first a simple one, and later becomes tuberculous, or whether it is tuberculous from the outset. My own belief is that in most cases the process is a tuberculous one from the beginning.

Children who are by inheritance predisposed to tuberculosis and those also who are prone to glandular enlargements—two conditions which are by no means identical—are the ones most liable to be affected. Attacks of acute infectious diseases, particularly measles, scarlet fever, and influenza, frequently play the rôle of exciting causes.

The age of those affected corresponds very closely with that at which children are most often seen with hypertrophied tonsils and adenoid growths of the pharynx. The subsidence of symptoms about the time of puberty, is also characteristic of both conditions.

Lesions.—It has been already stated that in the great majority of cases the cervical lymph nodes are involved, and generally they are the only ones affected. In 155 cases of tuberculous glands in the series reported by Treves,* those of the neck were the seat of disease in 145 and the only seat in 131; those of the axilla were involved in 17, but alone only in 4; the groin in 8, and alone in 6. This indicates the close association of the disease with infection through the upper respiratory tract. The nodes first affected are most frequently the upper set of the deep cervical group; sometimes, however, it is the superficial nodes of the submaxillary, or the parotid group, and occasionally the submental or the pre-auricular.† The chain of deep cervical nodes which is involved, follows the carotid artery, and often extends some distance below the clavicle. These deep nodes are sometimes connected with the bronchial group.

The process in all tuberculous glands is essentially a chronic one, but pathologically the cases may be divided into two groups, corresponding somewhat to the forms of disease seen in the lungs. In one group the process is more rapid, and tends to early caseation and softening; the products of inflammation are mainly cellular, and the amount of fibrous tissue is small. In another group the course is slower, and fibrous tissue predominates, caseation and softening being infrequent.

In the first group the glands in the early stage are swollen, of a pale pink colour, and homogeneous; later they become more firm, and show, as the first gross evidence of tuberculous deposits, small grayish-white spots, which are generally numerous and scattered through the affected gland; these spots enlarge, and may coalesce to form one large gray mass, involving nearly the whole gland. Subsequently there is caseation and then softening, usually beginning in the centre of the caseous area. Inflammation within the gland is followed by that of the surrounding tissues, which may result in adhesions or in the formation of a periglandular abscess. The first change in the gland is the production of epithelioid and giant cells, about which there is a zone of small round cells; cheesy degeneration then begins in the centre. The caseous masses may become encapsulated by the production about them of fibrous tissue; or softening may occur at one or more foci, and an abscess form. Such an abscess contains curdy material but very little true pus, the contents being chiefly detritus from the broken-down node. Tubercle bacilli are usually more numerous in the early stages of the process, but are often difficult of detection in broken-down tissues, and the curdy pus is sometimes sterile. As the glands soften, the process gradually extends from the centre to the surface, and they become adherent to the surrounding structures—blood-vessels, nerves, or the fascia—they fuse together and form large knotty masses, and when they ultimately break down they lead to the formation of an abscess in the cellular tissue, finally involving the skin. In the form of suppuration which occurs in and about tuberculous nodes, an important part is often played by other bacteria, usually the staphylococcus or the streptococcus.

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* Scrofula and its Gland Diseases. Smith, Elder & Co., London, 1882.

† Nicoll, Glasgow Medical Journal, January, 1896.

In the second group of cases, where the process goes forward more slowly, the changes are not quite the same, the essential difference being that the amount of fibrous tissue is much greater. These nodes are not so vascular; they are tough and hard, appearing like small fibrous tumours. The capsules are greatly thickened, and under the microscope is seen fibrous tissue arranged in concentric layers, often inclosing small caseous masses. These nodes less frequently form adhesions to the surrounding tissues, and consequently are freely movable, while suppuration is quite exceptional. Although the separate tumours are much smaller than in the first group, the glandular mass is often a large one, because of the number of glands involved.

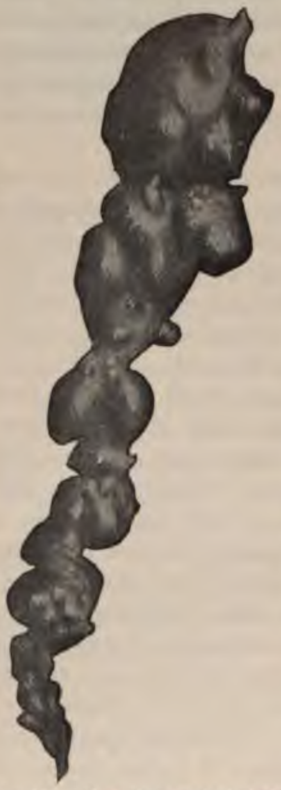

FIG. 175.—Posterior cervical chain of tuberculous lymph nodes.

The upper one showed giant cells and extensive cheesy degeneration; one at the middle showed early tuberculous changes—cell infiltration, giant cells, and a small area of cheesy degeneration; the lowest node showed one small tubercle with a cheesy centre. Child two and a half years old. (Dowd.)

It is seldom in either group of cases that the process is limited to a single node or even to two or three nodes. Very often an entire chain is involved (see Fig. 175). The pathological process under such circumstances usually varies in degree according to the distance from the main focus of infection; the nodes nearest show the most advanced changes; those at a distance, the early stages of the disease.

Tuberculous infection of the lymph nodes may terminate in resolution, encapsulation, calcification, or suppuration. The inflammation may subside before caseation has taken place and the inflammatory products undergo absorption. After caseation has occurred the masses may become encapsulated and contract to small fibrous nodules. Calcification of the glands in this location is rare. In other cases caseation is followed by breaking down, liquefaction, and an external abscess. The course which the local disease takes will depend upon the intensity of the infection and the general vigour and resistance of the child. There is seen in most cases a tendency of the inflammation to subside spontaneously about the time of puberty. Cure has sometimes followed an acute attack of intercurrent disease, such as erysipelas of the face, and even scarlet fever.

Symptoms.—In the early part of the disease there are no symptoms but the glandular swelling, and this begins very gradually. In most cases both sides are involved, but as the disease progresses the advanced changes are usually confined to one side. The enlargement is seldom continuous; it often increases for a time and then remains stationary or even diminishes, to take a new start from the stimulus of some fresh infection of the mucous membrane with which the glands are associated, such as an attack of measles or influenza, or simply from a deterioration in the patient's general health. During exacerbations, the glands may be painful and tender, and show the usual signs of local inflammation.

The whole course of the disease varies from several months to as many years. Treves gives three and a half years as the average duration where suppuration occurs. The glands first affected are usually those situated near the bifurcation of the common carotid artery. Such tumours usually make their appearance just in front of the sterno-mastoid muscle—sometimes behind it—and at the level of the upper border of the larynx or the hyoid bone. In the more rapid cases the tumours usually attain a considerable size in three or four months, sometimes in half that time. The usual size reached is from that of an almond to an English walnut. At first the tumours are movable and preserve their distinct outline; later they become adherent, first to the deeper tissues and to each other, finally to the skin, and there is formed an irregular nodular mass in which it is sometimes difficult to make out the individual glands. As the process approaches the surface there are small spots of softening; then there is distinct fluctuation; the skin becomes discoloured and finally gives way, and there is a discharge of thick, curdy pus, which may continue for an indefinite time, until the whole of the broken-down gland has been thrown off. This course is repeated with each successive gland which breaks down. In cases progressing more slowly the glands become adherent chiefly to one another, and suppuration is less frequent.

In what proportion of tuberculous lymph nodes suppuration occurs, it is difficult to say. Like other tuberculous lesions in the body, this one is more frequent than was once supposed; and in the past most of those which did not break down were not classed as tuberculous. It is probable that of the cases allowed to run their course about one half terminate in suppuration. Two forms of suppuration occur in connection with tuberculous glands—one an abscess of the gland proper, the other outside of and usually over it. In a typical case of the first variety, the gland is distinctly outlined and often superficial, there is very little inflammation, the spot of softening and fluctuation is small, and the pus discharged is always curdy. In the second variety the abscess is preceded by a more diffuse swelling, and the outline of the gland may not be made out; the signs of inflammation are more marked, the area of fluctuation is larger, and the pus is more like that of any ordinary abscess. Often the two varieties are combined; as when a gland beneath the deep fascia breaks down and there is formed directly over it an abscess in the cellular tissue, which communicates through a narrow opening with the gland beneath. In such cases the sinus continues open for a very long time, until the whole of the gland has been discharged. If healing occurs before this, the cicatrix soon breaks down.

Where abscesses are allowed to open spontaneously, large, irregular, and usually very intractable ulcers form. The skin is undermined for a considerable distance, and it has an unhealthy appearance. Such ulcers sometimes continue for many months in spite of all treatment, particularly if the patient's general health is poor. The scars left after them are large and unsightly, and sometimes positively deforming (Fig. 176). Their appearance is quite characteristic. They often have many tabs of skin attached to them; they may form prominent ridges which undergo contraction like those after burns; they are of a purplish-red

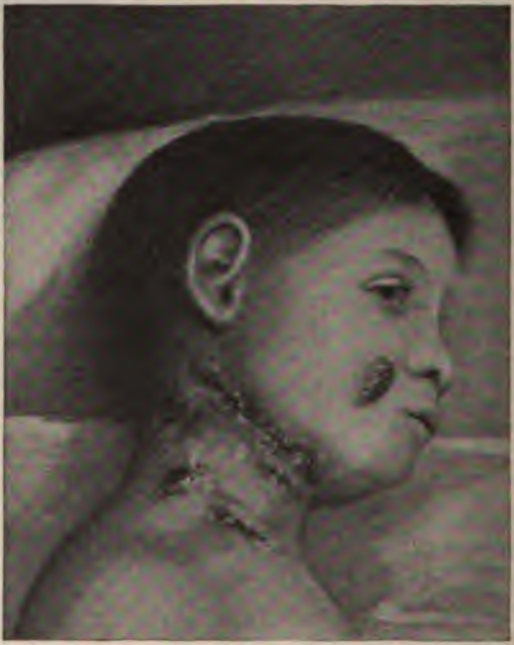

FIG. 176.—Cicatrices following a neglected case of tuberculous adenitis, in a girl seven years old. There is also a tuberculous patch upon the skin of the cheek in a very frequent location.

colour, and adherent to the deeper tissues. They are often sensitive and painful. As time passes they atrophy and become less conspicuous, though they remain through life.

The general health of children with tuberculous glands may be much or little affected, and not a few remain in good condition throughout the whole course of the disease, particularly when suppuration does not occur, but sometimes even when it is protracted.

Prognosis.—Tuberculosis of the external lymph nodes is seldom if ever the direct cause of death; although the course is often very protracted, ultimate recovery can usually be predicted. As previously stated, it is surprising that this process is so frequently the only tuberculous lesion in the body. Treves states that the percentage of those who die from general tuberculosis is so small that this danger is not to be considered an argument for operation. Poore * reports that of 58 cases treated by operation, only 2 were known to have died from tuberculosis. Dowd † has collected reports of 309 cases treated by removal more or less complete, whose histories were followed for several years after operation. Of these, 202, or 65·4 per cent, were apparently cured; 57, or 18·4 per cent, were living, though suffering from either local or general tuberculosis; 50, or 16·2 per cent, died of tuberculosis. These statistics surely do not support the hopeful views of the writers first quoted, but they are, I think, more in accord with general experience.

Diagnosis.—The diagnostic features of tuberculous glands are the age of the patient—usually from three to ten years—the site of the primary swelling, the indolent course, the trifling original cause, and most of all the disposition to slow caseation, softening, and abscess. The cases of simple hyperplasia are usually in children under three years, their progress is much more rapid, there is often some definite cause, and in most cases they nearly or quite disappear in the course of three or four months. They suppurate, if at all, during the first month. Syphilitic disease is to be recognised mainly by discovering the evidence of syphilis elsewhere, and by the effect of treatment. In Hodgkin's disease, glandular groups in other parts of the body are involved simultaneously or in rapid succession. There are no signs of inflammation or caseation; and the swellings are accompanied by very marked and definite constitutional symptoms—anæmia, emaciation, and general prostration. Malignant growths are very rare; they increase rapidly, often attaining a great size in a few months.

Treatment.—The general treatment of tuberculous glands is to put the child under the very best surroundings possible. The seaside has a great reputation for such cases, and no doubt the majority do very well there; but some are benefited even more by a dry, mountain climate.

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* New York Medical Journal, June 23, 1892.

† Annals of Surgery, May, 1899.

At all events, a child from the city should be sent into the country whenever this is possible. Internally the only remedies which have any special virtues are cod-liver oil and the syrup of the iodide of iron. The latter should be given in full doses—i. e., twenty or thirty drops, three times a day, to a child of six years. Arsenic and iron are useful as general tonics. Local applications are of little value and most of them positively harmful; painting with iodine and poulticing should be discarded altogether. The parts should be protected against cold, and should be rubbed or handled as little as possible.

It is important in every case to remove from the nose and throat all sources of local irritation. Hypertrophied tonsils should be excised, and the adenoid tissue of the pharynx removed even when not very extensive, since these are the two regions which most frequently harbour the tubercle bacilli. Any pathological conditions in the nose, such as hypertrophy of the turbinated bodies, should receive attention; so also should chronic otitis, chronic conjunctivitis, carious teeth or ulcers in the mouth. All these, if they do no more, keep up a constant glandular irritation, and produce conditions which are most favourable for the activity of the tubercle bacillus.

Operative measures.—These are indicated if, after two or three months of constitutional treatment, the glands affected continue to increase in size and number, or if softening occurs. The advantages of operation over leaving the case to Nature are, that it leaves a clean scar instead of a large, irregular one; that it shortens the disease and prevents the long, tedious suppuration of cases left to themselves; that it is a radical measure; and that it avoids the danger of general infection by removing the tuberculous focus.

The radical operation which aims at removal of all the diseased nodes through a free incision, is steadily growing in favour in New York. The best results follow this operation when it is done early before the skin is involved or the glands have softened or have formed extensive adhesions to the great vessels and neighbouring structures; also where a chain of glands is involved and where the inflammatory process is slow or indolent. In most cases operation requires a free incision and a prolonged and careful dissection, for the purpose is the removal not merely of two or three large glands which were evident before the incision was made, but the entire chain of fifteen or twenty smaller ones (see Fig. 175), some of which may not be larger than a pea, and are just beginning to be affected. If performed early a thorough operation by a good surgeon in the majority of cases will result in a permanent cure. However, the operation is not contra-indicated in cases which have gone on to a later stage, although the results may not be quite so satisfactory.

Other less radical operations are curetting, cautery puncture, and injections. Curetting is adapted to single large glands which have softened and are adherent to the skin. It may be done at any time except during a period of acute inflammation. Cautery puncture is an operation much done in Europe, though but little in this country. It is not applicable to glands smaller than a cherry. This operation is done with a small cautery point, which is thrust through the skin into the gland, and then in two or three directions through it, after which some soothing dressing is applied. The substances chiefly used for injection are iodoform emulsion, chloride of zinc, and carbolic acid. Injections and cautery puncture are to be advised only when the general or the local condition contra-indicates the radical operation.

Glandular abscesses should in all cases be opened as soon as pus forms, to prevent the extensive undermining of the skin, which is so likely to occur. The opening should be a small one, and all squeezing of the gland or surrounding tissues avoided.

HODGKIN'S DISEASE (ADÉNIE).

This is a rare disease in which there is a general hyperplasia of the lymphatic glands throughout the body, with growths of lymphoid tissue in the spleen, liver, and other internal organs. It is accompanied by marked anæmia, is progressive in its course, and usually terminates fatally. The cause is unknown. It is much more common in males than in females. Its occurrence in childhood is exceedingly rare.

The changes in the glands consist in a simple hyperplasia, which may be extreme. Suppuration and caseation are very rare, if indeed they ever occur. Any of the external or internal groups of lymph glands may be affected, and in severe cases the disease may involve almost every chain of glands in the body. Of the external groups, the cervical and the axillary are usually most affected; of the internal groups, those of the mediastinum and the retro-peritoneal region. The spleen and the liver are moderately enlarged, and lymphoid growths, varying in size from a pin's head to a grape, are usually scattered throughout their substance. There may be changes in the bone-marrow.

Symptoms.—The disease develops very gradually, often insidiously. The external glandular swellings are usually the first noticed, but sometimes it is the anæmia which first attracts attention; occasionally it is the local symptoms resulting from the pressure of internal glands, which may give rise to œdema, pain, cough, or dyspnœa. The progress is generally slow but steady, and the glands may reach an immense size. The blood changes are inconstant. As a rule, there is a relative increase in the lymphocytes, while the total number of white cells is generally less than normal, although sometimes increased.

Treatment.—This is very unsatisfactory. Arsenic in full doses appears to benefit some patients. The use of the X rays has produced striking, though in most cases only temporary improvement in the external glands.

CHAPTER III.

DISEASES OF THE SPLEEN.

Weight.—From one hundred and forty observations made at the New York Infant Asylum the following were the weights recorded at the different ages:

Weight of the Spleen in Infancy and Early Childhood.

AGE.	Ounces.	Grammes.
Birth	¼	7·7
Three months	½	15·5
Twelve "	¾	23·2
Two years	1¼	38·5
Three "	1½	46·4

Position and Methods of Examination.—The normal position of the spleen is close against the diaphragm, its external surface being opposite the ninth, tenth, and eleventh ribs. Its anterior border comes as far forward as the middle axillary line, its posterior border being usually near the vertebral column. In infancy it is practically impossible to outline the spleen by percussion, unless it is enlarged. During full inspiration the spleen is often depressed enough to be felt at the free border of the ribs, but at other times it can not be felt unless it is enlarged or pushed downward by some pathological condition in the chest. Normally, the long axis of the spleen is nearly parallel with the ribs, but when the organ is much enlarged, its axis corresponds nearly with a line drawn from the axillary line at the border of the ribs to the middle of Poupart's ligament.

The thin abdominal walls of young children render palpation of the spleen much easier than in adults; and this is a much more satisfactory method of examination than is percussion. In fact, the results from percussion are so uncertain and misleading that in most cases one may dispense with it, and rely on palpation to determine the size of the spleen. For satisfactory palpation it is necessary that the abdominal walls should not be tense. It is therefore important that the child should be quiet, and that the examination be made as gently as possible, and no force or undue pressure used. The child should lie upon its back with the thighs flexed and the skin, of course, bared. The physician, always having taken the trouble to warm his hands, should stand upon the left side of the patient and make pressure with the tips of the fingers, which are semiflexed. The pressure should be at first light, and gradually increased, the fingers being then held stationary during two or three respiratory movements. It is sometimes better to use the fingers of one hand for palpation, and make pressure with the other directly over the first. Palpation should be made in the axillary line. If the examination is satisfactory, and in the great majority of cases it is so if the child is quiet, the spleen can easily be felt when it is sufficiently enlarged to be of any diagnostic importance. With a little practice one can readily detect even slight degrees of enlargement.

When moderately enlarged, the lower border of the spleen is an inch or so below the free border of the ribs; when greatly enlarged, it forms a tumour which may nearly fill the left half of the abdomen. A tumour in the left hypochondriac region is recognised to be the spleen, by the fact that it is freely movable laterally and at its lower border or extremity, while it is attached above; also its inner border can usually be felt to be thin and sharp, and marked about its middle by quite a deep notch.

ENLARGEMENT OF THE SPLEEN.

In Acute Disease.—The spleen is most frequently and most constantly enlarged in malarial and typhoid fevers, but it is occasionally so in all the acute infectious diseases.

In most of these cases the enlargement is chiefly from congestion, but there may be acute hyperplasia and an increase in size of the Malpighian bodies. It may contain small hæmorrhages, and in extremely rare cases the spleen may rupture. In appearance it is generally dark-coloured, soft, and somewhat friable. In the cases which recover, the splenic swelling subsides with the original disease.

In Chronic Disease.—Like the lymph nodes, the spleen is much more often enlarged in children, particularly young children, than in adults. Enlargement is seen at times in almost all the chronic diseases of early life; but it occurs most frequently in rickets, syphilis, malaria, tuberculosis, the blood diseases, and in amyloid degeneration. Besides, it may be the seat of a primary growth, either benign or malignant.

Rickets.—The splenic enlargement which accompanies rickets is generally seen during the first year; at this period it is very frequent. The swelling is usually moderate, but occasionally it is so great that the lower border is three or four inches below the ribs. It belongs to the most severe forms of the disease.

Syphilis.—Enlargement of the spleen is one of the most constant lesions in congenital syphilis. It is present with great uniformity in children born with syphilitic lesions, and very frequently during the active period of the disease in early infancy. It is seen at a later period during infancy or childhood, associated with other late symptoms. The degree of enlargement is often great. In several cases I have seen it sufficient to form a large abdominal tumour. The liver also is increased in size, but not to such a degree. The pathological changes in the spleen in syphilis are considered with that disease.

Malaria.—The swelling in these cases may be very great. The liver is not so often enlarged as in syphilis. There is usually a history of exposure in a malarial district.

Tuberculosis.—It is rare to find anything more than a moderate swelling of the spleen in tuberculosis. In the most acute cases this may be due to the fever and general infection; in those which are less rapid, it depends either upon tuberculous deposits or passive congestion from venous obstruction, the result of the pulmonary disease.

The blood diseases.—Marked enlargement of the spleen is found in many cases of simple anæmia accompanied by moderate leucocytosis. This is quite peculiar to infancy and early childhood. The spleen is constantly swollen, and usually greatly so, in the pseudo-leukæmic anæmia of infants, in leukæmia, and in Hodgkin's disease. In the last two diseases the liver is also enlarged, but to a much less degree than the spleen; in the others it is but slightly changed.

Amyloid degeneration.—The causes of this condition and its general symptoms are mentioned in connection with amyloid disease of the liver (page 463). The spleen is constantly involved, and the enlargement of this organ, as well as that of the liver, may be very great. The changes resemble those found in the liver.

Cardiac disease.—In all forms of cardiac disease, and in other conditions in which there is obstruction to the systemic venous circulation, the spleen is enlarged. It is seen in congenital as well as in acquired cases. The liver is usually enlarged to about the same degree as the spleen, and there may also be dropsy of the feet.

New-growths, tumours, etc.—It is seldom in early life that the spleen is the seat of new-growths; these are usually varieties of sarcoma, but carcinoma has also been reported.

Primary spleno-megaly.—The rare cases of immense primary enlargement of the spleen have been variously interpreted. By some writers the condition has been regarded as lymphoma. Bovaird * has reported two cases in children, sisters, one of which was carefully studied microscopically, and the conclusions reached that the process was an *endothelial hyperplasia.* The condition was first described by Gaucher. Clinically the disease is characterized by a slowly progressing enlargement of the spleen which begins in early childhood and may continue for from five to twenty years; the size attained is very great, it often nearly filling the abdomen. In one of Bovaird's cases the weight was twelve and a half pounds. The other symptoms are a simple anæmia, inflammation of the gums with hæmorrhages from the nose, gums, and sometimes beneath the skin, and finally secondary symptoms due to the abdominal tumour. The course is very chronic, and thus far no known treatment has been of any avail.

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* American Journal of the Medical Sciences, October, 1900.

CHAPTER IV.

DISEASES OF THE BONES AND JOINTS.

ACUTE ARTHRITIS OF INFANTS.

THE terms *acute purulent synovitis, acute epiphysitis, pyæmia of bone* and *acute osteo-myelitis,* have all been applied to this condition. The disease is really a form of pyæmia. The causes and lesions may differ considerably in the different cases, but clinically they all have certain features in common, viz., an acute joint inflammation with suppuration.

The acute arthritis of infants is essentially a disease of the first year, and is much more frequently seen in the first six months. The inflammation may begin in the joint, at the epiphyseal junction, or in the medullary canal; but however it may start, the joint is soon invaded. The nature of the arthritis varies somewhat with the exciting cause. When it is due to the gonococcus, it is usually confined to the joint; there is in most cases a superficial inflammation involving the synovial membrane, but rarely leading to destructive changes in the cartilage, ligaments or bone. When it is due to the streptococcus or staphylococcus, it may begin elsewhere than in the joint, which, however, is usually soon involved, and complete disorganisation may follow. It may also result in a diffuse osteo-myelitis, in a subperiosteal abscess, or separation of the epiphysis. As a late result there may be a pathological dislocation or a "flail joint"; less frequently there is ankylosis.

Etiology.—The cause of acute arthritis in infants is the entrance of pyogenic organisms into the circulation. In my own cases the organism most frequently found was the gonococcus; next to this the streptococcus and staphylococcus; very rarely, the pneumococcus. In most cases occurring during the first two months of life, the portal of entry is probably the umbilical cord. Less frequently infection takes place through the skin, conjunctiva, genital tract, or the mouth. In the cases developing later it is often difficult to determine the point of entry, especially when the cause is the gonococcus. During the last few years twenty-six cases of acute gonococcus arthritis have been observed in the Babies' Hospital, only two of which, occurring during the first month, could be classed as infections of the newly born. The cases were observed during a hospital epidemic of gonococcus vaginitis, and yet nineteen were in male children, in no one of whom was there any genital lesion, and in only one was there conjunctivitis. Of the seven cases occurring in girls, only two had vaginitis. The portal of entry in these cases could not be definitely determined.

I once saw acute arthritis following pneumonia in an infant, in which the pneumococcus was obtained in the pus from the shoulder.

Symptoms.—The general symptoms often precede the local ones. In the most acute cases the temperature is high and widely fluctuating, accompanied by other symptoms of a severe infection. The earliest local symptoms are pain and tenderness, soon followed by swelling, which may develop quite rapidly in a single joint, or in several joints simultaneously. In those superficially situated there is redness of the skin, and fluctuation may be evident in three or four days. In cases coming on more gradually the temperature may be only from 100° to 102°, and suppuration may not occur for two or three weeks. In the most severe cases the progress is rapid, one joint after another being involved, with general symptoms of pyæmia, and death may occur in a week or ten days, usually from some visceral inflammation, pneumonia, pericarditis, or meningitis. This very severe course is less frequent than the more protracted one where symptoms last from two to four weeks. Unless the pus is evacuated extensive burrowing may take place.

In Townsend's collection of 73 cases, the joints were involved in the following order: hip, in 38; knee, in 27; shoulder, in 12; wrist, in 5; ankle, in 4; elbow, in 4; small joints, in 4. In three-fourths of these cases only a single joint was affected. No bacteriological examinations were reported. In my own 26 gonococcus cases, the localisation was as follows: finger or metacarpus, in 20; ankle, in 18; knee, in 17; wrist, in 12; toe or metatarsus, in 10; shoulder, in 9; elbow, in 5; temporomaxillary, in 1; hip, in 1. The average number of joints involved was four or five, the largest number being eight. The tendency of the gonococcus infections to involve the small joints is rather striking.

Diagnosis.—When several joints are involved, the disease has often been mistaken for rheumatism, which, however, at this age is so rare it may be ignored. Syphilitic epiphysitis resembles it in the localised tenderness and disability; but the rapid swelling and the severe constitutional symptoms are lacking.

Treatment.—Cold applications or wet dressings may be useful in relieving the symptoms. In some cases, most frequently when the cause is the gonococcus, the inflammation subsides without suppuration. In infections due to other organisms, suppuration almost invariably occurs and early free incision should be practised, followed by fixation of the joint. The results depend in no small degree upon the promptness with which the pus is evacuated. In the gonococcus cases there may be complete recovery. In most of the others the functions are impaired.

THE TUBERCULOUS DISEASES OF THE BONES AND JOINTS.

The chronic forms of tuberculous bone-disease, on account of their insidious onset and the frequency with which they simulate other diseases, more frequently fall, in the early stage at least, into the hands of the physician than into those of the general or orthopædic surgeon. All that will be attempted in this chapter will be to outline in a general way the most important forms—viz., disease of the vertebræ, hip, and knee—dwelling particularly upon the early symptoms and diagnosis. For their fuller discussion, particularly as to the details of treatment, the reader is referred to text-books on general or orthopædic surgery. The causes are the same, and the lesions are very similar in all forms, and will therefore be considered together.

Etiology.—The age at which tuberculosis of the bones most frequently begins, is from the third to the eighth year, it being comparatively rare before the end of the second year. The sexes are affected with about equal frequency. Tuberculous bone disease may occur in a child who has previously been in apparent health, but more often in one who has been reduced by some previous illness, especially the infectious diseases; of these, it most frequently follows measles and whooping-cough.

A family history of tuberculosis is present in a large number, but by no means in a majority of the cases. Like tuberculosis of the cervical glands, it is rarely preceded by other tuberculous processes, although it may be followed by them. It usually appears as an example of primary infection; but it seems very improbable that such should actually be the case. It is more likely that there has previously been a latent focus of tuberculosis elsewhere in the body. In many cases, antecedent disease of the bronchial glands has been demonstrated by autopsy. Infection from these or from other tuberculous lymph glands is the most probable explanation of the origin of infection in cases of bone disease. However, by some writers, notably Baumgarten, tuberculous disease of bone is regarded as due to direct inheritance, and is to be compared to the bone lesions which occur as late manifestations of hereditary syphilis.

Traumatism is often an exciting cause, and it may determine the site of the disease.

Lesions.—The tuberculous joint diseases of childhood are, as a rule, secondary to disease of the bones. Hip-joint disease usually begins in the head of the femur, and knee-joint disease in one of the condyles; ankle-joint disease in the lower epiphysis of the tibia, etc.

The frequency with which disease is seen in the different locations is shown by the following table, which gives the number of cases of each form applying for treatment at the Hospital for Ruptured and Crippled, New York, during ten years:

Spine	2,145	cases,	or	37·5	per cent.
Hip	1,937	"	"	34·0	"
Knee	1,222	"	"	21·5	"
Ankle or tarsus	255	"	"	4·5	"
Elbow	71	"	"	1·2	"
Wrist	50	"	"	0·9	"
Shoulder	24	"	"	0·4	"
Total	5,704			100·0	

The character of the bone disease upon which chronic joint disease depends is generally a primary ostitis, which affects the articular extremities of the long bones usually beginning near the epiphyseal line; in the short bones it is a central ostitis. The stages in the process are first congestion, swelling, and cell infiltration, followed by caseation, and frequently by softening and suppuration. In the early stage, the bone is slightly enlarged, and on section one or more yellowish foci of disease are seen. The disease may be arrested in this stage, encapsulation of the inflammatory products taking place; or it may continue until there is a more or less extensive breaking down or disintegration of the affected bone. As the disease extends there are involved, the periosteum, the articular cartilage, and finally the joint itself. Abscess may form in the joint or in the soft parts surrounding the bone. The process is quite analogous to tuberculous disease of the lung. As the disease advances ligamentous attachments are loosened, and displacement of the parts occurs with the production of deformity, due partly to muscular contraction and partly to the weight of the body. The inflammatory process with its resulting disintegration generally goes on to a certain point, where it is arrested. Gradually the broken-down bone substance is separated and thrown off in small particles in the discharge, and a reparative process begins, with the formation of healthy bone. Where joint structures have been destroyed, cure takes place by bony ankylosis. Sometimes the disease finds its way to the surface without involving the joint; at other times the disease may be arrested, and its products become encapsulated within the bone. Inflammation of the joint may occur by a gradual extension of the inflammatory process, or by a sudden perforation of the articular lamella. As a result of extensive disease, all the joint structures may be affected,—the synovial membrane, ligaments, articular cartilages, and the cellular tissue surrounding the joint. The process of disintegration and that of repair are both very chronic and measured by months or years. The entire course of the disease is from one to ten years, three years being about the average duration. In the great proportion of cases but one joint is involved, although it is not infrequent in hospitals to see two, three, and sometimes four of the large joints affected in the same patient.

Secondary lesions.—Abscesses form in a considerable proportion of the cases, and often burrow a long distance before they reach the surface. Amyloid degeneration of the liver, spleen, and kidney, and sometimes of the villi of the intestines, occurs as the result of the prolonged suppuration, chiefly in connection with disease of the hip or spine, occasionally with that of the knee. General or localized tuberculosis, particularly tuberculous meningitis, may develop at any time and prove fatal.

CARIES OF THE SPINE—POTT'S DISEASE.—This consists in a chronic inflammation of the bodies of the vertebræ, usually beginning in the central portion and extending to the periosteum, ligaments, cartilages, and, in fact, to all the contiguous structures. Secondarily it involves the membranes of the cord, the roots of the spinal nerves, and even the cord itself. The number of vertebræ usually affected is from two to five. The gross appearance of the lesion in a well-marked case is shown in the accompanying cut (Fig. 177). After the bodies of the vertebræ have become softened and partially broken down by disease, the pressure from the superincumbent weight of the body causes them to fall together and produces a backward displacement of the spinous processes, giving rise to the deformity known as kyphosis, which in its extreme form is popularly known as "hunchback."

Any part of the vertebral column may be affected; but the disease is most frequent in the dorsal region, as shown by the following statistics from the Hospital for Ruptured and Crippled: of 2,143 cases, 72·5 per cent affected the dorsal region, 15·3 per cent the lumbar region, and 12·2 per cent the cervical region.

Symptoms.—The onset is gradual, often insidious, and the early symptoms are frequently overlooked or misinterpreted. The case may go on for weeks or even months before the true nature of the disease is recognised, which is often not until deformity has occurred. In nearly all cases, however, the early symptoms are sufficiently characteristic to enable a careful observer to make a diagnosis before the stage of deformity.

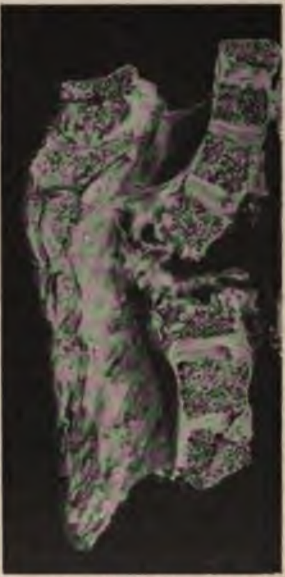

FIG. 177.—Pott's disease of the upper dorsal region; a vertical section of the spine, showing disintegration of the bodies of the vertebræ and encroachment upon the spinal canal. (From a patient dying in the Hospital for Ruptured and Crippled.)

The most constant early symptoms are: (1) pains caused by the irritation of the nerve roots and referred to various parts of the body, following the distribution of the spinal nerves; (2) rigidity of the spine from muscular spasm, this being an attempt to prevent motion at the seat of disease; and (3) the assumption of various postures calculated to relieve pressure upon the diseased vertebral bodies. Sometimes the first symptoms are those of pressure-paralysis (page 829); at others they are the local signs of abscess. In addition to the local symptoms mentioned, there is usually disturbed sleep, often accompanied by moaning.

Cervical disease.—The pains are often felt above the point of disease, frequently in the form of occipital neuralgia; sometimes they are referred to the front or the side of the neck. They may be so frequent and so severe that the face assumes a constant expression of anxiety or distress. In other cases pain is excited only by an attempt at movement. The muscular spasm most frequently takes the form of slight torticollis, sometimes of slight opisthotonus; sometimes there is simply a fixation of the head by a tonic spasm of all the muscles of the neck; both active and passive motion is resisted, and any movement may be so painful that the child involuntarily steadies its head with its hands. These symptoms come on gradually and are persistent. Sometimes they are overlooked, and the first thing to attract attention is a progressive weakness in the lower extremities, which proves the beginning of paraplegia. Occasionally the first marked symptoms are those due to the formation of a retro-pharyngeal or a retro-œsophageal abscess.

The deformity from cervical disease develops much later than when the disease is located elsewhere. Usually the neck appears broadened or thickened in a nearly uniform way, and often the head seems to have settled downward upon the shoulders. In the lower cervical region, a kyphosis is not infrequent; but in the middle and upper regions there is more often an anterior prominence, which may be felt in the posterior wall of the pharynx.

Dorsal disease.—The referred pains are now below the seat of disease, and take the form of intercostal neuralgia or pain in the epigastrium or the abdomen. They are often ascribed to cold, malaria, indigestion, or worms. There is a disposition to assume the prone position while sleeping, and also to lean across a chair or the lap of the nurse. The child walks carefully, holding the spine erect and very stiffly, and exhibits great caution in getting into or out of bed, or in rising from a recumbent position. In the beginning there may be a slight lordosis, or forward curve at the seat of disease, instead of the usual kyphosis or backward projection, but the latter soon takes its place, and with it is seen the compensatory lordosis in the lumbar region.

Lumbar disease.—The first symptoms here are often pain and lameness, referred to one of the lower extremities. This frequently leads to the suspicion that the hip is the seat of disease. In addition to the lameness there may be a tilting of the pelvis to one side, and sometimes quite a distinct lateral curvature of the spine. Referred pains are not so frequent nor so severe as when the upper part of the spine is affected; they may be felt in the groin, in the loin, in the thigh, in the buttock, or in the hypogastrium. The gait and attitude are very characteristic: throwing the shoulders well back, the patient walks stiffly with short steps, holding the spine with the greatest care. He rises from the floor awkwardly and with difficulty. Deformity is not usually so early or so marked as when the disease is dorsal, and often before it is visible there are symptoms due to the formation of psoas abscess,—lameness, flexion of one thigh, and a tumour deep in the iliac fossa or at the upper and inner aspect of the thigh; in both locations it has often been mistaken for hernia.

Physical examination.—Whenever any of the above symptoms are present, the child should be stripped and submitted to a thorough examination, the purpose of which should be to determine, first, the existence of any deformity; secondly, the mobility of the spine; thirdly, the presence of any secondary lesions, such as abscesses or paralysis. The mobility of the spine is best determined by studying the attitude, gait, and posture of the child, and the manner of stooping or rising from the floor. The gait has already been described with the symptoms of lumbar disease. As it has been aptly put, "the child walks with its legs but not with its back." In stooping, the same disinclination to bend or move the spine is seen. It is often impossible to induce the child to stoop at all, and when it does so, to pick up some object, there is acute flexion at the knee and hip, but as little bending of the spine as possible. In rising from the recumbent position the same thing is seen. The posture and attitude of the child will be modified by the position of the disease, and somewhat by the activity of the process at the time; however, by comparing the movements referred to with those of a healthy child, the great difference will at once be apparent. If the symptoms point to cervical disease, a digital exploration of the pharynx for deformity or abscess should be made, and the extremities should be examined for paralysis. If the disease is in the lumbar region, deep palpation of the iliac fossa should be made to discover a psoas abscess, and the passive movements of the thigh should be carefully tested to determine whether there is any resistance to extreme extension, this often being present before the psoas tumour. No matter how clearly the lameness may be at the hip, it should be remembered that this often results from disease of the lumbar spine. If the thigh is flexed and freely movable except in extension, the symptoms are probably the result of psoas irritation, for in hip-joint disease the other movements of the joint are also resisted.

The deformity of Pott's disease is often spoken of as "angular" curvature of the spine. While this is a true description of the disease at an advanced stage, there is often in the early stage only a general curve. Later a slight knuckle is seen from the unnatural projection of a single spinous process. This deformity may increase and finally involve five or six vertebræ. It is usually greatest in the upper dorsal region. A slight prominence, which does not disappear on suspending the patient, is always suspicious.

Tenderness upon pressure over the spinous processes and increased sensitiveness to heat and cold, are rarely present. Pain may sometimes be produced by downward pressure upon the head or shoulders in the axis of the spine. This symptom is not necessary for diagnosis, and the attempt to elicit it is strongly condemned by Gibney, who has seen serious harm follow such a test.

Course of the disease.—Caries of the spine is a very chronic disease, its course being measured by months or years, but marked, as in all chronic diseases, by periods of remission and exacerbation. An exacerbation may follow traumatism, and is often accompanied by the formation of an abscess. After the disease has lasted from one to three years, the destructive inflammation usually ceases and repair begins, a cure being finally effected by a process of consolidation of the fragments of the diseased vertebræ, and the production of ankylosis. Relapses are easily excited by traumatism, by improper treatment or by discontinuing the use of mechanical supports before the disease is arrested.

Abscesses.—The frequency with which abscesses occur depends somewhat upon the treatment. Townsend states that of 380 cases, abscess was present in 20 per cent. They are rarely seen earlier than three or four months from the beginning of symptoms, and usually belong to the second year of the disease. They sometimes form with acute symptoms, but more frequently they appear as typical cold abscesses. Those connected with cervical disease are retro-pharyngeal or retro-œsophageal, or they may open externally, usually just above the clavicle, in front of the sterno-mastoid muscle. Those with disease of the lower cervical and upper dorsal vertebræ, are apt to burrow along the spine, appearing in the lumbar region; rarely they may rupture into the œsophagus or the pleural cavity. Those with disease of the lower dorsal or lumbar vertebræ, may open just above the iliac crest posteriorly, or burrow anteriorly between the abdominal muscles, but the usual course is for them to follow the psoas muscle, appearing in the groin just above Poupart's ligament or at the upper and inner aspect of the thigh.

Paralysis occurs in about one half the cases in which the disease affects the lower cervical and upper dorsal vertebræ, but it is rare when the disease is below the middle dorsal region (see Compression Myelitis).

Prognosis.—The actual mortality of Pott's disease is difficult to state, so many of the consequences of the disease being remote and not fully appreciated until adult life is reached. The general mortality from all causes is from ten to twenty per cent. The causes of death are exhaustion from prolonged suppuration, amyloid degeneration, myelitis, general tuberculosis, and tuberculous meningitis. Sudden death occasionally occurs from pressure upon the cord in the upper cervical region, or from the pressure effects of abscesses in the posterior pharynx or in the posterior mediastinum.

The prognosis as to the amount of permanent deformity, will depend upon the seat of the disease, the time at which treatment is begun, and upon the thoroughness with which it is carried out. The best results as to deformity are obtained when the disease is below the middle dorsal region. With improved methods of treatment begun early, a large number of these patients recover with an insignificant amount of deformity, and some with none whatever.

Diagnosis.—The spinal deformity resulting from Pott's disease may be confounded with rachitic kyphosis or with rotary lateral curvature. Rachitic curvatures (page 261) are usually seen in children under eighteen months of age, a time when Pott's disease is rare; there are other signs of rickets present, and instead of rigidity there is usually undue mobility of the spine. What is true of rickets may be said of all curvatures depending upon malnutrition. Rotary lateral curvature is seen about puberty, rarely in young children except in connection with rickets. A slight lateral deviation of the spine, sometimes seen in the early stage of caries, may resemble a case of incipient rotary curvature. The latter is not attended by pain or rigidity, and is most frequent in young girls from eleven to fourteen years of age.

Other abscesses may be mistaken for those dependent upon vertebral caries. This difficulty is likely to exist in the cases attended by very little spinal deformity. These abscesses are most frequently in the iliac fossa or in the lumbar region, and may be due to perinephritis or appendicitis. The latter are more acute than those depending upon bone disease and usually accompanied by fever. Tumours of the vertebræ or of the spinal cord may give rise to symptoms almost identical with those resulting from compression myelitis due to Pott's disease, but both of these are extremely rare.

Treatment.—The treatment of Pott's disease is both general and local, and neither should be neglected. The constitutional treatment should be similar to that employed in other forms of tuberculosis.

The indications for local treatment are to put the diseased parts at rest, by immobilizing the spine and removing the superincumbent weight of the body. With the great advances made in orthopædic surgery it is no longer necessary to confine these patients in bed, as was formerly practised, to secure this result. It may be accomplished either by plaster-of-Paris, or some other form of jacket, or a properly fitting steel brace. A head-support should be attached to all forms of apparatus, if the disease is above the middle dorsal region. The closest attention to details and much experience in the use of apparatus are required to secure the best results. In perhaps no class of cases has the beneficial results of modern scientific treatment been more apparent than in those of Pott's disease. For the details in regard to the mechanical treatment and the different forms of apparatus, the reader is referred to works on general or orthopædic surgery.

ARTICULAR OSTITIS OF THE HIP—HIP-JOINT DISEASE—MORBUS COXARIUS.—In early childhood this generally begins as a chronic ostitis in the head of the femur, starting near the epiphyseal line. Exceptionally, and according to Gibney, oftener in older children, it begins in the acetabulum. The pathological process, as well as the clinical history, is generally described as consisting of three stages. In the first stage—that of ostitis—the lesions are limited to the bone; in the second stage—that of arthritis—all the joint structures are involved, and in this stage suppuration usually occurs; in the third stage there is breaking down and absorption of the head and sometimes of the neck of the femur, which, with destruction of the ligaments, leads to marked displacement of the parts from muscular contraction. The disease may be arrested in the first or in the second stage, or it may continue through all three stages.

Symptoms.—Clinically, the usual duration of *the first stage* is three or four months; it may last only for a few weeks, it may extend over two or three years, and the disease may be arrested in this stage. The onset is usually very gradual, and the symptoms are often considered of trivial importance until they have continued for some weeks. Generally the first thing noticed is slight lameness, due to stiffness of the joint. In the beginning this may be seen only in the morning, wearing off during the day. It may be accompanied by some tenderness about the hip and a disinclination to walk. A little later the child complains of pain, which is most frequently referred to the front of the knee or the inner aspect of the thigh, but only in rare cases to the hip itself. This is slight at first, but gradually increases in frequency and severity, and soon there are added the "starting pains" at night, which are one of the most characteristic features of early hip-disease. These pains are produced by a sudden spasm of the muscles during sleep. The child often cries out sharply without waking, sometimes wakes with a cry; this is often repeated several times during the night. Soon restlessness and fretfulness during the day are present. The lameness, which at first was slight and occasional, or noticed only in the morning, comes to be a constant symptom, and week by week increases in severity. The evolution of these symptoms may take only a few weeks, but sometimes they come and go in the most inexplicable manner during a period of several months, or even one to two years, before they are fully developed.

Physical examination.—Every child with a suspicious lameness, or with pains like those mentioned, should be stripped and submitted to a thorough examination. The first points to be observed on inspection relate to the general contour of the hip; every prominence and depression should be carefully noted. Then the attitude and gait should be studied; and finally all the functions of the joint should be carefully tested, and the limbs measured, to determine the existence of shortening or atrophy. At every step a comparison should be made with the sound limb. The contour of the hip is changed quite uniformly: there is broadening and flattening of the whole gluteal region; the trochanter is unnaturally prominent; the gluteal fold is shortened, and often single instead of double. There is no characteristic position of the limb in this stage. There is atrophy of the thigh and often of the calf. In Fig. 178 is shown the appearance of a typical case in the full development of the first stage. In walking, the child favours the diseased side, throwing the weight as much as possible upon the sound limb; but all these symptoms are of much less importance for diagnosis than is an examination of the functions of the joint.

For this purpose the child should be placed upon a table upon its back, and the various movements of the hip—abduction, adduction, flexion, extension, and rotation—should be executed, first with the sound limb and then with the suspected one, the two being carefully compared at every point to determine the degree of motion allowed. It is not necessary that force should be employed or pain inflicted. If the symptoms have existed for some weeks, there is generally a limitation of motion at the hip in all directions, but first usually in abduction, rotation, or extension. In more advanced cases, no motion whatever may be permitted at the joint, the pelvis tilting with the slightest movement of the femur. This fixation of the hip is due to tonic muscular spasm. Crowding the articular surfaces together, by pressure upon the heel or trochanter, produces pain, which is usually referred to the joint. This test should be carefully made, lest injury be inflicted. Gibney cautions against examinations under ether, since in this way serious injury may be done unconsciously.

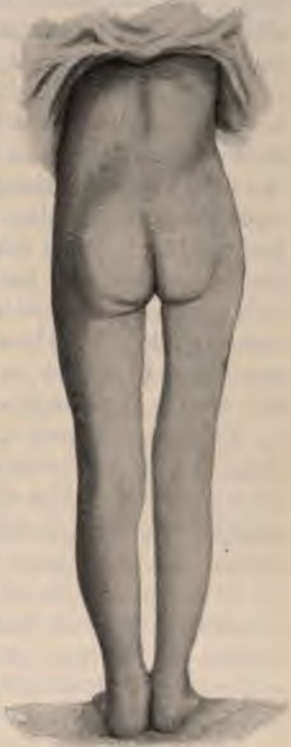

FIG. 178.—Hip-joint disease, at the end of the first stage, showing muscular atrophy, prominence of the trochanter, flattening of the gluteal region, and a single gluteal fold.

Second stage.—This has been called the stage of arthritis. Its existence may be assumed when the limb takes the position of marked permanent deformity, which is due at this period to muscular action, not to destructive bone changes. The transition from the first to the second stage is in most cases a gradual one, and the line between the two can not be sharply drawn; sometimes, however, it is rapid, and marked by a sharp exacerbation of all the symptoms. This may indicate a sudden perforation of the joint, and the rapid development of suppurative arthritis. Such is the usual result when an abscess which has been slowly forming in the bone, opens into the joint; or acute joint inflammation may be lighted up without so evident a cause. Sometimes the pus reaches the surface below the capsular ligament, and the joint remains intact. An acute exacerbation is indicated by increased pain, excessive tenderness about the hip, often by inability to walk, or even to bear any weight upon the limb, and frequently by fever. The position assumed by the limb is now fairly characteristic. The foot is generally everted, the thigh slightly flexed and rotated outward, and the limb apparently lengthened. There may be infiltration anywhere about the hip, due to the formation of an abscess. The muscular spasm is so great that the joint is locked,—no motion whatever being allowed. Abscesses may form at any point about the hip; they are especially frequent at the upper and outer aspect of the thigh, and may burrow long distances before reaching the surface. The duration of the second stage also is indefinite, but it usually lasts from a few months to a year, or the disease may be arrested in this stage.

Third stage.—There is now marked deformity, which is the result of muscular contraction after absorption of the head and sometimes the neck of the femur, and destruction of the ligaments. The position of the limb is a very constant one, and resembles that present in dislocation upon the dorsum of the ilium. There is shortening of from one to four inches; the thigh is strongly flexed, adducted, and rotated inward, and the foot is inverted; the trochanter lies against the outer surface of the ilium, and is above Nélaton's line. In this position the joint may become ankylosed. The displacement usually comes on gradually, but it is sometimes so sudden as to be mistaken for a true dislocation, although the latter is exceedingly rare in the course of hip-disease.

There is now marked atrophy of all the muscles of the limb, and the thigh may be two or three inches smaller than its fellow. No motion at all is usually allowed at the hip, but this is compensated for to some degree, by the exaggerated mobility of the lumbar spine. The spinal curvature—lordosis—is very marked both upon standing and walking. The duration of this stage may be several years. From time to time exacerbations occur, often excited by falls, and accompanied by the formation of new abscesses. In protracted cases, all the soft parts about the hip may be seamed with cicatrices from old sinuses. After the disease has gone on to the third stage, cure can take place only by ankylosis.

Diagnosis.—The important point in the early diagnosis of ostitis of the hip, is the gradual evolution of the symptoms, the most characteristic of which are lameness, "starting pains" at night, and impairment of all the functions of the joint. Mistakes in diagnosis most frequently arise from a failure to obtain a careful history, and from relying too much upon the symptoms of lameness and deformity. The essentially chronic character of the disease should constantly be borne in mind. In the vast majority of cases, with a careful history, and a thorough examination, there can be but little doubt as to the diagnosis except at the very outset. The proportion of obscure and irregular cases to those following the regular course, is small.

In the early stage, hip-joint disease may be confounded with a strain of the joint, with muscular rheumatism, poliomyelitis, periostitis of the shaft of the femur, phlegmonous inflammation in the neighbourhood of the joint, or with caries of the lumbar spine. In the second stage there is even less difficulty in diagnosis, although abscesses resulting from perinephritis or appendicitis have been mistaken for those arising from hip-disease. In the third stage, a mistake is almost impossible.

Prognosis.—This is to be considered both with reference to life and limb. The records of the Hospital for Ruptured and Crippled show the mortality of hospital patients with hip-disease to be nearly 25 per cent. This includes deaths directly or indirectly traceable to the disease. The causes are nearly the same as in caries of the spine,—exhaustion from prolonged suppuration, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

Under the most favourable conditions, the disease may be arrested in the first stage, and recovery occur without lameness or any noticeable impairment of the joint functions. This result, however, is not often obtained, because the disease is usually well advanced before it is recognised, or because of the difficulty in the way of carrying out all the details of treatment in the best possible manner. If the disease has advanced to the second stage, and suppuration has occurred, there always results some impairment of the joint functions; usually there are decided lameness and marked muscular atrophy, but very little shortening or deformity, provided the limb has been kept in the proper position. If the disease has advanced to the third stage, there are always marked shortening, deformity, and lameness.

Treatment.—The indications for constitutional treatment are the same as in caries of the spine. The purpose of local treatment is to secure constant and complete rest for the diseased parts, and to prevent deformity. Rest is secured by overcoming the muscular spasm by means of extension, by immobilizing the joint, and by transferring the weight of the body, in walking, from the hip to the perinæum. All these indications are now met, while the patient is up and about, by the use of the most approved apparatus. Formerly, rest and immobilization could be secured only by keeping the patient in bed, with the use of the weight and pulley. The general opinion of orthopædic surgeons at the present day is against excision, except in cases where, in spite of treatment by apparatus, the disease has advanced to the third stage, and in cases where life is threatened from prolonged suppuration and exhaustion. Under these conditions, excision should be performed; but early excision gives results very much inferior to those obtained by mechanical and constitutional treatment.

ARTICULAR OSTITIS OF THE KNEE—KNEE-JOINT DISEASE—WHITE SWELLING.—Ostitis of the knee usually begins in one of the condyles of the femur, the inner much oftener than the outer one; less frequently it begins in the head of the tibia. The pathological process is very much like that at the hip. There is in the first stage a central ostitis accompanied by infiltration and expansion of the part of the bone affected. The disease may remain limited to the bone, the inflammatory products becoming encapsulated, or softening and breaking down may occur, with the formation of an abscess. Gradually the process extends outward, and the periosteum and the soft parts are involved. The disease may invade the joint itself in a destructive inflammation, or pus may escape externally without seriously involving the joint structures. The degree to which the joint is involved, varies much in different cases; there may be only a simple synovitis, a suppurative arthritis, or a destruction of the cartilages and articular ends of the bones, synovial membrane, and ligaments, so that in the advanced stage all traces of a joint structure are lost.

If the process remains limited to the bone, recovery may take place with very little impairment of the joint functions. If suppuration in the joint has taken place, there will be more or less stiffness and fibrous or bony ankylosis. When there is destruction of the ligaments and articular ends of the bones, the limb assumes a characteristic position—the joint is flexed, the tibia is displaced backward and rotated outward, and there is marked over-riding of the femur. Bony ankylosis in this position is often seen.

Symptoms.—The earliest symptoms of disease at the knee are usually a slight stiffness of the joint, with a disposition to flexion and slight lameness. At first these symptoms are noticed only occasionally; finally they become constant and there is pain, which is usually referred to the knee. In some cases there are "starting pains" at night, although these are less constant and less severe than in hip-disease. Swelling is noticed early, as the diseased parts are so superficial. At first this is chiefly of the bone itself; the condyle, usually the inner one, is enlarged and elongated, often to a marked degree, before there is any infiltration of the soft parts. Later there is a general fusiform swelling, involving the entire joint and effacing all the normal outlines. Some tenderness upon pressure over the bone affected is present quite early, and there may be atrophy of the muscles of the thigh and calf. The knee is flexed and slightly rotated outward, the position which secures the most complete relaxation of the joint structures. In some cases there is seen the characteristic swelling due to distention of the synovial membrane. Abscesses may form anywhere about the joint; very frequently they burrow beneath the tendon of the quadriceps extensor as far as the middle of the thigh. Gradually the deformity increases until the leg may be flexed at a right angle, and rotated outward over an arc of twenty or thirty degrees.

The course of the disease resembles that of ostitis of the hip and the spine. During periods of remission, pain and tenderness often subside for several months so completely as to lead to the supposition that the disease has been arrested. An exacerbation is often excited by a fall or a strain of the joint, or it may follow an attack of acute illness. The disease may then progress rapidly and abscess after abscess form, with extensive destruction of all the joint structures and the production of permanent deformity.

Prognosis.—The danger to life is considerably less than in disease of the hip or spine. Death, however, results from the same causes—exhaustion, amyloid degeneration, and general tuberculosis or tuberculous meningitis.

With an early diagnosis and proper treatment the disease may, in a considerable proportion of cases, remain limited to the bone, and the resulting lameness and deformity be very slight; but otherwise a certain amount of lameness results from the stiffness of the joint. This may be due either to fibrous thickening or to bony ankylosis. Nearly all patients are able to walk without crutches, and if proper treatment has been carried out there is neither marked shortening nor deformity, although there is always great muscular atrophy.

Diagnosis.—The important symptoms for diagnosis, are the gradual onset, the early swelling which is due to enlargement of the bone, and the constant lameness and deformity. The disease may be confounded with rheumatism, with synovitis, and even with scurvy. In all these cases the resemblance exists only during the period of exacerbation. A careful history, however, will usually clear up the diagnosis.

Treatment.—The general treatment is the same as in other forms of joint disease. The indications for local treatment are the same as in hip-disease,—viz., to immobilize the affected limb and prevent deformity. This is accomplished by a form of apparatus which transfers the weight of the body from the joint to the perinæum, and which overcomes the muscular spasm which produces flexion and inward rotation of the joint. As in hip-disease, the results with mechanical and constitutional treatment are decidedly better than from early operative measures; but late operations are indicated under the same conditions.

TUBERCULOUS OSTEO-MYELITIS.—This disease is rarely seen except in the short tubular bones, most frequently those of the hand and fingers. From this fact it is often called *scrofulous* or *tuberculous dactylitis*. It is described by many writers under the name of *spina ventosa*. Unger* gives the following figures showing the frequency with which the different bones were affected: fingers in 43, toes in 3, metacarpus in 41, metatarsus in 14, radius in 2, ulna in 2, tibia in 3, jaw in 3. The first phalanx of the index finger is the bone which is most frequently the seat of disease. In the majority of cases the process is confined to a single bone, although it is not rare to see five or six affected. In such cases the disease is seldom symmetrical. The process is a chronic inflammation, beginning in the centre of the bone with the deposit of tuberculous material. The swelling which follows causes an expansion of the bone and thinning of the shaft, until a mere shell may remain. The later changes are, inflammation of the periosteum and the soft parts, the formation of abscesses and sinuses, necrosis, the exfoliation of sequestra, etc. The entire disease lasts from one to three years, and causes in most cases marked deformity.

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* Archiv für Kinderheilkunde, Bd. ii, 233.

Tuberculous dactylitis is essentially a disease of early childhood, being seen most frequently during the second and third years. In a considerable proportion of the cases there is a family history of tuberculosis. The disease frequently appears to be the only tuberculous lesion in the body, but tuberculosis of the hip, knee, ankle, or spine may be associated.

Symptoms.—Tuberculous dactylitis usually begins as a painless enlargement of one of the phalanges, most frequently the first one of the index finger. It may be two or three months before it is of sufficient size to attract much attention. Exceptionally the inflammation is a more active one, and is accompanied by both pain and tenderness. The swelling is quite characteristic; it is smooth, hard, uniform, and generally spindle-shaped, involving the entire phalanx of the affected finger. The appearance of a severe typical case is shown in Fig. 179. Later there is discolouration of the skin, and usually there is suppuration. The abscess generally opens at the side of the finger, and a curdy pus is evacuated. If the opening is enlarged by an incision there is found a cavity partly filled with caseous matter, and dead bone is felt, and perhaps a loose sequestrum. The cavity is surrounded by a thin shell of new bone, which is formed from the periosteum. If no operation is done the discharge continues for weeks or months, other abscesses often form, and finally several small

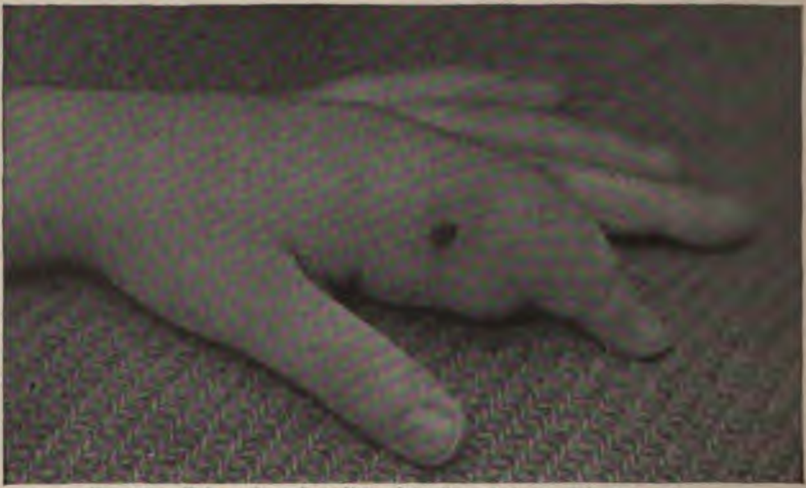
FIG. 179.—Tuberculous dactylitis of the first phalanx of the index finger.

sequestra are exfoliated,—sometimes a single large one, which is the shell of the diseased phalanx almost entire.

In some cases the disease is arrested before necrosis occurs, but in the majority this is not so. After the wounds have all healed the finger remains shortened, deformed, and often useless. In some cases the disorganization is so extensive that amputation is necessary.

Diagnosis.—The recognition of dactylitis is usually easy, but as symptoms identical in almost every particular may be seen in a syphilitic inflammation, it is often difficult to tell with which of the two forms one has to deal. The tuberculous form is very much more frequent; it may occur in a patient with tuberculous antecedents, or it may be associated with other tuberculous lesions. Syphilitic cases are distinguished by the fact that the lesion is more frequently multiple, that it is often symmetrical, and that other manifestations of syphilis are generally present. It is affected by anti-syphilitic remedies, which is not the case in the tuberculous variety.

Treatment.—Painting with iodine and like measures are useless. The diseased part should be kept at rest,—if a finger, by the application of a splint. Every means should be taken to build up the patient's general health, as this is the most effective way to influence the local process. The general verdict of surgeons is against early excision as a means of arresting the disease. Abscesses should be opened early and freely, all diseased bone removed, the finger kept in proper position, and the wound treated according to general surgical principles. Under almost any treatment the disease is a protracted one, and rarely lasts less than a year.

THE SYPHILITIC DISEASES OF BONE.

The bone lesions of hereditary syphilis are not infrequent, but were long unrecognised, and have only within comparatively recent times been fully understood.* They may be divided into two groups,—those occurring with the early symptoms, and those which belong to the late manifestations of the disease.

Acute Epiphysitis.—This is the most frequent variety of bone disease in early hereditary syphilis. It may begin even in intra-uterine life, and it forms one of the most characteristic lesions of the disease. To some degree it is almost invariably present in syphilitic fœtuses and in syphilitic infants who are still-born.

In the early stage, there is an increase in the cartilage cells and delayed ossification. Later, a line of softening forms at the epiphyseal junction, which may cause loosening of the cartilages and ultimately complete separation of the epiphysis from the shaft, by the formation of granulation tissue between them. In cases receiving proper treatment, recovery may take place with good union, perfect function, and without any deformity. In other cases degenerative changes continue, and infection with pyogenic germs may be added. The periosteum and the soft parts in the neighbourhood are now involved, with the formation of external abscesses; or the disease extends to the medullary cavity, giving rise to acute osteo-myelitis, which may lead to necrosis; or the contiguous joint may be invaded, causing an acute suppurative arthritis. This last result is more likely to occur where the epiphysis joins the shaft within the joint cavity. The large joints are usually affected, and the lesions are frequently symmetrical. Acute suppurative arthritis may occur independently of changes at the epiphysis; but even when these are seen in syphilitic infants they are to be regarded as of pyæmic rather than of syphilitic origin. Secondary to the changes at the epiphysis, there is periostitis and inflammation of the soft parts. Periostitis of the shaft is rare in early infancy,

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* See Taylor, Bone Syphilis in Children, New York, 1875; also G. Wegner, Virchow's Archives, Bd. 1, Heft 3.

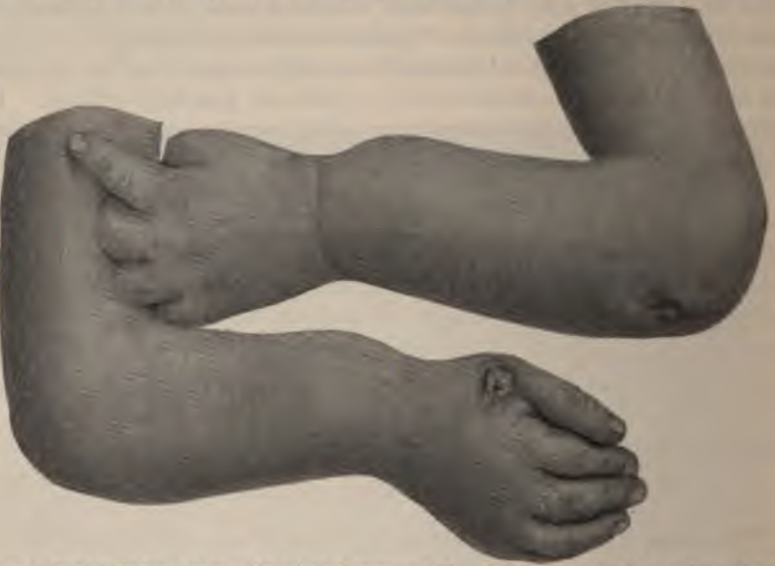

FIG. 180.—Syphilitic bone disease in a boy four years old. The lower end of the radius of both arms is enlarged as a result of former epiphysitis; there are sinuses leading to dead bone over the metacarpal bone of the right thumb, and over the upper extremity of the left ulna. The last two are recent lesions.

The bones most frequently the seat of acute epiphysitis are the humerus, radius and ulna, although any of the long bones may be affected.

Symptoms.—The early symptoms are usually quite acute, and appear during the first six weeks of life; they may precede any other manifestations of syphilis. In some cases there is first noticed an inability on the part of the child to move the limb, which may easily be mistaken for paralysis. It is, in fact, often described as "syphilitic pseudo-paralysis." The limb lies perfectly motionless, and any attempt at passive movement causes evident pain. There is tenderness on pressure and soon swelling is seen, both being most marked at the epiphyseal line. If the bone affected is superficially situated, as the lower epiphysis of the humerus, radius, or tibia, swelling is very apparent, while it may be scarcely perceptible at the upper epiphysis of the humerus. The swelling is usually cylindrical and moderate in degree, being limited to the extremity of the bone. In the more severe cases it may involve a great part of the limb. Abscess may form and separation of the epiphysis take place, so that crepitation may be obtained by moving the limb. Separation of the epiphysis not infrequently occurs even when there has been no suppuration.

In the milder cases, or those which have been subjected to active treatment, both the swelling and the tenderness subside rapidly without suppuration; and even though the epiphysis has separated from the shaft, it speedily unites. Where pseudo-paralysis has been the chief symptom, very rapid improvement occurs under treatment, and usually complete recovery of function in two or three weeks. If the disease extends to the joint, or if osteo-myelitis develops, the case is almost certainly fatal.

Diagnosis.—This is usually easy, from the age of the patient—generally under three months—the early prominence of pain and apparent loss of power, with the later appearance of swelling and signs of inflammation at the epiphyseal junction. In all these respects the disease closely resembles scurvy; but the latter is rare before the eighth or tenth month, there is usually a history of the long-continued use of some proprietary infant food, and it is cured by dietetic treatment alone.

The apparent loss of power may lead to the diagnosis of birth palsy, especially of the upper-arm type (page 112). The presence of acute pain and tenderness, the absence of the characteristic deformity, and the prompt recovery under constitutional treatment, usually make the distinction between the two conditions an easy one.

Treatment.—This is the same as in all early syphilitic manifestations, for which see the article on Syphilis. Locally, the part requires in the early stage only protection and rest. Should suppuration occur in the neighbouring joint, or should osteo-myelitis develop, these conditions should be treated surgically as they are when due to other causes.

CHRONIC OSTEO-PERIOSTITIS.—This is the usual form of bone disease which is seen in late hereditary syphilis, and it is one of the most frequent and most characteristic lesions of that stage of the disease. Occurring in adults, this would be classed as a tertiary symptom. Chronic syphilitic osteo-periostitis is rarely seen before the third year, and most of the cases occur between the fifth and fourteenth years. The most frequent seat of disease is the tibia, and next to this the bones of the forearm and the cranium. The following is the frequency with which the different bones were affected in the series of cases reported by Fournier:* tibia in 91 cases, ulna in 22, radius in 15, cranium in 16, humerus in 12, all others in 37. The process may result either in a diffuse or a localized hyperplasia of bone or in necrosis.

The typical changes are seen in the tibia. The shaft of the bone is principally or solely affected. There is often produced a very characteristic deformity, consisting of a forward curve of the anterior border of the tibia, which has been compared to a sabre blade (Fig. 181). In some cases the bone is bent inward at its lower third, resembling somewhat a rachitic curvature (Fig. 182). Sometimes the entire shaft of the bone is affected, and it may be enlarged to nearly twice its normal dimensions.

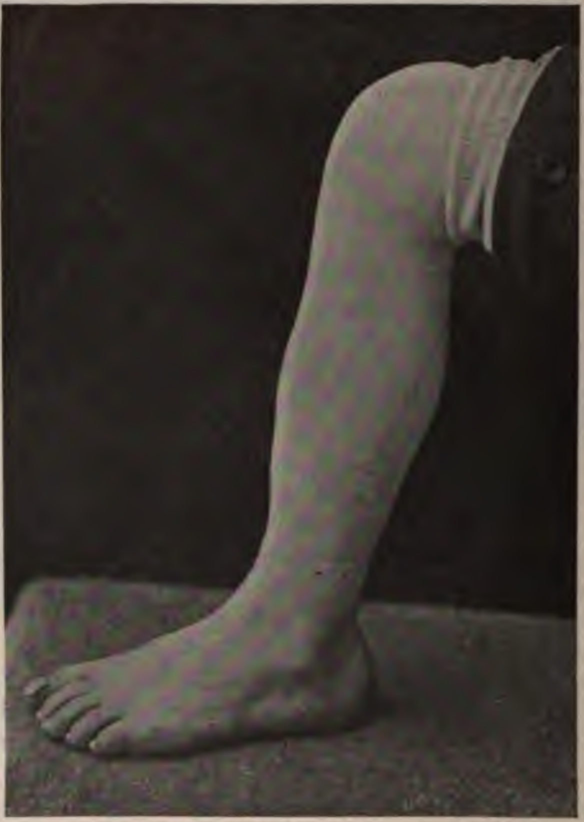

FIG. 181.—Syphilitic disease of the tibia, showing the sabre-like deformity, in a boy nine years old.

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* Syphilis Héréditaire Tardive, Paris, 1886.

At other times the swelling is chiefly near the epiphysis, where large bosses may form of sufficient size to interfere with the functions of the joint. Instead of affecting the bone uniformly, the disease often affects only certain parts, leading to the formation of large nodes which are more likely to be followed by necrosis than are the other lesions. In most of the cases the process is purely a hyperplastic one, leaving the bone permanently enlarged. Less frequently, there occur gummatous deposits in or beneath the periosteum, which may soften, suppurate, and lead to superficial necrosis, with the formation of sinuses that remain open until the sequestrum is exfoliated (Fig. 183). Syphilitic deposits sometimes take place in the interior of the bones, generally near the articular ends; these may soften and break down with abscesses, sinuses, etc., very much after the manner of a tuberculous inflammation (Fig. 180).

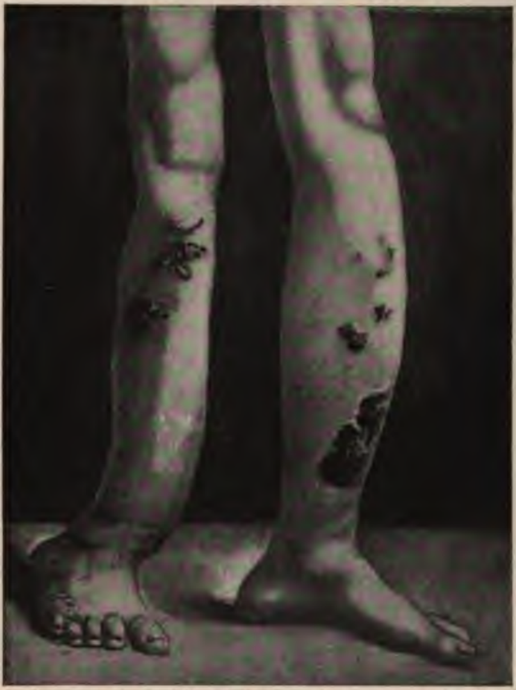

FIG. 182.—Syphilitic disease of both tibiæ. The left shows a general enlargement of the bone, the characteristic curve of its anterior border, with ulcers due to necrosis. The enlargement of the right tibia is less marked, and there is a pseudo-rachitic curve at its lower third. Cicatrices near the knee mark the site of former ulcers. (After Fournier.)

The lesions of the other long bones are essentially the same as of the tibia. They are nearly always symmetrical and often multiple. In a case recently under observation in a boy of four years, the disease involved both tibiæ, both radii, the right ulna, the left metatarsus, and the metacarpal bone of the left thumb. The course of syphilitic osteo-periostitis is very chronic, and some permanent deformity is the rule, unless cases come very early under treatment.

When affecting the bones of the cranium the disease usually takes the form of a gummatous periostitis, which leads to the formation of large nodes. These may remain as permanent deformities, or they may break down and suppurate, with necrosis of one or both tables of the skull. This may be followed by inflammation of the dura, the pia, and even of the brain itself.

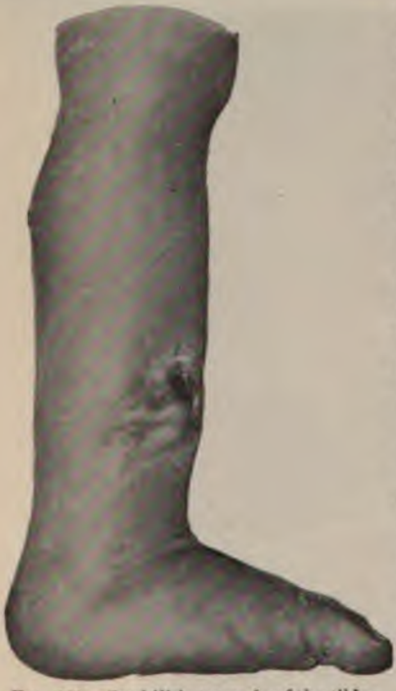

FIG. 183.—Syphilitic necrosis of the tibia, showing moderate enlargement of the bone and a sinus. (From the same patient as Fig. 180.)

Symptoms.—When the long bones are affected, the symptoms are pain, tenderness and deformity. These come on very gradually, and often the deformity is noticed before either pain or tenderness is sufficiently marked to attract attention. The pain is regularly worse at night, and often felt only at that time; it may be mild and occasional, or so severe as virtually to prevent sleep. There is tenderness on pressure over the bones affected, the acuteness of which will depend upon the activity of the process. When suppuration occurs, it comes very slowly, and never with symptoms of acute inflammation. Sinuses usually continue to discharge until a sequestrum is exfoliated. The course of the disease is very tedious, and the whole duration is usually several years.

When the cranium is affected, there are seen the irregular nodes, especially upon the frontal and parietal bones. They are from one to two inches in diameter, and project from one eighth to one fourth of an inch above the general outline of the skull. There may be pain, tenderness, softening, suppuration, and necrosis, as in the long bones.

Diagnosis.—It is so very rare that disease of the bones of the cranium is due in childhood to any other cause than syphilis, that this disease may always be assumed to exist if traumatism can be excluded. The bosses upon the cranium in rickets (page 262) are always large, smooth, and regular in position, and belong to infancy.

Syphilitic disease of the long bones is recognised by the nocturnal pain, the tenderness and peculiar deformity, and by the association of other late manifestations of syphilis,—i. e., the peculiar notched teeth, the interstitial keratitis, the enlarged epitrochlear glands, etc. Tuberculous disease generally affects the articular ends of the bones; syphilis nearly always the shaft. The diffuse hyperplasia of the tibia and the sabre-like deformity of its anterior border are rarely if ever due to any other cause than syphilis.

The deformities of the long bones have in some cases a certain resemblance to those due to rickets, but on close examination there are seen striking differences. The epiphyseal enlargement at the wrist in rickets affects both bones (Plate V, page 258); in syphilis it is usually of one bone only (Fig. 180). The differences between rachitic curvatures of the tibia and the deformities from syphilis may be readily seen by comparing Figs. 48, 49, and 50 (pages 263–265) with Fig. 182.

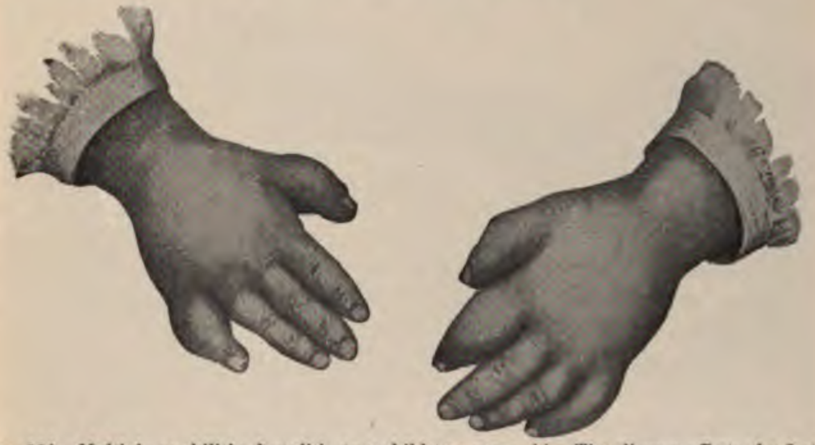

FIG. 184.—Multiple syphilitic dactylitis, in a child two years old. The disease affects the first phalanges of both thumbs, both little fingers, and the index finger of the left hand.

Treatment.—The constitutional treatment of these lesions is the same as that of the other late manifestations of syphilis,—mercury and the iodide of potassium; for details, see the chapter on Syphilis. Surgical treatment is required in cases which terminate in necrosis, whether of the cranium or the extremities. They are to be managed like the same conditions in adults.

SYPHILITIC DACTYLITIS.—This belongs to a somewhat earlier period of syphilis than the disease just described, and is usually seen in children under five years old. It is not a frequent manifestation of syphilis, and as compared with tuberculous dactylitis it is rare. It was first fully described by Taylor (New York). The symptoms closely resemble the tuberculous form. It may begin as a periostitis but more frequently as an osteo-myelitis. Like the tuberculous form it usually goes on to suppuration and necrosis. According to Taylor, dactylitis is more often single than multiple, but in my own cases several phalanges have generally been involved, and the lesions have often been symmetrical (Fig. 184). In one case, the first phalanx of every finger of both hands was affected. This occurred in a child nine months old who was under observation for over two years, and who presented during this period almost every lesion of hereditary syphilis.

The symptoms and course of syphilitic dactylitis are essentially the same as in the tuberculous form. The differential diagnosis is considered with the latter disease. The prognosis is much the same in the two varieties, with the exception that in the early stage the syphilitic cases may often be arrested by constitutional treatment. This is the same as in other late lesions of syphilis. The same local treatment should be employed as in the tuberculous cases.

CHAPTER V.

DISEASES OF THE SKIN.

The skin at birth is covered with a whitish sebaceous secretion, the vernix caseosa. The skin itself is of a deep purplish colour, which changes to a bright red over the face and trunk in a few minutes, with the establishment of normal respiration, and in a few hours the whole body has the same tint. This excessive redness slowly fades during the first month, at the end of which time the skin has assumed the pale pink of infancy. On the third or fourth day there are usually seen the first signs of icterus; this generally fades by the end of the second week.

The epidermis which is present at birth soon loosens and is thrown off. This normal desquamation usually begins upon the fourth or fifth day, and is completed in ten days or two weeks. If the skin is frequently oiled and properly bathed, desquamation is scarcely noticeable unless a close examination is made. In some infants, especially those who are delicate and cachectic, it is very much more marked, and closely resembles that seen in scarlet fever. Ritter has described an *exfoliative dermatitis* of the newly born, appearing generally during the second and third weeks, a condition which is regarded by Kaposi as simply an exaggeration of normal physiological desquamation. This process may be mistaken for that due to hereditary syphilis; the latter, however, is rarely general, appears later, and is much more prolonged.

Perspiration is rarely present before the end of the fourth month, and is then seen only upon the forehead. In healthy infants it is scarcely noticeable during the first year. Copious perspiration is most frequently a symptom of rickets; less marked perspiration may occur with any general weakness or during acute illness.

CONGENITAL ICHTHYOSIS.

Congenital, or more properly fœtal, ichthyosis, sometimes known also as diffuse keratoma, is a rare disease, characterized by the formation, usually all over the body, of a thick, horny epidermis resembling parchment. This is divided by fissures or shallow furrows into irregular patches; sometimes these are two or three inches wide, at others as small as a pin's head. The disease begins in the early months of fœtal life, and is an abnormality in the development of the skin, there being an excessive proliferation of the layers of the epidermis.

Symptoms.—In the gravest form of the disease the child often lives but a few hours, and rarely more than a week. The openings of the nostrils and the ears may be occluded by the excessive production of epithelial cells. The eyes are in a condition of ectropion, and there are often deformities of the mouth and other orifices due to the contractions of the skin. The nails and hair are usually imperfectly developed. The body seems incased in a hard, horny covering, and looks as if it had been varnished or covered with collodion. The skin cracks or splits and the edges curl up, an appearance which has been aptly compared to the skin of a boiled potato.

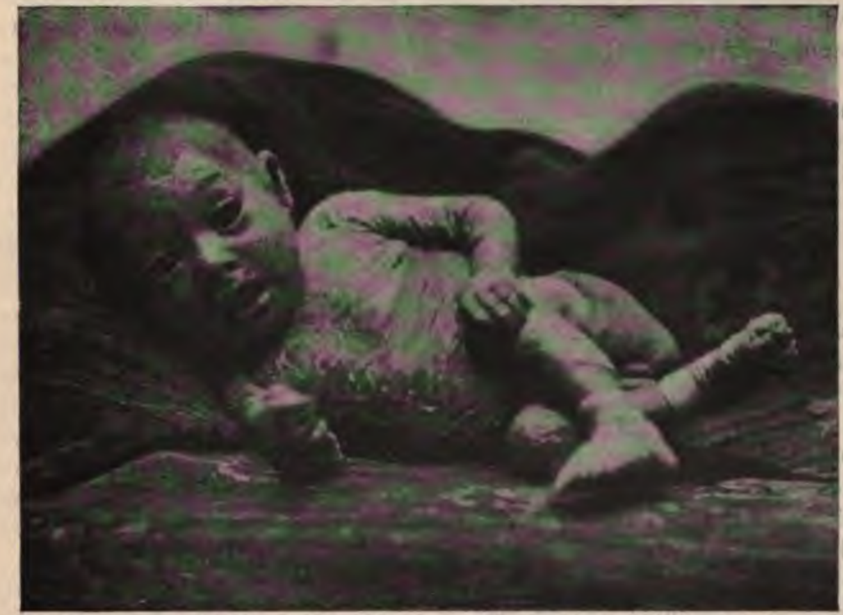

FIG. 185.—Congenital ichthyosis in a child ten months old. The large scaly patches are well shown on the lower part of the right chest and abdomen, and the constricting bands upon the legs. (From a photograph by Dr. Cabot.)

In the milder form, the duration of life is indefinite, depending upon the degree of development of the disease; but even in such cases there are frequently seen the deformities at the orifices of the body, and there may also be a continued exfoliation of the epidermis in large irregular patches. After this has separated, the skin beneath appears red and moist, but gradually becomes dry, hard, and shining, slowly contracting until it splits in various directions. In a case recently under observation in the Babies' Hospital,* a picture of which is shown in the accompanying illustration (Fig. 185), it was stated by the mother that during the first ten months of life complete exfoliation of the skin had occurred in the course of every two or three months.

The outlook is bad in all cases; in most of the severe forms death occurs in infancy, but in some of the milder ones, life may be prolonged throughout childhood. The "alligator boy" of the Dime Museum is an example of this class.

Treatment.—The indications are to keep the skin moist and soft by the use of oils, continuous baths, etc., and to prevent infection by perfect cleanliness. Although a certain amount of improvement usually follows these measures, a cure is not to be expected.

MILIARIA.

The term miliaria is applied to an obstruction of the sweat glands, which may occur either with or without inflammation. The non-inflammatory form is known as *sudamina*, the inflammatory forms as *miliaria rubra*, *miliaria vesiculosa*, and *miliaria papulosa*.

Sudamina.—In this form there is no inflammation. The sweat ducts, according to Crocker, are blocked by an accumulation of epithelial cells while no perspiration is going on; and when the process is restored the fluid, being unable to escape, accumulates in the form of tiny vesicles. These appear like small pearly bodies very closely set, and disappear in the course of a few days by absorption. Fresh crops may appear from time to time. Sudamina may be seen in any of the continued fevers or exhausting diseases. It requires no treatment.

Miliaria Rubra.—This condition, also known as *red gum*, *strophulus*, etc., is a sweat rash, usually seen in young infants as the result of excessive clothing. It is most frequently observed upon the cheeks and neck, often upon the side of the face upon which the infant sleeps, or the side held against the mother's body while nursing, if this is done upon only one breast. The eruption consists of scattered red papules, sometimes with tiny vesicles. Miliaria rubra is an inflammation about the sweat glands, the result of which is a retention of their secretion. There is generally little or no itching. The treatment consists in the removal of the cause, and the application of some absorbent powder, such as boric acid and starch.

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* This case has been fully reported by Cabot, New York Medical Record, July 6, 1895. For fuller description of the disease, see Ballantyne, Diseases of the Fœtus, vol. ii, 1895; also Archives of Pædiatrics, April and June, 1894.

Miliaria Papulosa (Lichen Tropicus, Prickly Heat, etc.).—This is the most common and most important variety of miliaria. There is in this disease an obstruction of the sweat glands by inflammatory products. The lesion consists in the formation of bright-red papules, which are very closely set, the summits of some of them being surmounted by tiny vesicles, and here and there in severe cases even small pustules may be seen. If not interfered with by scratching, the vesicles dry up without rupture, and are followed by a slight desquamation. Where there is much scratching, an eczematous condition may result. Miliaria papulosa comes out with great rapidity, especially upon the neck, forehead, back, and chest. It is accompanied by an almost intolerable itching and stinging sensation. Over other parts of the body profuse perspiration occurs. The disease is produced by very hot weather and excessive clothing. Although the duration of a single attack is but two or three days, in susceptible patients it may keep recurring for weeks, being exceedingly intractable. Where there is much scratching the resulting eczema is very troublesome. It is not infrequently followed by furunculosis.

The diagnosis of miliaria rubra and miliaria papulosa is usually easy. They are distinguished from eczema by the suddenness with which they appear, by the associated sweating of other parts of the body, by the transitory character of the eruption, and by the fact that the rash never occurs in circumscribed patches. Prickly heat sometimes resembles the rash of scarlet fever, but the fact that the tiny papules are in some places crowned by vesicles and that constitutional symptoms are absent, usually make the distinction an easy one.

Treatment.—Prickly heat is to be prevented by light clothing, frequent bathing, and the plentiful use of a good toilet powder, such as boric acid and starch. During an attack, the bowels should be freely opened by calomel or a saline, and secretion of the kidneys stimulated by the use of citrate of potassium or the sweet spirits of nitre. The skin should be protected against the irritation of flannel undergarments by the interposition of silk or linen. When the inflammation is at its height, relief is obtained by the application of a calamine and zinc lotion (page 933), or by a dilute solution of the acetate of lead; carbolic acid may be added to either, where the itching is intense. In some cases powders are preferable to lotions. One of the best is the stearate or the oxide of zinc, twelve parts; bismuth, three parts; powdered camphor, one part; or equal parts of starch and boric acid may be used, or simply rice flour. All of these must be very freely applied. The diet should be light and fluid, and if milk is the food it should be considerably diluted.

eczema of the face and scalp among the manifestations of infantile "scrofula." We can not connect eczema with any single diathetic condition; but it is much more often seen in children with gouty antecedents than in others; or to state it differently, the most frequent manifestation of gout during infancy is the tendency to eczema. Children of rheumatic families are also prone to the disease. Eczema of the face is common in fat, healthy-looking infants, both in those who are nursing and in those who are artificially fed. It also occurs in poorly nourished children, but rarely in those suffering from marasmus.

The exciting causes of eczema may be external or internal. Of the former the most important are heat, cold dry air, and winds—as in the familiar chapping of the face—the use of hard water or of strong soaps in bathing. The disease may be due to the irritation of clothing, to want of cleanliness, or to irritating discharges from mucous surfaces, as in the eczema of the upper lip, thighs, or buttocks. It accompanies most of the parasitic skin diseases, particularly pediculosis, scabies, and ringworm.

What part is played by micro-organisms in the etiology of eczema has not yet been fully determined. The observations of Gilchrist and others seem to indicate that as a primary factor they are not of the first importance. Secondary infection, however, occurs in most of the cases, and is a factor of the greatest importance in keeping up the disease.

The internal causes of eczema are chiefly associated with deficient elimination from the kidneys and bowels, and digestive disturbances. It often accompanies chronic constipation where there is intestinal torpor and the white stools of deficient biliary secretion; and it is seen where the urine is scanty and concentrated because children partake too largely of solid food. The latter is true both in the first and second years.

Eczema may be produced by any form of digestive disturbance, but it is especially frequent in the intestinal indigestion which results from overfeeding, or the too early or excessive use of farinaceous food, or from breast milk in which the percentage of fat is very high. From personal experience in the post-mortem room, I can confirm the observation of Bohn regarding the frequency with which fatty liver occurs in very fat infants. Enlargement of the liver may sometimes be made out during life. It is highly probable that the interference with the hepatic functions which accompanies these fatty changes has much to do with the production of eczema in such subjects. In children fed upon cow's milk the excessive fat may be the cause, or it may be due to excessive proteids. Of farinaceous articles, the two which are most often to be blamed are potato and oatmeal. Although eczematous patients usually appear to be well nourished, it is rare that some symptoms of indigestion are not present.

Eczema is often due to some form of reflex irritation. Such are the cases which accompany dentition, and the rare ones due to genital irrita-

SEBORRHŒA.

Seborrhœa is considered by dermatologists generally, as a functional disease of the sebaceous glands; although Unna regards all such cases as inflammatory, and classes them as seborrhœic eczema, which is of parasitic origin (page 929). The disease may affect almost any part of the body, and children of any age, but the most frequent form is that which is seen upon the scalp in young infants. This is the most important variety, and the only one which will be here considered.

Seborrhœa of the scalp is characterized by the formation upon the vertex, of dirty-yellow crusts, which are soft, greasy, and friable. They are composed of epithelial cells, fat-globules, and granular masses, to which is always added dirt. In neglected cases the hairy scalp is nearly covered by a dense crust, which may be as thick as heavy pasteboard. If the crusts are removed the underlying scalp may be found perfectly healthy, but more frequently, in cases of long standing, it is eczematous. The eczema is set up by the decomposition of the exudation, or by the efforts to remove the crusts by such means as the fine-toothed comb, commonly employed in domestic practice. There is little tendency to spontaneous improvement or recovery, and the condition often lasts for months. Every seborrhœa should be treated, for when neglected it furnishes a favourable soil for the development of eczema.

Treatment.—Only local measures are required. The crusts are first to be softened with oil, and then removed by washing thoroughly with warm water and soap, after which an ointment of resorcin (resorcin, gr. x; ungt. aquæ rosæ, ℥j) or sulphur (precipitated sulphur, ʒj; lanoline, ℥j) should be applied. The oil and soap and water are repeated every few days, or as often as the crusts form. In the meantime the scalp is kept covered with the ointment.

ECZEMA.

Eczema may be defined as a catarrhal inflammation of the skin. It is the most frequent and altogether the most important disease of the skin in early life. The scope of the present work permits only a discussion of such features and varieties as are peculiar to infants and young children. The eczema of older children does not differ in any essential points from that of adults.

Etiology.—The conditions in infancy which predispose to eczema are, first, that the skin is extremely delicate, and hence more easily affected by external irritants and micro-organisms; secondly, its more intense glandular activity. While all children are susceptible, there are certain ones in whom the susceptibility is very marked, and in them the slightest amount of external irritation, or the most trivial disturbance of digestion may produce a severe eruption. It was formerly the fashion to class eczema of the face and scalp among the manifestations of infantile "scrofula." We can not connect eczema with any single diathetic condition; but it is much more often seen in children with gouty antecedents than in others; or to state it differently, the most frequent manifestation of gout during infancy is the tendency to eczema. Children of rheumatic families are also prone to the disease. Eczema of the face is common in fat, healthy-looking infants, both in those who are nursing and in those who are artificially fed. It also occurs in poorly nourished children, but rarely in those suffering from marasmus.

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Eczema is often due to some form of reflex irritation. Such are the cases which accompany dentition, and the rare ones due to genital irritaof secretions, or a seborrhœic inflammation. Intertrigo is seen in the folds of the groin, between the scrotum and the thighs, between the buttocks, about the anus, in the axillæ, in the neck, or behind the ears. Its essential causes are moisture, friction, want of cleanliness, and sometimes infection. The disease is generally seen in its worst form about the thighs, genitals, and buttocks; it sometimes covers the sacrum and extends down to the middle of the thighs. There is an intense uniform redness, and in some cases the epidermis is denuded over large areas, and the surface is moist. There is no thick crusting and little or no itching. Intertrigo is usually easy to control except in very poorly nourished or marantic children, among whom it is especially frequent.

Diagnosis of Eczema.—This is usually quite an easy matter. In the majority of cases, the disease affects the face or the scalp, and its appearances are typical. Eczema of the body or extremities may be confounded with scabies or syphilis, and occasionally with other forms of skin disease. Scabies resembles eczema in its intense itching and multiform lesions; but in the former, one may often find evidences of its presence in other members of the family; the parts most frequently affected are the flexures of the wrists, the elbows, the skin between the fingers, the margins of the axillæ, the lower part of the abdomen and back, and, in boys, the penis; and by careful examination with a lens some of the characteristic burrows are certain to be discovered.

Syphilis is likely to be confounded with papular eczema of the buttocks. The latter affects the parts near the anus, and the irritation may lead to the development of spots closely resembling mucous patches. The local appearances may at times be indistinguishable from syphilis, and the diagnosis is to be made only by the other symptoms present. In syphilis the characteristic eruption is seen usually upon the face, hands, legs, and sometimes the palms and soles; there is no itching and very little evidence of inflammation; the eruption is dark-coloured, and occurs as small circumscribed spots; there are usually present other symptoms, such as the coryza, the syphilitic cachexia, and enlargement of the spleen.

The diagnosis from pediculosis and ringworm of the scalp, rarely presents any difficulties.

Prognosis.—All cases of chronic eczema are tedious. There is only a slight tendency to spontaneous improvement, and very little to spontaneous recovery during infancy. In a given case, the prognosis depends upon the duration of the disease, its severity, and very much upon the co-operation of the mother or nurse. The results obtained depend not only upon the particular line of treatment adopted, but upon how well it is carried out. Usually it must be continued for several months. Eczema of the face is especially intractable when occurring in children suffering from chronic indigestion and constipation. Intertrigo is in most cases easily cured, unless the patient is suffering from marasmus.

Treatment.—It is never dangerous to cure an eczema, and always desirable to do so, in spite of the strong prejudice to the contrary, which still exists in the minds of the laity and in some members of the medical profession. The general tendency is to treat the eczema rather than the patient who is suffering from it. A judicious combination of general and local measures is necessary for the best results. One should first seek to discover and correct what is wrong with the child's digestion, assimilation, and elimination; unless nutritive disturbances can be removed, local treatment will give only temporary relief. External causes also must be investigated. The local measures employed must be chosen with reference to the condition present; stimulating applications should not be ordered for an acutely inflamed skin, nor sedative applications in very chronic conditions.

Diet.—A thorough investigation into the food is necessary, not only as to its character, but as to quantity and preparation, the manner and frequency of feeding, etc. If the patient is a nursing infant, an examination of the nurse's milk is indispensable to intelligent treatment. If the child is very fat and well nourished, it is generally the case that the fat of the milk is too high and must be reduced according to the rules given elsewhere (page 173), the most important thing being to exclude from the nurse's diet malt liquors and alcohol in all forms, and reduce the amount of meat. In a smaller number of cases the trouble is with the proteids of the milk; there will then be other signs of indigestion, such as colic, the appearance of curds in the stools, etc. The amount of food should be reduced by lengthening the period between the nursings, and shortening the time which the child is allowed to remain at the breast at one nursing. Plain water, or better, some alkaline water, should be given freely between the nursings. In children fed upon cow's milk, the trouble may be with the sugar, the proteids, or the fat. The physician should try the effect, first of giving a milk which is low in proteids and moderately high in fat (e. g., formula G or H, page 209) afterwards, one in which both fat and proteids are low (e. g., formula II or III, page 194). These and other changes are to be made in the manner described in the chapter on Infant Feeding. During the latter part of the first and the entire second year, the usual error is that of overfeeding with in most cases an excessive use of solid food, especially farinaceous articles. The diet should then be much reduced, and the amount of farinaceous food restricted, potatoes and oatmeal being absolutely prohibited. The diet which suits most children best is one composed of milk, beef juice, broth, fruit, eggs, and a little red meat, with the addition in some cases of rice, wheat, or barley. In severe and obstinate cases, however, as complete a change in diet as possible is sometimes the best prescription. Any form of indigestion which exists is to be managed according to the special indications in each case.

The diet of older children needs to be watched no less closely than that of infants. The general rules laid down elsewhere for feeding after the second year should be observed. The great majority of cases do best upon a diet which is largely fluid, and composed principally of milk or some of its substitutes—kumyss or matzoon.

Elimination by the kidneys should be stimulated by the very free use of water, to which it is well to add—especially in cases with a gouty tendency—the citrate,* or acetate of potassium, from ten to twenty grains daily.

Attention to the condition of the bowels is of the greatest importance. To overcome the tendency to constipation is in many cases to cure the eczema. Suggestions under this head will be found in the chapter on Chronic Constipation. Special importance is to be attached to the occasional use of a purge of calomel, one half to one grain being given every third or fourth night. The best effects from this are seen in over-fed children. It has a favourable effect upon the kidneys as well as upon the bowels. The bowels must not only be opened, they must be kept freely open by the daily use, if necessary, of some of the milder laxatives, such as phosphate of sodium, rhubarb, or cascara. Sometimes nothing acts so well as castor oil, which may be given in from half a teaspoonful to teaspoonful doses every night for two or three weeks at a time. It should be administered in emulsion.

When the disease occurs in flabby, anæmic, or poorly-nourished children, iron and bitter tonics are required, and occasionally alcohol and cod-liver oil. In other words, the child's general condition should be treated just as if no eczema existed. Arsenic is indicated in a chronic or recurring form of eczema with dry, scaly eruption. It is in no sense a specific remedy, but sometimes of great value.

The *general management* of cases is important. The skin must be carefully protected by an ointment whenever the child is in the open air; if the weather is very cold, or there are high winds, children with active eczema should not go out, but take the fresh air indoors. Never should an eczematous surface be washed with plain water, and much less with castile soap and water, so frequently employed by the ignorant. Where washing is necessary, it may be done with bran water, milk and water, or starch and water, to which borax (a teaspoonful to the quart) may be added. The clothing should not be so excessive as to keep the child constantly in a perspiration. Napkins should not be washed in strong soda solutions, nor, in case of eczema of the buttocks, should they ever be used a second time after being simply dried.

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* While the citrate can not be depended upon as a diuretic, unless dispensed from a newly opened bottle, it is generally to be preferred, as being more easily administered.

In eczema of the face it is absolutely necessary to prevent the child from scratching the parts. The use of a mask is not always sufficient, nor the wearing of mittens; nor is the local application of anti-pruritic lotions or ointments invariably successful. In severe cases mechanical restraint is absolutely indispensable. The most satisfactory method is to surround the arms at the elbows by pasteboard splints, and hold them in place by bandages. This allows free use of the hands, but makes it absolutely impossible for the child to reach the face.

Local treatment.—Local treatment is always necessary, for not only are the causes sometimes entirely external, but the condition may persist after the original internal cause has been removed. There are several indications to be met by local treatment at different stages in the disease: (1) To remove crusts and other inflammatory products; (2) to allay congestion and acute inflammation; (3) to relieve itching; (4) to protect the delicate new skin which is forming; (5) to prevent infection; (6) to stimulate the skin in the chronic stages of the disease.

Preparatory to the use of any application, the scales, crusts, and other products of inflammation must be softened and removed in order that the diseased surface may be reached. In most cases it is sufficient to soften the crusts by the use of olive oil for twelve or twenty-four hours, and then remove them by soap and warm water. If the crusts are very hard and thick, they can be softened by a poultice. During the stage of acute inflammation only sedative applications should be used. One of the best of these is a lotion of zinc and calamine:

℞ Pulv. calaminæ preparatæ	ʒ ij
Zinci oxidi	℥ ss.
Glycerinæ	℥ j
Liquor calcis	℥ ij
Aquæ rosæ	℥ viij.

A piece of muslin should be dipped in this solution, and applied to the affected part, being kept in place by a bandage. If there is much itching, one per cent of carbolic acid may be added.

Another plan of treatment, where there is much secretion, is to keep the surface covered with equal parts of boric acid and starch or dolomol powder. An application which is often successful in allaying the intense burning and itching is black wash. This is applied several times a day in full strength or diluted and allowed to dry on, after which a protective ointment is used.

A soothing application in general eczema is one composed of equal parts of lime water and sweet-almond oil; sometimes this may be advantageously followed by smearing the body with a thick starch paste and allowing it to dry on.

As a simple protective ointment, one containing starch, zinc oxide, or bismuth, either alone or in combination, may be used. An excellent formula is Lassar's paste:

℞ Acidi salicylici	gr. x
Zinci oxidi	ʒ ij
Amyli	ʒ ij
Vaseline	℥ j

Later, when the inflammation is less acute and the itching severe, nothing is so generally useful as a combination of tar and zinc, as in the following:

℞ Ungt. picis liquidæ	ʒ iij
Zinci oxidi	ʒ iss.
Ungt. aquæ rosæ	ʒ vi

For more chronic cases, the amount of tar may be increased. All ointments used should be spread upon muslin, and kept in close contact with the inflamed part by means of a bandage or mask. Little or nothing is accomplished by simply rubbing the ointment upon the affected part. Where it is difficult to keep a mask applied, or in situations where it is impossible to use the ointment, Pick's paste may be tried:

℞ Pulv. tragacanthæ	ʒ j
Glycerinæ	ʒ iss.
Aquæ rosæ	℥ iv

To this may be added zinc oxide gr. xl and carbolic acid gr. v, or tar ♏ x. A similar basis for ointments, made from gum tragacanth has been suggested by Elliot and is known as bassorin paste. It may be combined with tar, zinc, salicylic acid, or resorcin.

The methods of treatment above mentioned are especially applicable to eczema of the face and scalp. For pustular eczema of the scalp the best application is the white-precipitate ointment, which should be combined with three or four parts of vaseline. This is excellent also for small eczematous patches upon the body, but it is not to be used over a large surface.

In intertrigo, the treatment should have reference to the pathological condition which is present. Cases of simple erythema usually yield promptly to cleanliness and the free use of absorbent antiseptic powders, such as boric acid and starch in equal parts, or if the skin is very sensitive, aristol or dolomol with aristol may be used. If there is an acute dermatitis, the calamine and zinc lotion may be used, and later some protecting ointment. When infection has been added, lotions of resorcin or ichthyol, one half or one per cent strength, should first be applied, and the skin then covered with one of the powders mentioned; both are to be repeated as often as the parts are wet by urine or soiled by fæces. It is important in all cases that the diseased surfaces should be kept separated, which is best done by starch or aristol and absorbent cotton. All napkins should be immediately removed when soiled.

In cases of chronic eczema, where the skin remains thickened, red, scaly, and itching, stimulating applications are to be used, such as the tincture of green soap or stronger preparations of tar than those mentioned. They should be applied every three or four days.

In the seborrhœic form of eczema, whether affecting the face, scalp, or body, nothing is so generally useful as resorcin:

℞ Resorcin	gr. x
Ungt. aquæ rosæ	℥j

This may also be advantageously combined with bassorin paste.

FURUNCULOSIS.

A furuncle, or boil, is a circumscribed inflammation of the subcutaneous cellular tissue, usually beginning in a hair follicle, and usually ending in suppuration. When severe, it may result in necrosis of the follicle, which forms the "core," or the necrotic process may extend to the surrounding tissues for a variable distance. The ordinary boil need not be described, as it presents nothing peculiar in early life. The condition, however, which is characteristic of young children is the formation of small ones in great numbers. It is to this more especially that the term *furunculosis* is applied. The principal location of these small abscesses is, in nearly all cases, the scalp, face, and shoulders, although they may be found upon any part of the body. They are sometimes numbered by hundreds, and appear in crops for a period of several months. In size, they usually vary from a pea to an almond, and they rarely contain a core. Infants are much more often the subjects of this disease than are those who have passed the second year. In the great majority of cases the condition is not serious, yet, occurring, as it often does, in infants who are already suffering from extreme malnutrition or marasmus, whose tissues possess but little resistance, the process may develop into a gangrenous dermatitis, which may prove fatal.

Furunculosis is seen in children who are in other respects apparently healthy, even robust; but the majority are in a more or less debilitated condition, and often are the subjects of digestive disturbances. The disease is quite frequent in syphilitic infants; but these simple abscesses are to be sharply distinguished from those which result from the breaking down of gummata of the skin. Want of cleanliness of the skin is a factor of some importance in producing the disease. Furunculosis may be associated with eczema. The exciting cause in all cases, as shown by all recent investigations, is the entrance of the staphylococcus pyogenes aureus, sometimes with other organisms, into the follicles of the skin.

Treatment.—The internal treatment is to be directed toward any disturbance of digestion or general nutrition which is present. General tonics are indicated in most cases, particularly iron, arsenic, and the compound syrup of the hypophosphites. But little reliance can be placed upon internal remedies, such as sulphide of calcium, for the purpose of arresting this disease. Local treatment should have for its first object thorough cleanliness of the skin. This is best secured by frequently bathing the parts affected with a saturated solution of boric acid. Single furuncles may often be aborted by the frequent application of spirits of camphor, or a few applications of tincture of iodine, or by touching them with pure carbolic acid. The last mentioned, although efficient, can hardly be intrusted to the hands of a mother or nurse. A remedy which has been used with considerable success is a plaster of salicylic acid. In my experience the best plan of treating the multiple small furuncles, is to delay incision until they have pointed, then to incise freely and empty the follicle completely by compression. It is then washed out thoroughly with a solution of bichloride (1 to 2,000), and small pledget of absorbent cotton applied till the bleeding has ceased. After this the part should be covered with simple collodion or that in which iodoform has been dissolved. Where the abscesses are of large size and upon the scalp, it is wise to make compression by applying a snug bandage for a day. It is very exceptional for abscesses so treated to refill. When the suppuration is more diffuse and there is necrosis of the cellular tissue, ichthyol, either in the form of an ointment or lotion (one to five per cent strength), is one of the best applications. Early and free incisions must be practised in all such cases.

GANGRENOUS DERMATITIS.

This is not a frequent disease, and is seen almost exclusively in infancy. It may be primary or it may follow other diseases, and hence has been described under many different names—viz., *varicella gangrenosa*, *ecthyma gangrenosa*, *pemphigus gangrenosa*, etc.

The lesion consists in small, discrete areas of inflammation of the skin, ending in necrosis. In the primary cases there is usually first seen a vesicle, about as large as a pea, with a dusky areola; it increases in size and becomes a pustule. Crusts form which are quite adherent, and on removing them a loss of tissue is seen. The ulcers usually have sharp but not undermined edges, often presenting a "punched-out" appearance. By the coalescence of several small ones, ulcers an inch or more in diameter are sometimes formed.

The primary form of gangrenous dermatitis occurs in wretched, poorly-nourished infants, and is most often seen upon the buttocks. In this location it may be mistaken for syphilis. The secondary form is more common, and usually follows varicella, less frequently vaccinia, measles, or pemphigus. My own experience with this disease has been confined to cases following varicella. In such, the lesion is usually seen upon the upper half of the body, especially upon the neck and chest. It follows the ordinary lesions of varicella and continues usually, in spite of treatment, from one to four weeks, in most cases ending fatally. The disease always occurs in infants of poor vitality, often in those suffering from marasmus, and is seldom seen outside of institutions. It may be accompanied by fever, and other severe constitutional symptoms.

For the production of the disease, two factors are necessary: first, the constitutional condition referred to; and, secondly, the entrance of pyogenic germs, usually the streptococcus pyogenes.

Treatment.—Every means possible should be employed to build up the general health of the infant by tonics, fresh air, careful feeding, etc. Locally, strict cleanliness and antiseptic applications are necessary. The best application is a solution of bichloride (1 to 5,000), or an ointment of ichthyol or iodoform.

IMPETIGO CONTAGIOSA.

Impetigo contagiosa is a disease characterized by the formation of discrete vesiculo-pustules, occurring most frequently upon the hands and face. Cases are usually seen in groups affecting several children in one family or institution. It may be communicated from one person to another, and spread by auto-inoculation from one part of the body to another.

One rarely has an opportunity to see the disease until vesicles have formed. These are usually from one fourth to one half an inch in diameter, and are flaccid, never distended. Later, their contents become slightly yellowish; then they rupture and dry, forming thick yellow crusts, which have the appearance of being "stuck on," the surrounding skin being quite healthy. After the crusts fall off, a small red patch remains, which slowly fades. The true skin is not involved, except in poorly-nourished, cachectic subjects, as a result of continued local irritation, like scratching. Under such conditions ulceration may occur. Instead of the small vesiculo-pustules described, bullæ from one to two inches in diameter may form, filled first with serum, afterward with sero-pus. Very little inflammation is seen about these patches, and in most cases the intervening skin is normal.

The favourite seat of the eruption is the face, especially about the chin, next the hands, the neck, the feet and legs, the forearms, and the scalp; it is rarely seen upon the abdomen, and never upon the back. There may be only half a dozen vesiculo-pustules, or from thirty to forty may be present. The smaller ones sometimes coalesce and form others of considerable size. Itching is never a prominent symptom, and in most cases it is absent altogether.

The usual duration of impetigo contagiosa is two or three weeks; it, however, runs no regular course, and by continued auto-inoculation may last much longer than this.

The studies of Gilchrist (Baltimore) point to a streptococcus of low virulence as the cause of this disease. European investigators, however, have with considerable uniformity found the staphylococcus pyogenes aureus in the vesicles. Impetigo contagiosa may occur in any child, but is seen most frequently in one who is poorly nourished.

The diagnosis is not often difficult, and is made by the following features—viz., the occurrence of several cases together, the isolated vesiculopustules situated upon the face and hands, the slight itching, and the prompt cure by local measures only. The bullous form, however, is frequently confounded with pemphigus; many cases in which the diagnosis of pemphigus is made are examples of impetigo.

Treatment.—This is simple and usually very effective. The crusts are to be softened and removed by thoroughly washing the part with soap and water or a bichloride solution, after which the white precipitate ointment, combined with three parts of vaseline, should be applied.

URTICARIA.

Urticaria is a frequent disease in early life, and presents some features, particularly in infants and young children, which are quite different from those seen in adults. This is due to the fact that papules and vesicles, and occasionally pustules, are associated with the wheals. As the wheals quickly subside, it frequently happens that the other lesions mentioned are the only ones present. This fact has given rise to considerable confusion in names, and the urticaria of infancy has been called *lichen urticatus*, *urticaria papulosa*, *strophulus*, etc. It is now pretty generally agreed that the clinical picture, which is a familiar one, belongs to a single disease, and that this is urticaria.

The initial lesion is the wheal, but on account of the extreme susceptibility of the skin in young children, the process is more intense than in older patients, so that it may result in the formation of an inflammatory papule or a vesicle. In a few hours the wheals may subside, and only the papules or vesicles remain, and without a good history the disease may be a very obscure one. The papules and vesicles occur with greatest frequency upon the hands and feet, particularly the palms and soles. The more severe form of the disease in poorly nourished children is sometimes accompanied by a pustular eruption, and there may even be deep ulceration (ecthyma). The usual appearance of the eruption is a number of small inflamed red papules whose tops are covered with scabs, the result of scratching. The eruption may be limited to the extremities or it may be general. It is as a rule more severe in regions accessible to scratching.

There is usually severe itching, which leads to loss of sleep, and often in this way the disease affects the general health of the child. The urticaria of older children does not differ essentially from the same disease in adults.

The character of the eruption in urticaria and even its distribution strongly suggest scabies; and unless one has had an opportunity to witness the development of the lesions, a differential diagnosis may be very difficult, as almost every lesion, except the wheal, may be identical in both diseases. Other cases may resemble varicella.

Urticaria in early life is most frequently the result of some disturbance in the digestive tract. Almost any sort of derangement may produce it, the exciting cause varying with the patient. Exceptionally, it may result from other forms of irritation, such as dentition or intestinal worms, and it has been ascribed to malarial poisoning.

Treatment.—The milder forms of urticaria usually respond quickly to treatment; but when it is severe and has existed for several weeks, it is one of the most troublesome and intractable skin diseases of childhood. The treatment is to be directed primarily toward the condition of the digestive organs. Children should be put upon a milk diet, and even milk may need to be partially peptonized. The bowels should be kept freely open by calomel, a nightly dose of castor oil, or a morning dose of magnesia. If the urine is excessively acid and scanty, alkaline diuretics should be given. The drugs most useful for the indigestion with which urticaria is associated are salicylate of soda and nitro-muriatic acid, each of which is to be given after meals.

All local causes of irritation, such as rough flannel underclothing, should be removed. The sleep may be so much disturbed as to require the use of trional or bromide and chloral. The two remedies which are of most value for the disease itself are antipyrine and atropine; they may be used separately or in combination, and should be administered in moderately large doses.

The local irritation and itching may be relieved by a lotion of menthol (gr. ij, water ℥ j), by a very dilute solution of the subacetate of lead or carbolic acid, or by a mixture of vinegar, or the fluid extract of hamamelis, and water. Where pustules are present, the white-precipitate ointment may be used, combined with four parts of vaseline; in the papular and vesicular forms, an ointment of ichthyol or naphthol, one per cent strength. In many cases the improvement in the general health by the use of tonics, change of air, etc., will accomplish more than any measures directed especially to the relief of the urticaria.

SCABIES.

Scabies is a contagious disease due to the burrowing into the skin of the female acarus, with secondary lesions which result from scratching. This disease is not a common one in New York, even among dispensary patients, while among the better classes it is extremely rare.

The burrowing of the acarus is usually where the skin is thinnest—viz., between the fingers, on the flexor surfaces of the wrists, the axillæ, and, in males, the genitals. It is not seen upon the face, except in infancy, when it may be infected by contact with the breasts of the mother.

The lesion excited by the acarus is usually a papule or a vesicle, sometimes a pustule. In some cases no evidences of inflammation are present, but in infants and young children they may be marked—pustular eruptions being frequent and often extensive, especially upon the hands and feet. The characteristic burrow is from one fourth to one half inch in length, and appears as a fine brown or black line, at the end of which the acarus may be discovered as a small white speck. The burrows are often difficult to find in infants. They are generally to be seen along the inner border of the hand and between the fingers. The intensity of the inflammatory lesions varies greatly in different cases; in some they are very few, while in others, particularly in delicate, cachectic, and neglected children, they are sometimes very severe, so that the skin of the affected part is nearly covered with pustules. These secondary lesions are due to infection by the streptococcus or staphylococcus. A pustular eruption upon the hands should always suggest scabies. The lesions which result from scratching may be found on any accessible portion of the body. There are usually at first linear, bloody marks, but after a time these may not be visible. In little children urticaria is often associated.

The diagnosis of scabies is usually quite easy, as several children in a family are likely to be affected, particularly if they occupy the same bed. The diagnostic features of the eruption are the presence of papules, vesicles, or pustules, especially upon the hands, wrists, and genitals. A careful examination with a lens will usually disclose some of the characteristic burrows, or even the acarus. In infancy, scabies may be easily confounded with the vesicular form of urticaria, unless the development of the lesions has been observed.

Scabies may always be cured, provided sufficient precautions are taken to prevent re-infection. This necessitates boiling or baking, not only the patient's clothes, but all the bedding as well.

Treatment.—This should always be begun by a hot bath, in order to soften the epithelial scales about the burrows. The body should be thoroughly scrubbed with soap and water, preferably with a nail-brush, the bath being continued for at least half an hour. It is well to do this at night. After the bath, the body is anointed with the parasiticide, which should be thoroughly rubbed into the skin, clean clothing applied, and the child put into a perfectly clean bed. In the morning the ointment may be washed off, but none of the clothing previously worn should be put on. This treatment is to be repeated on two or three successive nights, and if thoroughly done it will effect a cure. The ordinary sulphur ointment is too irritating for use in little children, and one of the following may be substituted: naphthol, 15 parts; creta preparata, 10 parts; vaseline, 100 parts (Kaposi); or, precipitated sulphur, 1 part; balsam of Peru, 1 part; vaseline, 8 parts; or the simple balsam of Peru may be applied without dilution. After the use of the parasiticide there is generally required for a few days, some soothing application like those mentioned in the chapter upon Eczema.

TINEA TONSURANS—RING-WORM OF THE SCALP.

Ringworm of the scalp is a very frequent disease in institutions for children, often occurring as an epidemic. According to Crocker, the primary lesion consists in a red papule surrounding a hair, which soon increases to a small circular patch; this spreads at its outer margin, gradually increasing in size until it is from one to two inches in diameter, but rarely larger than this. Sometimes several of the patches coalesce. These affected areas always have rounded borders, and are sharply outlined. Here the hairs are very brittle, and often broken off close to the scalp, so that it may appear to be bald. Where they have not fallen off, the hairs have lost their lustre. The stumps of the broken hairs point in all directions.

The fungus which produces the disease is the *trichophyton tonsurans.* It penetrates the shaft of the hair, both the spores and the mycelium being seen under the microscope. The spores are present in great numbers in the hair, but the mycelium is most abundant in the scales. The amount of inflammation found in the diseased areas varies much in the different cases. There may be only a scaliness of the scalp, or a formation of pustules in the hair follicles, the hairs loosening and falling out in consequence. In young infants where the hair is scanty and thin, the disease resembles tinea circinata—i. e., it is superficial, and the hair follicles are often not involved. Children of all ages are liable to tinea tonsurans. It flourishes particularly in those who are dirty and poorly cared for.

The diagnostic feature of the disease is the presence of scaly patches, with loss of hair. The patches are usually circular, and by examination with a lens the stumps of broken hairs are seen all over the diseased area. By a microscopical examination the fungus is discovered. In typical cases the diagnosis is easy if the process is at all advanced, but there are many atypical forms and many mild cases where the recognition of the disease is difficult. The symptoms are often masked by the inflammatory conditions present. The disease may be confounded with seborrhœa; but in the latter the lesion is diffuse, never sharply defined; there is general thinning of hair over the scalp, and never the stumpy, broken hairs. Psoriasis has points of resemblance, but it is usually found on other parts of the body, especially the knees and elbows, and upon the scalp the patches are more numerous and smaller. In eczema the loss of hair in circumscribed patches is never seen, nor are the broken stumps.

Tinea tonsurans is always curable, provided the patient can be kept under close surveillance, and treatment thoroughly carried out. There is no tendency to spontaneous recovery. In a recent case, treatment must usually be continued for one or two months, and in chronic cases, from six months to one year, with the closest watchfulness.

Treatment.—The great difficulty in treatment is to get the parasiticide deeply enough into the scalp to reach the fungus, since this is often at the very bottom of the hair follicles. As a first step, the hair should be cut short all over the patch and for at least an inch beyond it; this is necessary in order to get at the diseased part and to detect new foci of infection early—if possible before the fungus has extended deeply into the follicles. The parasiticide should be applied not only upon but around the patch, and the entire scalp should be washed thoroughly two or three times a week. To prevent the disease spreading, all the scales are to be kept softened by the use of carbolic soap. The hair should not be brushed, as this tends to scatter the spores and spread the disease. All patients while under treatment, should wear a cap of muslin or oiled-silk, or one lined with paper, in order to prevent infecting others. In institutions, affected children should invariably be isolated.

To destroy the fungus almost every germicide on the list has been advocated at one time or another, which proves that the disease is a very obstinate one, and that no one application is invariably successful. Those which have the sanction of the widest use are the tincture of iodine, the bichloride, white precipitate, and oleate of mercury, kerosene, creosote, and croton oil. As a vehicle for ointments, lanoline is greatly to be preferred to vaseline or lard; according to Crocker, the addition of three parts of lanoline to one part of olive oil is much better than lanoline alone. Most of the germicides mentioned are used in the strength of one to five per cent, according to the age of the child and the irritability of the scalp. In an epidemic of ring-worm in the New York Infant Asylum the following combination of bichloride and kerosene proved extremely satisfactory: ten grains of the bichloride were dissolved in alcohol, and to this were added two and a half ounces each of olive oil and kerosene. This was applied every day, being thoroughly rubbed into the diseased patches, and the whole scalp saturated with it. Considerable irritation usually resulted, and every few days the parasiticide was omitted and some simple emollient applied until the irritation had in a measure subsided. In some of the cases, the tincture of iodine was alternated with the bichloride and kerosene. Twenty-six cases were treated after this plan and all cured, the average duration of treatment being eight and a half weeks.*

Epilation is necessary in many cases as an accessory to the application of germicides, particularly in older children.

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* A full report of these cases was made by C. G. Kerley, M. D., in the New York Medical Journal, October 10, 1891.

CHAPTER VI.

ACUTE OTITIS.

Otitis is a frequent affection during infancy and early childhood, attacks usually occurring in the cold season. Of all the inflammatory conditions which may be met with in early life, there is perhaps none which more frequently gives rise to obscure febrile symptoms than this.

Etiology.—Acute otitis is, as a rule, a secondary disease, and is generally preceded by some infectious process in the rhino-pharynx. The usual avenue of infection is through the Eustachian tube. Downie (Glasgow) gives the following statistics of 501 cases of tympanic involvement:

Originated during	measles	131	cases, or	26·1	per cent.
" "	scarlet fever	63	" "	12·6	" "
" "	whooping-cough	15	" "	3·0	" "
" "	mumps	3	" "	0·6	" "
" "	simple catarrh	147	" "	29·4	" "
" "	dentition	101	" "	20·0	" "
Syphilitic		8	" "	1·6	" "
Doubtful		33	" "	6·7	" "
		501		100·0	

The most severe forms of otitis usually follow scarlet fever, epidemic influenza, measles, diphtheria, or pneumonia. The entrance of fluids through the Eustachian tube from the nasal douche or nasal syringing may cause acute otitis. It sometimes results as an extension of inflammation from meningitis, especially the cerebro-spinal form.

The micro-organisms concerned in the production of acute otitis vary with the condition of which it is a complication. With scarlet fever, measles, influenza, or simple catarrh, the streptococcus, the pneumococcus, or the staphylococcus may be found either separately or together, inflammations associated with the organism last mentioned being usually of a milder character than with the other two. In cases complicating diphtheria, the Klebs-Loeffler bacillus may be found with any of the forms mentioned, or may occur alone. In chronic cases any of the pyogenic organisms may be present, and not very infrequently the tubercle bacillus.

Lesions.—The ordinary course of events in the pathological process is, first, acute hyperæmia and swelling of the mucous membrane of the rhino-pharynx, which extends into the Eustachian tube, causing obstruction more or less complete. The inflammatory process may be limited to the tube, or it may extend to the mucous membrane lining the middle ear.

There are two varieties of acute inflammation of the middle ear: (1)

The catarrhal form, which usually accompanies simple catarrh of the rhino-pharynx or complicates measles. This is an inflammation of the mucous membrane merely, and its products are serum and mucus or muco-pus. It is not usually accompanied by great pain or followed by serious consequences. It is generally confined to the lower part of the tympanic cavity, and is the form more frequently seen in infants. (2) The phlegmonous form, which affects older children principally. This is a much more serious inflammation, and is often excited by the infectious catarrh of scarlet fever, diphtheria, or epidemic influenza. In this variety micro-organisms find their way into the middle ear in great numbers, and set up an inflammation of a more or less virulent type, which may involve not only the mucous membrane lining the tympanum, but also the cellular tissue in the upper part of the tympanic cavity.

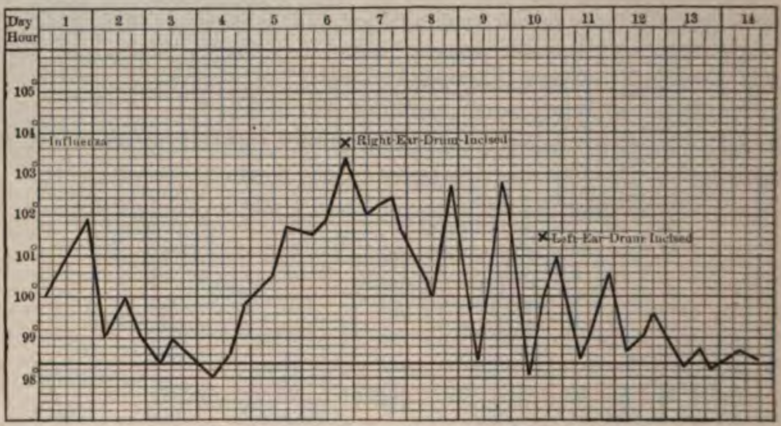

FIG. 186.—Temperature chart of acute otitis following influenza, in a child three years old.

The catarrhal form of inflammation frequently subsides in a few days with proper treatment, the only result being a slight deafness, which is temporary. The phlegmonous form causes a stoppage of the Eustachian tube, rupture or sloughing of the tympanic membrane and discharge of the products of inflammation, or rarely pus finds an outlet by burrowing between the cartilages. The inflammatory process may extend to the bones, causing necrosis of the ossicles or the bony walls of the tympanum. The remote results are periostitis and necrosis of the petrous bone, pachymeningitis, infectious thrombosis of the lateral sinus, general purulent meningitis, and cerebral abscess. These will be considered under Complications.

Symptoms.—These are usually few in number, but present great variability as regards their combination and intensity. The two most constant symptoms are pain and fever. In a typical case in an infant, there is generally at the beginning some discharge from the nose, slight congestion of the pharynx and tonsils, and a temperature of 100° to 102° F. There is nothing characteristic about this catarrh. After two or three days the objective symptoms subside, but the infant continues to be restless, worries much of the time, wakes frequently at night with a start, nurses poorly, and if the thermometer is used, it is found that the temperature remains elevated, usually from 100° to 103° F. (Fig. 186). The infant seems decidedly ill, and yet no very definite symptoms are present. Sometimes there is marked tenderness about the ear, and the child refuses to lie upon the affected side, or shows signs of pain when the ear is touched. After a week or ten days a discharge is found in the auditory canal, and usually there follows a rapid subsidence of the constitutional symptoms. In some cases there is seen only a high temperature, ranging from 101° to 104° F., which persists for several days without outward evidences of pain or other signs of inflammation, the discharge being the first symptom which leads the physician to suspect disease of the ear. In other cases there is marked dulness, apathy, anorexia, and sometimes nausea and vomiting, but for several days no evidence of pain; the temperature may be but little elevated. Thus, in most of the attacks seen in infancy, pain is not very marked, and it is this which so often leads to the great obscurity of the symptoms.

In older children the symptoms are more characteristic. Pain is usually sharp and severe, and is complained of early in the attack. The temperature is nearly always elevated two or three degrees, and occasionally it is 103° or 104° F. (Fig. 187), with severe headache, extreme restlessness, and even delirium or convulsions, so that meningitis may be suspected.

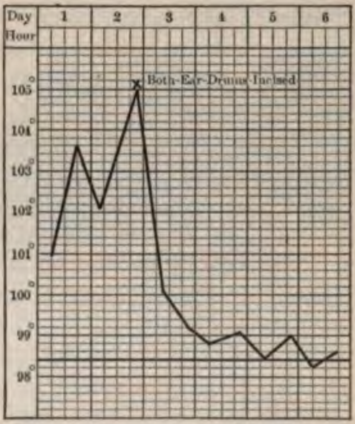

FIG. 187.—Temperature chart of acute otitis aborted by early paracentesis. Boy nine years old; attack followed a mild catarrh; severe pain in both ears began in afternoon of second day. Both drum membranes found acutely congested and bulging; incision followed by free hæmorrhage and immediate relief of pain. Ears syringed with bichloride solution; no suppuration occurred; patient well on fifth day.

The inflammation does not necessarily go on to suppuration and rupture. There are even more frequently seen, accompanying ordinary head-colds or mild attacks of influenza, cases in which the pain is quite severe for twenty-four or thirty-six hours, and accompanied even by a moderate elevation of temperature, and yet which rapidly subside without further symptoms. In these cases the pain is too constant and too prolonged to be an attack of neuralgia. They are simply cases of a mild form of inflammation.

In infants suffering from severe malnutrition or marasmus, otitis often comes on without any objective symptoms, the first thing noticed being the discharge.

Of all the symptoms, fever is the most constant, and is present in all except the cases just mentioned. The usual range of temperature is from 100° to 102° F.; exceptionally it may be from 103° to 105° F. The course of the temperature is irregular. After spontaneous rupture or incision of the drum membrane the temperature usually falls, but often not immediately. Pain is more marked in older children than in infants: first, because in the latter the drum membrane is not so firm, yields more readily, and ruptures earlier; and, secondly, because the inflammation is usually of the catarrhal and not the phlegmonous type. Tenderness is sometimes elicited by pressure just in front of the external auditory meatus; there may be increased sensitiveness of all parts of the ear and even of the whole side of the head. Children often complain of noises in the ear. One little girl with obscure symptoms and high temperature, first called attention to her ear by the remark, that she "heard pussy in the room." Cerebral symptoms are infrequent, and occur chiefly in cases not receiving proper early treatment; they may indicate meningeal congestion, or less frequently localised meningitis or thrombosis.

In secondary otitis, especially when complicating severe scarlet fever, diphtheria, measles, or typhoid fever, all subjective symptoms are frequently wanting; unless the ears are examined the disease may be overlooked until rupture has taken place.

The local appearances in the early stage—provided a view of the tympanic membrane can be obtained—are marked redness and congestion; later there is distinct bulging. If perforation has taken place, its site may or may not be visible, but its existence may be assumed if bubbles of air are seen deep in the canal, and if much mucus or pus is present, as inflammation of the external canal seldom causes much discharge. The pus sometimes burrows between the cartilages and opens externally behind or at the side of the ear. In the catarrhal form, the discharge is at first sero-mucus and quite profuse, later it is purulent. In the phlegmonous form it is always purulent, and liable to a sudden arrest with an increase in the constitutional symptoms.

Diagnosis.—Otitis in infancy is frequently obscure, because the patient is too young to direct attention to the seat of pain, or because the pain is slight or absent. The temperature is almost invariably elevated, and the usual problem presented is to discover a cause for this fever. In the absence of definite otoscopic signs, one must rely upon the presence of faucial congestion, a history of a previous acute catarrh, restlessness and the absence of signs in the throat, lungs, or digestive tract, which might explain the fever. Local tenderness, deafness, or noises in the ears are of much significance when present. Otitis is so common a cause of high temperature in infants during the cold season, that one should always have it in mind.

Complications and Sequelæ.—Remote consequences are most likely to be seen in cases following scarlet fever, probably because of their severity, particularly when early treatment has been neglected.

Mastoiditis.—This is the most frequent complication of acute otitis. In infancy the mastoid process is small and contains but a single cavity, the mastoid antrum, which communicates directly with the vault of the tympanum. It is probable that in every severe case of acute suppurative otitis there is some pus in the antrum. This is usually discharged into the middle ear after the tympanic membrane is incised or ruptures spontaneously. The principal cause of mastoid involvement is want of proper early treatment in acute otitis, particularly the practice of allowing these cases to take their natural course instead of securing early drainage by incision of the drum membrane.

The important symptoms of acute mastoiditis are fever, mastoid tenderness, and swelling. If mastoiditis develops rapidly after acute otitis the temperature may be high —103° to 105° F.; if it develops gradually and appears late the temperature may be scarcely above 100°. Abrupt cessation of an ear discharge should always arouse suspicion. It is always difficult to determine the presence of a slight amount of mastoid tenderness, but persistent tenderness of one side only is significant. It is often most marked close behind the auricle just over the antrum. The early swelling is due to œdema; later there may be an accumulation of pus. Post-auricular abscess causes a very characteristic swelling, the ear standing out from the head (see Fig. 188). It is usually due to spontaneous rupture through the outer bony wall just over the antrum; it may occur where there has been no discharge from the ear. It is a frequent result of severe cases of acute mastoiditis not operated upon, especially in young children.

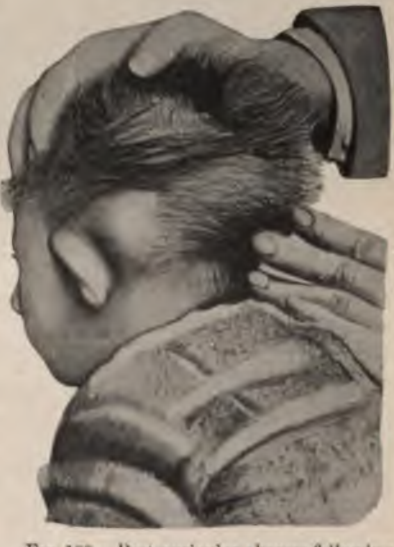

FIG. 188.—Post-auricular abscess following acute otitis.

The characteristic otoscopic appearances of acute mastoiditis, according to Bacon, are, bulging of Shrapnell's membrane, and drooping of the upper posterior wall of the external meatus.

Meningitis.—This may be a cause of death in young children. There may be a localised pachymeningitis with the formation of pus—an epidural abscess—or less frequently general purulent meningitis. It may be secondary to other lesions, such as thrombosis of the lateral sinus, or the rupture of a cerebral abscess, but is usually due to infection through the roof of the tympanum, or along the internal auditory meatus. Meningitis may occur either with acute or chronic cases. Its symptoms are those of a severe acute secondary meningitis; its duration is short; its termination, almost invariably in death.

Cerebral Abscess.—This is due to a direct extension of the infection from the bone, veins, or dura mater. In about two thirds of the cases the abscess is in the temporo-sphenoidal lobe. The next most frequent seat is the lateral lobe of the cerebellum. Körner states that disease of the mastoid and middle ear leads to cerebral abscess, and disease of the labyrinth to cerebellar abscess. Abscesses may be complicated by thrombosis or by meningitis. They are often latent until just before death, which more frequently occurs from the development of purulent meningitis than from any other cause. They are rare except in otitis of long standing.

Thrombosis of the lateral sinus may be simple or septic. In the former there is occlusion of the vessel by a fibrinous clot; in the latter there are in addition micro-organisms.

Simple thrombosis causes no important symptoms. Septic thrombosis is relatively infrequent and causes very marked and severe symptoms. It follows operation upon the mastoid, and occurs as a complication of mastoiditis quite apart from operation. The temperature is usually of a high and widely fluctuating type, and there may also be chills. In some cases the constitutional symptoms, except fever, may not at first be severe but may suddenly become very grave. Marked cerebral symptoms often develop rapidly, and death may follow in from twelve to twenty-four hours. At autopsy there may be found a soft broken-down clot in the sinus, which may extend into the jugular. It may be followed by secondary lesions of a general pyæmia, or by localised or general meningitis.

The labyrinth is not frequently involved, although cases are recorded by Pye, Phillips, and others, in which the necrosis and discharge of the entire labyrinth has occurred after scarlet fever. In most of these cases the deafness was complete, and in several vertigo was present.

Facial paralysis rarely occurs in the acute cases, but accompanies a considerable proportion of the chronic ones. It is due to an extension of the inflammatory process from the bone to the seventh nerve, where it passes through the canal. The symptoms are those of ordinary peripheral facial palsy.

Treatment.—Something may be done in the way of prophylaxis. It is of the first importance to secure a normal condition of the mucous membrane of the naso-pharynx by the removal of enlarged tonsils, adenoids, etc. The occasional attacks of earache accompanying these conditions are pretty sure to be followed by more serious trouble unless they are relieved. Whether during attacks of measles or scarlet fever, much can be done to prevent otitis, is still a mooted question. Personally I believe the risks of infection of the middle ear when judicious nasal syringing is employed are less than when nothing is done to cleanse the naso-pharynx.

The medical treatment of acute otitis aims at the relief of pain and arrest of the inflammation. If the case is seen in the early stage, the inflammation may sometimes be cut short by local blood-letting, the use of heat, and free catharsis. Blood-letting is not to be advised in the case of infants, but may be used in older children. Either leeches or wet cups may be employed. They should be applied just in front of and close to the tragus. Dry heat is to be preferred to moist heat, both as a means of arresting inflammation and of relieving pain. It may be applied by means of a bag of hot water, salt, or bran, or by a hot brick or soapstone. These may be placed beneath a thin pillow, upon which the child's head rests. If the child will not lie upon his hot pillow, a small bag of salt or hot water may be bound over the ear, which has been first covered by cotton. Neither oil nor laudanum should be dropped into the ear as is so often done in domestic practice; but there is no objection to a few drops of a four-per-cent solution of cocaine, or a five-per-cent solution of carbolic acid, either of which may relieve intense pain. Frequent irrigation with a warm boric-acid solution is often useful. If the child is not soon comfortable, an opiate should be given which may not only relieve pain, but may have a favourable influence upon the inflammation.

A continuance of pain in spite of these measures, with an increasing temperature, calls for operative interference. If in addition there is mastoid tenderness immediate paracentesis of the drum membrane is imperative. An early incision is usually followed by a discharge of blood only; but tension is relieved, pain disappears, and the inflammation often quickly subsides without the formation of pus. (See Fig. 187.) Much suffering is thereby avoided; the wound rapidly heals, and much less damage is done than by allowing the disease to go on to a spontaneous rupture. Later operation may be required either for the relief of pain or for the evacuation of pus to prevent, if possible, the disease from spreading to the bony parts. The advantages of early paracentesis in acute otitis can hardly be overstated. Properly performed, it is free from risk, causes little or no shock, and should be advised in many cases even in which the indications are not so clear as those above described. I favor incising the drum membrane in cases of doubt rather than waiting for more definite indications with the attendant risks of delay.

In the secondary otitis of scarlet fever, measles, and diphtheria, the indications for paracentesis are usually to be derived from the appearance of the drum membrane alone, other symptoms being absent or masked by the primary disease.

After incision or spontaneous rupture of the drum membrane the ear should be syringed every two or three hours with a warm solution of bichloride (1 to 5,000), or a saturated solution of boric acid, or simply with boiled water. A bulb ear-syringe of soft rubber is the most satisfactory instrument for general use. A further rise in the temperature usually means that drainage is imperfect; if it is accompanied by pain, a second incision may be necessary. If the temperature remains high, one should be on the lookout for mastoid disease or other complications.

In most cases the discharge ceases in from one to three weeks; should it continue longer, some measures for checking it may be used. Dench advises as better than other applications, the use of a few drops of a 1-to-3,000 solution of bichloride in 65 per cent alcohol, after syringing. It should be applied with a medicine dropper. Where the discharge has become fetid, syringing once a day with a solution of peroxide of hydrogen (1 to 2) is often useful. A persistent discharge often depends upon the fact that the child's general condition is poor, and improvement in this is more important than any variation in local treatment.

Mastoiditis.—When symptoms pointing to acute mastoiditis are present, early free incision of the drum membrane is indicated, even though there may be no bulging, and a mastoid ice-bag should be applied continuously for thirty-six or forty-eight hours. In addition, in older children, the artificial leech may be placed over the antrum or mastoid tip. With these measures the inflammation often subsides. Regarding operation upon the mastoid, my own belief is that it is now performed too frequently and with insufficient indications, especially in infancy and early childhood. The operation is a serious one, and at this age its immediate risks are considerable. I have personally known of a number of deaths directly connected with it, and of others occurring at a later period, where the child was worn out by the long after-treatment, dying perhaps from some intercurrent disease or from exhaustion. On the other hand, the dangers to which patients are exposed who are not operated upon have, I think, been greatly exaggerated. In my own experience, meningitis, sinus thrombosis, and cerebral abscess do not occur in anything like the proportion of cases that the surgeons would have us believe.*

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* The records of the New York Foundling Hospital, with a resident population of about 800 infants and young children, showed 573 cases of acute otitis in five years (1900 to 1904 inclusive). During this period there were three extensive epidemics of measles with a total of 1,034 cases; 166 cases of scarlet fever; 578 cases of diphtheria; and 1,505 cases of pneumonia, many of which complicated influenza. With the 573 cases of otitis, acute mastoiditis was recognised and recorded in but 17 patients. It is not

While I fully appreciate the value of the operation, and am quite sure that lives are often saved by its timely performance, I would insist that it be done only with very positive and clear indications. In infants, localised tenderness is difficult to determine; and fever after acute otitis may be due to many other conditions. In very young patients we should therefore insist upon other symptoms before deciding to operate. The risks of waiting for clearer indications are, I believe, much less than those attendant on unnecessary operation. Often the cause of the temperature is found in the lungs; and not very infrequently a moderate pulmonary congestion or bronchitis becomes a pneumonia as a consequence of the prolonged anæsthesia necessary for the operation. With infants therefore in case of any doubt, as to diagnosis or the progress of the case, one should invariably decide against operation, or at least for postponement. With older children, however, conditions are somewhat different; diagnosis is easier and the operative risk much less.

The treatment of chronic otitis and of the associated conditions is largely surgical, and belongs to the specialist; but it is extremely important that the general practitioner should be familiar with their symptoms, and realize the danger from these neglected cases, not only to the function of hearing, but also to life itself. The essential thing in treatment is to operate sufficiently to secure free drainage, and to permit thorough cleansing of the parts. Too much can not be said against the expectant treatment of these cases, or against the practice of prolonged poulticing.

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improbable that other mastoid inflammations were overlooked. In this institution, however, nearly every fatal case comes to autopsy, and if an unrecognised mastoiditis had led to a fatal result the autopsy records should show. In the five-year period, 900 autopsies were made. There was no instance recorded of abscess of the brain following otitis. There were but two examples of acute meningitis following otitis with mastoiditis; but there were 14 cases of acute meningitis secondary to other conditions —pneumonia, 10; to pericarditis, 2; to empyema, 1; to diphtheria, 1. During the period mentioned there were 11 mastoid operations performed in the hospital, with 6 recoveries and 5 deaths, all from causes directly connected with the operation.

If mastoiditis follows otitis, complicating the acute infectious diseases of early childhood as often as has been claimed, we must admit that a very large proportion of the patients may get well without operation.

SECTION IX.

THE SPECIFIC INFECTIOUS DISEASES.

ACCURATE classification of the infectious diseases is at the present time impossible, but there are two quite distinct groups into which, with one or two exceptions, those here considered may be placed.

The first group includes scarlet fever, measles, rubella, varicella, and pertussis. The nature of the specific poison in each of these is as yet unknown. They are, strictly speaking, contagious; for it is practically certain that any of them may be contracted by proximity to a person suffering from the disease, without actual contact. In no one of these diseases is the poison given off in a single definite discharge, and in no one is there a characteristic visceral lesion. Mumps resembles the members of this group in all points except the one last mentioned. These peculiarities, together with the fact that thus far the poison of each of these diseases has resisted all attempts at isolation, render it not improbable that these poisons are some other variety of micro-organisms than bacteria.

In the second group may be placed diphtheria, typhoid fever, and tuberculosis, in each of which the specific poison is a known form of bacterium. Each of these diseases is associated with definite and characteristic visceral lesions. The poison is discharged from the body in a certain well-understood manner from the tissues which are affected by the disease, and in no other way. These diseases can not be contracted by proximity to a diseased person, but only by receiving into the body the specific germs, either by contact with a person suffering from the disease or contact with something upon which the special germs of the disease have been discharged. In other words, though communicable, they are not, strictly speaking, contagious.

Syphilis, influenza, and malaria have not been included in either of the above groups. Syphilis must still be placed in the doubtful class, although its general characteristics ally it with the second group. In its communicability, influenza resembles the first group, although there is now little doubt that it is due to a form of bacterium—Pfeiffer's bacillus. Malaria belongs in a class by itself, differing in nearly all its essential features from the other diseases of this general group, as its specific cause is a form of protozoon.

CHAPTER I.

SCARLET FEVER.

Synonym: Scarlatina.

SCARLET FEVER is an acute, contagious, self-limited disease, one attack usually protecting the individual through life. The period of incubation is usually from two to six days; that of invasion, from twelve to twenty-four hours; that of eruption, from four to six days; that of desquamation, from three to six weeks. The disease may be communicated at any time from the first symptom of invasion throughout desquamation, and sometimes even during the existence of purulent discharges from the nose or other mucous membranes. It is usually ushered in by vomiting, high fever, and sore throat, and is characterized by an erythematous rash appearing first upon the neck and spreading rapidly over the entire body. Its chief complications are otitis and membranous inflammations of the pharynx, which frequently extend to the nose, more rarely to the larynx. The most important sequelæ are otitis and nephritis.

Etiology.—Analogy leads to the belief that scarlet fever is due to a micro-organism, but as yet its nature has not been discovered. The complications are usually associated with the development of a streptococcus. Some have gone so far as to claim that this germ is the cause of the disease. From present knowledge, however, it appears rather to play the rôle of a secondary or accompanying infection, for the development of which the mucous membranes of a person suffering from scarlet fever seem to afford most favourable conditions. To the streptococcus may be ascribed the membranous inflammations of the tonsils and pharynx, the otitis, the inflammation of the lymph nodes and the cellular tissue of the neck, and probably also the nephritis, endocarditis, pneumonia, and joint lesions. In many of the above conditions the streptococcus is associated with other pyogenic germs, and in some cases with the diphtheria bacillus.

Predisposition.—The susceptibility of children to the scarlatinal poison is much less than to that of measles; still, it is much greater than that of adults. Billington (New York) records observations made in twenty-six families living in tenements where little or no attempt at isolation was made. In these families there occurred 43 cases of scarlet fever; but 47 other children, although unprotected by previous attacks and constantly exposed, did not contract the disease.

Johannessen reports that of 185 children under fifteen years who were exposed, 28 per cent contracted the disease; while of 314 adults, only 5 per cent contracted the disease. It may be stated that, approximately, not more than one half of the children exposed take the disease. The susceptibility is not great in early infancy, but it increases until about the fifth year, after which it steadily diminishes. Both sexes are equally liable to scarlet fever. Epidemics are more frequent in the fall and winter than in summer, and cases occurring in the cold months are apt to be more severe. Whitelegge, in 6,000 cases, found the highest mortality in the month of October; and in Caiger's report of 1,008 cases this was also the month showing the greatest mortality.

Incubation.—Of 113 cases * in which the period of incubation could be accurately determined, it was as follows:

24 hours or less	6 cases.	8 days	2 cases.
2 days	15 "	9 "	5 "
3 "	28 "	11 "	1 case.
4 "	25 "	14 "	1 "
5 "	6 "	21 "	1 "
6 "	15 "		
7 "	8 "		113 cases.

Thus in 87 per cent of these it was between two and six days, and in 66 per cent between two and four days. The incubation is rarely over a week; it is particularly short in surgical cases, a well-authenticated instance being on record in which it was but six hours. Speaking generally, if, after exposure, a week passes without symptoms, the chances of infection are very small. A short incubation is more frequently seen in severe than in mild cases.

Mode of infection.—The chief source of infection is the patient himself. It is somewhat doubtful whether the poison of scarlet fever can be conveyed by the breath, but it may be by discharges from the mucous membranes involved, from the scales during desquamation, and probably from all the excretions of the patient—urine, fæces, and perspiration. Infection often takes place from the carpets or furniture of the sick-room, and from the clothing of the patient. In a city the bed-clothing, while airing in the window, has been known to convey the disease to an adjoining house. Instances are recorded of the spread of scarlet fever by the washing of infected with other clothing. Toys or books may be carriers of the disease. A bouquet of flowers sent from a sick-room to an institution, in one instance proved a vehicle of infection. Cats, dogs, and other domestic animals are known to have conveyed the disease. Scarlet fever is sometimes spread by food, particularly by milk (page 141). It is possible, under these circumstances, that a disease resembling scarlatina existed in the cows; but that this was identical with scarlatina, as seen in man, was not demonstrated.

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* Part of these are from personal observation, but the great majority are isolated cases scattered through medical literature, occurring under circumstances which made it possible to determine the exact length of the incubation.

The transmission of the disease through a third person is not frequent, but numerous instances of it are on record. The persons most likely to carry it are the nurse and the physician. Physicians have in many cases carried scarlatina to their own children, but only when there had been pretty direct contact with the patient, and where the interval before seeing the second child was short. The clothing of the nurse may be almost as infectious as that of the patient. The transmission of the disease by one who, although living in the house, does not come in contact with the patient is extremely improbable. An instance is recorded in Allbutt (ii, 129) where scarlatina was transmitted through two healthy persons.

Duration of the infective period.—There is no evidence to show that the disease is communicable during the period of incubation. It is slightly contagious from the beginning of invasion, before the rash appears. Infection appears to be most active at the height of the febrile period—from the third to the fifth day—and, next to this, during the stage of active desquamation.

In simple cases, the average duration of the contagious period may be placed at six weeks, or until desquamation is complete. However, physicians generally have been accustomed to place too much stress upon the danger from the scales, and too little upon that from the discharges from the mucous membranes. Early infection comes chiefly from the throat, nose, or possibly the breath. Late infection may arise from a purulent otitis, rhinitis, chronic pharyngitis, suppurating glands, eczema, empyema, and possibly also from the urine in nephritis. The infectious nature of these purulent discharges has not been sufficiently recognised. It is possible for them to convey the disease during a period of several months. One case is recorded in which scarlatina was communicated through a purulent nasal discharge after eleven weeks; another in which the opening of a post-scarlatinal empyema in a surgical ward was followed by an outbreak of scarlet fever.

In winter especially, a chronic pharyngeal catarrh may long contain the germs of infection. Ashby found, on careful investigation, that from two to four per cent of patients discharged from a scarlet-fever hospital subsequently conveyed the disease. There is particular danger from a child who has recently had the disease sleeping with other children. Line records a case in which this was the means of conveying the disease after fourteen weeks, and when the patient had been considered perfectly well for three weeks. It is impossible to say that at any specified time absolute safety exists. All patients before being discharged from a hospital or released from quarantine in private practice, should be carefully examined as to the condition of the mucous membranes, and quarantine continued as long as catarrhal inflammations are present. The poison of scarlatina clings more tenaciously to clothing, upholstery, and apartments than that of any other contagious disease, possibly excepting tuberculosis. Authentic cases are on record in which more than a year had elapsed between the first and second cases, where the source of infection seemed certain.

Lesions.—The only characteristic lesions of scarlatina are those of the skin and the mucous membranes of the mouth and throat. The skin is the seat of an acute dermatitis of variable depth and intensity. There is first acute hyperæmia, followed by an exudation of serum and cells in the corium, especially about the blood-vessels and hair follicles. There results a death of the epidermis which is thrown off in the desquamation. The mucous membrane of the mouth, tongue, and throat is the seat of a catarrhal, membranous, or gangrenous inflammation which rarely invades the larynx, but very frequently the middle ear and nose. The entire œsophagus is often the seat of an intense congestion. From the ear the infection may extend to the mastoid cells, the meninges, or the brain, and from the nose to the accessory sinuses, particularly the antrum of Highmore. All the lymph nodes about the neck may be involved, the infection ending in cell-hyperplasia, suppuration, or necrosis. The cellular tissue of this neighbourhood may also become infiltrated, this being followed sometimes by suppuration and occasionally by gangrene.

The most constant change throughout the body, according to Pearce (Albany), is hyperplasia of the lymphoid tissue, which is seen everywhere. The other lesions are degenerations due to the scarlatinal poison alone, or in conjunction with the various forms of secondary infection, or to the latter alone. The most important are: fatty degeneration of the heart; areas of focal necrosis in the liver; acute degeneration of the kidney or acute diffuse nephritis; proliferation of the cells of the Malpighian bodies of the spleen; broncho-pneumonia, gangrene, or abscess of the lung; pleurisy, which is often purulent; endocarditis, pericarditis; abscesses in the cellular tissue and inflammation of the joints. These visceral changes will be considered more fully under Complications.

Symptoms.—*Invasion.*—As a rule, the invasion of scarlet fever is abrupt, the symptoms at the onset usually being directly in proportion to the severity of the attack. In the majority of cases there is vomiting, a rapid rise in temperature, and soreness of the throat. Often the vomiting is repeated; it is frequently forcible, and without nausea. In severe cases the rise in temperature is very rapid, to 104° or 105° F.; in the mildest cases it may not be above 101°. A child may complain of soreness of throat, or the throat symptoms may be entirely objective. In most severe cases, there is a uniform erythematous blush covering the pharynx, tonsils, and fauces, but on the hard palate there are minute red points. The appearance of this is usually coincident with the rise in temperature. Occasionally membranous patches may be seen upon the tonsils the first day, but not generally before the third or fourth day. In mild cases the throat shows only a very moderate congestion. Severe cases are sometimes ushered in by convulsions, especially in very young children. Diarrhœa is not uncommon in summer. There is general prostration, which is directly proportionate to the height of the fever.

Eruption.—This usually appears from twelve to thirty-six hours after the first symptoms of invasion; exceptionally, not until the third or even the fifth day. A later appearance than this is somewhat doubtful, for the rash not infrequently recedes and reappears, having been overlooked in the first instance. In 108 cases observed in the New York Infant Asylum, the duration of the rash was as follows:

Two days or less	5 cases.
Three to seven days	81 "
Eight to eleven days	16 "
Over eleven days	4 "
Recurring	2 "

These statistics are confirmed by the observations of most writers, that the rash lasts from three to seven days. The full development of the rash is generally seen in from twelve to twenty-four hours from its first appearance, and not infrequently the whole body is covered in the course of four or five hours. Very rarely its extension is so slow that it is two or three days before the body is covered. Its first appearance is almost invariably upon the neck and chest. In the cases of moderate severity the typical rash is seen. Its colour is red rather than scarlet, and on close inspection it is seen to be made up of very minute points upon a reddish ground, giving the appearance of a uniform blush. The rash covers the entire body, including the face. There is often a peculiar pallor about the mouth, which is quite characteristic of the disease.

Variations in the eruption are very frequent, and often extremely puzzling. In the mild cases the rash is not seen upon the face; it is often faint upon the body, and may be present only upon certain parts; when the rash is faint or scanty it is usually most marked in the groins and axillæ, or over the buttocks and back of the thighs; it may last only one day, and sometimes may be so slight as to escape notice altogether. It may be absent in some very mild cases, in certain others where the throat symptoms are severe, and in malignant cases. In the very severe cases many irregularities are seen, both as to the time of the appearance of the eruption and its character. Sometimes it occurs as large, irregular patches; again, it is macular, closely resembling the rash of measles; occasionally it is of a dark purplish colour; and very rarely it is hæmorrhagic. Not infrequently an eruption of fine vesicles is seen, especially on the chest and abdomen; this may be so pronounced as to make the diagnosis difficult. It is seen both in mild and severe cases. Much importance is attached by the laity to the early disappearance of the rash, an especial danger being believed to exist because the disease has "struck in." A well-developed bright rash indicates strong heart action, and a sudden recession of the rash is a sign of heart failure. Often a rash which is faint and doubtful in character, may be brought out fully by a hot bath.

With the eruption at its height, there is intense itching or burning of the skin, and in severe cases considerable swelling, chiefly noticeable upon the hands and face.

Desquamation.—Shortly after the rash has faded, about the eighth day, there begins an exfoliation of the dead epidermis, known as desquamation. This is even more characteristic of the disease than the rash. It is usually first seen upon the neck and chest, where it appears as fine flakes. The desquamation of the trunk is completed in from one to three weeks. If baths and inunctions are being used, it is scarcely perceptible. It continues longest where the epidermis is thickest—viz., upon the hands and feet—and here it lasts from four to seven weeks, and not infrequently eight weeks. The appearance of the fingers and toes during desquamation is characteristic. The finger tips usually peel first, and the new epidermis is pink and fresh-looking, while that which has not yet separated is of dull gray colour and loosened at the margin. Occasionally the epidermis of a considerable part of a finger may be loosened at once, so that a partial cast may be thrown off like the finger of a glove. Sometimes the patient comes under observation for the first time during desquamation, the history of the early symptoms being doubtful or absent. Such desquamation as has been described, occurring both upon the hands and feet, may be regarded as conclusive evidence of scarlet fever, no matter what the history may be.

1. *The mild cases.*—The symptoms may be so slight as to be entirely overlooked, nothing being noticed until desquamation occurs. Usually, however, there is a rather abrupt invasion, with vomiting and a temperature from 100° to 103° F. The tonsils and pharynx are congested, while the palate shows a punctate redness somewhat like the cutaneous eruption. The papillæ of the tip and borders of the tongue are enlarged. Nearly always within twenty-four hours the rash makes its appearance, generally first upon the neck and chest. Very often it is not seen upon the face, but is abundant on the rest of the body. The rash fades on the third or fourth day, and has disappeared by the fifth day. There is very little prostration, the child often being with difficulty kept in bed.

The highest temperature is coincident with the full eruption, and is usually seen during the first thirty-six hours of the disease. It gradually falls to normal by the third or fourth day. Some examples are shown in Fig. 189. In the mildest cases the temperature may never be above 100° F.

Desquamation is often faint over the body, but is unmistakable over the hands and feet. It begins about the end of the first week, always being most marked where the eruption has been most intense.

The mild cases are usually uncomplicated, but the possibility of otitis and of late nephritis should always be kept in mind, as these may occur even with the mildest attacks. The difficulties in diagnosis in mild attacks of scarlet fever are often great. It should be remembered that these cases are just as contagious as severe ones, and that from a mild attack a severe one is often contracted. It is frequently by these mild cases that this disease is spread in schools. In dispensaries I have often seen patients desquamating from scarlet fever, who had been attending school regularly up to the time when they were brought for treatment for nephritis or some other disease.

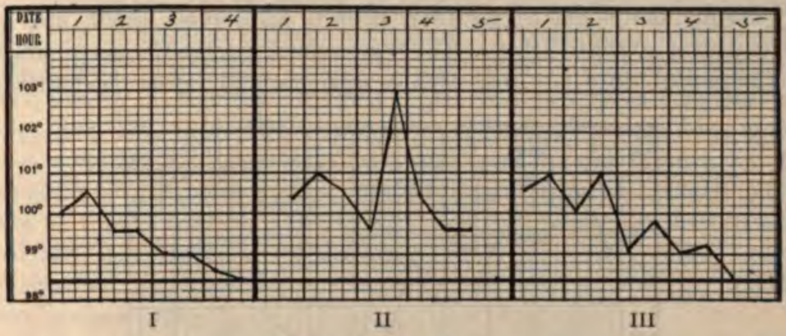

Fig. 189.—Mild scarlet fever.

Three cases occurring successively in the same family. Diagnosis not made until the third case developed, at which time the first one was found to be desquamating in a typical manner.

2. *Cases of moderate severity.*—The onset is sudden with vomiting, which is usually repeated, rarely with convulsions. The temperature rises rapidly, and by the end of the first twenty-four hours has reached 104° or 105° F. The rash usually appears within the first twenty-four hours, and its intensity is directly proportionate to the severity of the attack. Appearing first upon the neck or chest, it extends rapidly, covering the entire trunk and extremities, often in a few hours. It is usually typical in appearance, being made up of minute points, but giving the appearance of a uniform blush, which has been compared to a boiled lobster. Little change takes place in the rash for four or five days. After this it fades quite rapidly, and disappears by the sixth or seventh day.

The throat resembles that of the mild form, except that the redness is more intense and there is slight swelling of the tonsils, fauces, and uvula, and often pain upon swallowing. Occasionally small yellowish patches are seen upon the tonsils by the second or third day, but these can be wiped off and are not distinctly membranous. There is usually a moderate discharge of a sero-purulent character from the nose. The lymph glands at the angle of the jaw are swollen and quite tender. The tongue may be coated in the centre and show bright red points at its borders and tip, or it may be quite red and show the prominent papillæ everywhere—the "strawberry tongue"; while not exclusively seen in scarlatina this is of some diagnostic value and may continue several days or even weeks.

During the height of the fever there is restlessness, thirst, and not infrequently slight delirium. The temperature reaches the maximum by the second or third day, and usually falls gradually after the fourth or fifth day, but even in uncomplicated cases the fever often lasts from ten to fourteen days (Fig. 190). The pulse in the early part of the disease is rapid and full, but later it may be weak. There is much prostration, frequently followed by quite a marked degree of anæmia.

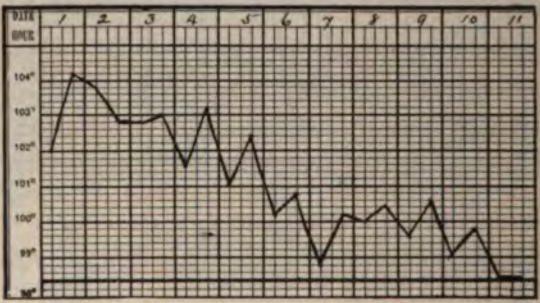

Fig. 190.—Typical temperature curve of uncomplicated scarlet fever of moderate severity; girl three years old.

This form of the disease rarely proves fatal apart from complications, but it may do so in very young infants. The complications seen most frequently in this form of scarlet fever are broncho-pneumonia or pleuro-pneumonia and otitis, the latter being usually double and occurring between the sixth and the fourteenth days. Nephritis is the only common sequel.

3. *The severe cases.*—The severe type of scarlet fever usually declares itself from the beginning. The incubation is short, and the full rash may be seen within a few hours after the initial symptoms. It usually covers the entire body, even including the face. The severity of the infection is shown by the fact that the temperature is higher and continues for a longer period, and by the frequency and severity of the complications, particularly those of the throat. For the first two days the throat may present nothing different from what is seen in the milder cases. By the third or fourth day, however, membranous patches often appear on the tonsils, and spread to the soft palate, uvula, and pharynx, sometimes to the nose and through

the Eustachian tube to the ear, rarely involving the larynx. The mucous membrane of the mouth is intensely congested, and often partly covered by membrane; there are sordes on the lips and teeth, and there may be superficial ulcers, which bleed readily. The glands of the neck swell rapidly, often to a great size, and the cellular tissue about them is infiltrated. The head is thrown back to relieve the dyspnœa which the pressure from this swelling occasions. There is an abundant discharge from the nose and mouth; the breath is offensive, often fetid. The general symptoms are those of a severe septicæmia. The temperature is steadily high, usually between 103° and 105° F., for about a week, after which in cases ending in recovery it slowly falls unless complications develop (Figs. 191, 193, 194). But even in uncomplicated cases the fever sometimes continues for three weeks. In fatal cases the temperature may be steadily high till death (Fig. 192), or it may fluctuate widely. The pulse is rapid, weak and irregular. There is complete anorexia; both food and stimulants have to be coaxed or forced down. There is low delirium or apathy, and sometimes all the symptoms of the typhoid condition are present.

Signs of a broncho-pneumonia are often found in the chest, and by the end of the first week or early in the second the ears may begin to discharge. The urine is rarely free from albumin, but the amount present is not usually great; there may be hyaline and epithelial casts, and sometimes blood. In some cases the throat symptoms predominate; in others, those of general sepsis, but more frequently the two are combined and are directly proportionate to each other. In still other cases, instead of the membranous inflammation, it may be of a gangrenous character, and extensive sloughing may take place in the throat, and even in the cellular tissue of the neck.

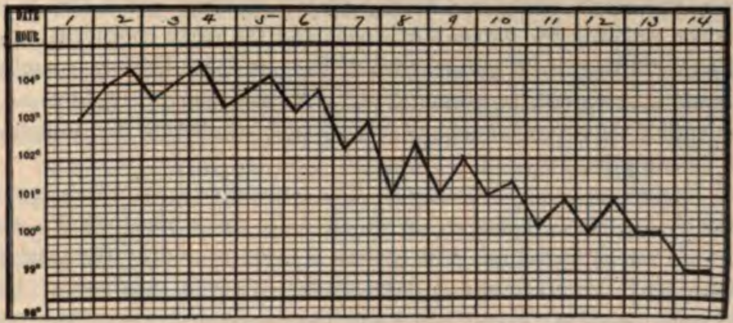

Fig. 191.—Typical temperature curve of severe scarlet fever ending in recovery.

Prolonged course due to severe throat symptoms lasting from second to sixth day, otherwise uncomplicated; boy twelve years old.

The duration of the symptoms in fatal cases is from six to fourteen days. There is general increasing prostration and finally a septic stupor, with death from exhaustion, from heart failure, or from some complication—broncho-pneumonia, pleurisy, nephritis, hæmorrhages following sloughing, laryngitis, pericarditis, or endocarditis. In cases which recover, the acute symptoms nearly always continue for a full month; and after escaping the dangers of sepsis and the early complications, the child has still to run the gauntlet of all the late complications—nephritis, pneumonia, endocarditis, pyæmia, etc. A case may prove fatal as late as the end of the seventh week; nearly all such results are due to nephritis or to its complications.

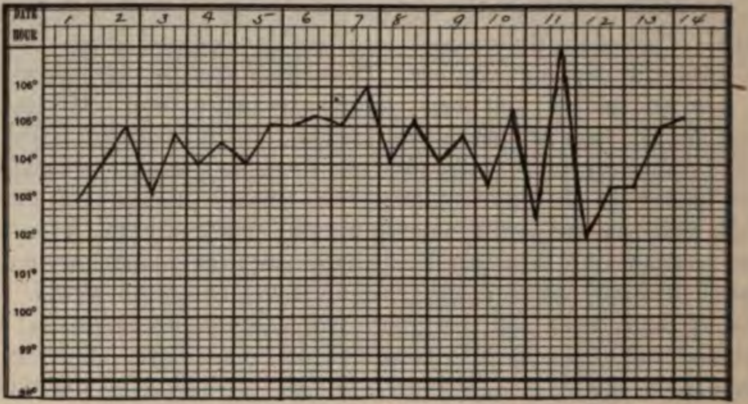

FIG. 192.—Severe scarlet fever, septic type; death on fourteenth day.

Intense angina; otitis; nephritis; necrotic inflammation of cervical lymph glands; girl seven years old; death from heart failure.

4. *Malignant or cerebral cases.*—These are rare cases which are more frequently described than seen, in which death takes place usually within the first forty-eight hours. The system is overpowered by the scarlatinal poison. Such cases are seldom seen except in severe epidemics. Under other circumstances, many cases are diagnosticated malignant scarlet fever which have no connection with this disease.

The onset is sudden and violent, usually with convulsions, the child passing in a few hours into a condition of deep stupor, with great prostration and hyperpyrexia, the temperature ranging from 105° to 107° F. Sometimes, however, the temperature does not go above 100° F. The rash appears irregularly, late, or not at all. It may be hæmorrhagic. There are frequently repeated convulsions, cyanosis, and invariably a fatal termination. The autopsy often gives no satisfactory explanation of these cases. Death occurs apparently from scarlatinal toxæmia, without any characteristic local evidences of disease.

5. *Surgical scarlet fever.*—The existence of a special form of scarlet fever occurring in patients with recent wounds or those who have been subjected to surgical operations, while stoutly maintained by several

writers, has been vigorously denied by others. The question is one difficult of solution on account of the close similarity at times existing between the symptoms of scarlet fever and sepsis, and the necessity of deciding in an undoubted case whether the infection with scarlet fever was dependent upon or coincident with the wound.

Hamilton * has recently studied the question anew and analysed cases, some 174 in number, that have been reported more or less in detail, with the following conclusions: That proof of the existence of a special form of scarlet fever rests upon the reports of cases usually meagre, and careful analysis of these would lead one to consider them rather as septic than as scarlatinal infections; that when there was undoubted evidence of scarlet fever, there was no proof that it was in any way due to the coincident wound, and that there is as yet no convincing proof in the literature that surgical scarlet fever is anything more than scarlet fever in the wounded.

Relapses, Recurrences, and Second Attacks.—As a rule, one attack of scarlatina gives immunity through life. The exceptions are very few, but some of them are well authenticated. Kinnicutt (New York) observed two attacks within eight months in a boy of five years; Pritchard (Glasgow) reports the case of a patient who had three attacks in the same hospital within two years; such cases are certainly extremely rare. I have never yet seen an undoubted instance of a second attack in the same individual.

Relapses or recurrences within a brief period after the first attack are more frequent. There are to be excluded the cases of pseudo-relapses in which the rash, having temporarily subsided for two or three days, reappears; also those where the rash varies in intensity from time to time; and, lastly, the cases in which, occurring late in the disease, it is due to septicæmia or pyæmia. True relapses are usually due to auto-infection, sometimes to a new accession of poison from without. They are analogous to the relapses of typhoid fever. They occur most frequently during desquamation, between the seventh and twenty-fourth days. There may be not only a new eruption but a rise of temperature, sore throat, and vomiting, just as in the initial attack. These recurrences are sometimes shorter and milder than the first attack, but this is by no means uniform, since Körner mentions eight cases where the second attack proved fatal.

In considering the subject of second attacks, the liability to errors in diagnosis must be borne in mind and only cases included which have presented typical symptoms.

Special Symptoms, Complications, and Sequelæ.—*Temperature.*—The temperature curve of this disease is quite characteristic. There is usually seen an abrupt rise, the maximum being reached on the second day; there follows a period of variable duration, generally lasting, according to the severity of the case, from two to five days, in which the fluctuations are very narrow; then a gradual decline to normal, which is reached in the milder cases in about a week; in those which are more severe, in about two weeks. This typical curve (Figs. 190 and 191) is seen in the great proportion of uncomplicated cases which end in recovery. Deviations from it, therefore, are important as indicating that some complication exists. The explanation is usually to be found in the development of otitis, nephritis, pneumonia, etc. Severe throat symptoms prolong the temperature but do not usually modify its course. In very severe cases ending fatally the high temperature is prolonged. In any case a rise after the fifth day is unfavourable.

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* American Journal of the Medical Sciences, July, 1904.

Throat.—Three distinct forms of angina are seen in scarlatina: simple or erythematous, membranous, and gangrenous.

1. Erythematous angina.—This can hardly be ranked as a complication, as it is nearly as constant as the scarlatinal rash. Usually there is only the general blush over the entire pharynx with the fine red points upon the hard palate; but there may be seen upon the tonsils grayish-yellow spots resembling those of follicular tonsillitis, which can be wiped off, leaving a clean surface. This simple angina is at its height with the maximum temperature, and fades as the temperature falls. It does not often extend to adjacent mucous membranes.

2. Membranous angina.—These cases were formerly classed as scarlatinal diphtheria, and whether this process was identical with primary diphtheria or not, was for a long time a subject of much discussion. Cultures have shown that the great majority of these inflammations are not true diphtheria, but are due to the streptococcus.

The lesions of this form of angina are considered in the chapter on Pseudo-Diphtheria. Usually on the second or third day of the disease an exudation appears upon the tonsils, and in the milder cases it covers only the tonsils. In the most severe form it may be seen within twenty-four hours of the onset, sometimes before the eruption appears. Beginning upon the tonsils, the membrane rapidly spreads to the entire pharynx, the mucous membrane of the nose, the mouth, the Eustachian tube, and even to the middle ear. In colour it may be gray, greenish, or almost black. There is so much swelling of the throat that swallowing becomes difficult. The infiltration of the cellular tissue of the neck and the enlarged lymph glands produce great external swelling, which may extend like a collar from ear to ear. The breath has a foul odour, the nasal discharge is thin and fetid, and nasal respiration is obstructed, so that the mouth is open constantly. It is surprising that the larynx is so seldom invaded.

These local changes are accompanied by constitutional symptoms of great severity, which are due to a general streptococcus septicæmia;

broncho-pneumonia and nephritis are very frequent, otitis is almost constant, and suppuration of the lymphatic glands is not uncommon. The eruption is often irregular and late in appearing.

The frequency with which diphtheria coexists with scarlatina depends much upon circumstances. In some epidemics, thirty per cent of the throats showing membrane have contained the diphtheria bacillus; in others the proportion is much smaller. There are some clinical features by which the two types may sometimes be distinguished. The streptococcus angina is usually seen at the height of the disease; true diphtheria may occur at any time, even during convalescence. The streptococcus angina is characterized by much swelling, redness, and œdema of tonsils and fauces, and by great external infiltration, showing a marked tendency to invade the ears, but very little to invade the larynx. In true diphtheria the evidences of inflammation are usually much less, while there is a great tendency to invasion of the larynx. Very little reliance is to be placed upon the appearance of the membrane. The only positive means of differentiation is by cultures, which should invariably be made from the throat of every patient admitted to a scarlet-fever hospital, and of every case in private practice showing any exudate upon the tonsils. If the first culture is negative and the throat symptoms increase, repeated cultures should be made.

3. Gangrenous angina.—This is seen only in the worst cases of scarlet fever. The process may be gangrenous from the outset, or preceded by a membranous inflammation. It is sometimes insidious in its development. There is a fetid odour to the breath, an irritating discharge from the nose and mouth, with very great glandular swelling. The tonsils are gray or grayish-black in colour, and large masses of necrotic tissue may be removed with the forceps from the tonsils, uvula, fauces, or pharynx, and sometimes sloughing occurs in the cellular tissue of the neck. Blood-vessels of considerable size are sometimes opened, and serious or even fatal hæmorrhage may result. Little or no tendency to a reparative process is seen. The constitutional symptoms are those of great asthenia, prostration, and profound cachexia, followed almost invariably by a fatal termination.

Lymph nodes.—These are swollen in all cases accompanied by severe angina. The inflammation may be simply an acute hyperplasia, or it may go on to suppuration or necrosis. Abscess does not often occur at the height of the disease, but may come at any time during convalescence. It may be confined to the glands or be complicated by suppuration in the cellular tissue of the neck. Disease of these glands is not an infrequent cause of torticollis.

Cellulitis of the neck.—This usually occurs toward the end of the first week, and is associated with grave throat symptoms. Rapid and extensive infiltration occurs, the skin becomes tense and brawny, the head is

held back, and there may be considerable dyspnœa. The i
be only in the neighbourhood of the lymph glands or it r
Unless relieved by early incision, the diffuse form may res
tion and extensive sloughing, which may be deep enough
large vessels of the neck. This is a complication of th
sible import. Death may occur from septicæmia before o
ing or from hæmorrhage due to opening by ulceration o
carotid or some of its branches; or there may be associa
of the jugular vein, leading to thrombosis of the lateral
gitis, or pyæmia.

Ears.—The otitis is due to direct extension of the i
the rhino-pharynx. It is the most frequent complication

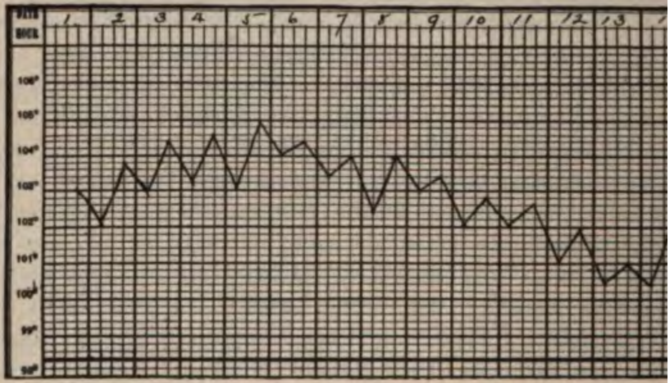

FIG. 193.—Severe scarlet fever; otitis; mastoiditis; deat

Typical symptoms and temperature curve until fourteenth day; sec
perature from otitis; double paracentesis on the fifteenth day; mastoid o
teenth day; death twelve hours later from septicæmia; boy five years old.

and in doubtful cases may have some diagnostic importan
the younger the child the greater the liability to otitis.
quent in winter than at other seasons, and is closely con
severity of the throat symptoms. In an epidemic occurri
York Infant Asylum in the spring and summer of 1889
cases of scarlatina and not one of otitis. In a fall a
demic in the same institution two years later, of 43 cas
had otitis. Of 4,397 cases reported by Finlayson, otit
10 per cent, and of 1,008 cases reported by Caiger, in 13
Burkhardt's statistics the proportion was as high as 33
cases accompanied by severe throat symptoms otitis is p
75 per cent.

As a rule, both ears are affected. Otitis is most frequ
second week, but may occur at any time, even during con
the cases where it develops at the height of the disease th

cases no new symptoms; in others there is pain and deafness and a rise in the temperature, which may fall after paracentesis or rupture of the drum membrane, or there may be rapid extension to the mastoid (Fig. 193). The otitis is often overlooked unless the ears are regularly examined. The form of inflammation may be catarrhal or phlegmonous, the latter being often accompanied by necrotic changes.

Bezold makes the following report upon 185 cases showing the results of scarlatinal otitis: "In 30 there was entire destruction of the membrana tympani, with loss of one or more bones; in 59 the perforation comprised two thirds or more of the membrane; in 13 there were smaller perforations; in 44 there were granulations or polypi; in 15 there was total loss of hearing on one side, and in 6 of the cases upon both sides; in 77 of the cases the hearing distance for low voice was less than twenty inches."

As a cause of permanent deafness and deaf-mutism, no disease of childhood compares in importance with scarlet fever. May (New York) has collected statistics of 5,613 deaf-mutes, of whom 572 owed their condition to otitis following scarlet fever.

Kidneys.—Albuminuria accompanies nearly all the severe cases of scarlet fever. In many this is simply the ordinary febrile albuminuria due to acute degeneration of the kidneys. In those with severe throat complications, and in nearly all the septic cases, there is often an acute diffuse nephritis with interstitial changes especially marked. This occurs at the height of the febrile process and is rarely accompanied by dropsy; but albumin, casts, and even blood may be found in the urine. The most severe and the most characteristic renal complication, and that generally designated as *post-scarlatinal nephritis,* is a diffuse nephritis, with changes in the glomeruli as the most striking feature. It usually develops during the third or fourth week of the disease, and may follow mild as well as severe cases (Fig. 194). It is very often

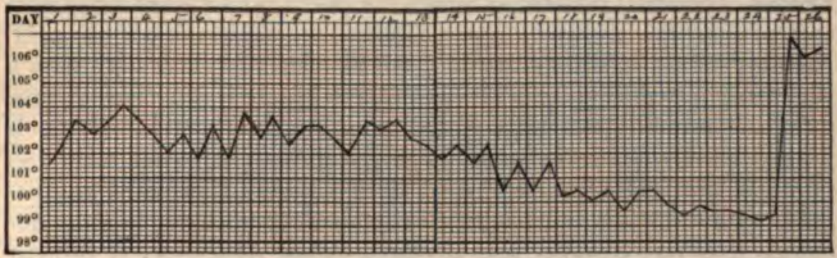

FIG. 194.—Scarlet fever of moderate severity followed by fatal nephritis.

Early symptoms typical and uncomplicated; twenty-first day vomiting; twenty-fifth day uræmic convulsions; death twenty-sixth day. No dropsy; urine never below 10 ounces in twenty-four hours; girl ten years old.

accompanied by general dropsy; the urine is scanty and not infrequently suppressed, and it contains a large amount of albumin, blood, and great numbers of casts of all varieties. It may cause death by the occurrence of acute uræmia, or it may be followed by permanent damage to the kidneys. It is more fully described with the Diseases of the Kidney.

Joints.—Acute articular rheumatism may occur coincidently with the development of the scarlatinal rash, and occasionally during convalescence in patients who have a predisposition to that disease. Acute swelling of the joints is sometimes of pyæmic origin. A case is reported by Henoch in which this was due to an infectious thrombus in the jugular vein, associated with cellulitis of the neck. In pyæmic arthritis the large joints are usually involved and the lesions are apt to be multiple. Joint disease may occur as a sequel of scarlet fever, where it is secondary to disease of the bone or to periarticular abscesses opening into the joint.

The foregoing include but a small proportion of the joint complications seen in scarlet fever. The most frequent and most characteristic form of inflammation is *scarlatinal synovitis,* often improperly called *scarlatinal rheumatism.* It occurs in different epidemics with varying frequency. Carslaw (Glasgow) in 533 cases of scarlet fever met with synovitis in 60 patients. It is seldom seen in children under three years of age, and is most frequent after five years. It may occur in mild as well as in severe cases. According to Ashby, it is more frequent when the febrile stage is prolonged owing to other complications. Synovitis develops quite uniformly toward the end of the first or the beginning of the second week. The symptoms are generally mild, and are followed by prompt recovery. Suppuration is rare. Any of the joints may be attacked, but those of the wrist, hand, elbow, or knee are most frequently affected. Demme (Berne) has reported a case in which every large joint in the body was involved. The symptoms are redness, moderate pain, swelling, which is usually due to synovial distention, and sometimes a slight rise in temperature. The duration is generally but three or four days, and in most cases there is spontaneous recovery. This disease is distinguished from rheumatism by several points: it is not more frequent in rheumatic patients; cardiac complications are rare as compared with those seen in patients with genuine rheumatism; in some epidemics it is very common, and in others seldom seen; there is little or no tendency to relapses; anti-rheumatic remedies are without striking benefit; it does not skip about from joint to joint, and usually fewer joints are involved.

Lungs.—The pulmonary complications of scarlet fever are neither so frequent nor so important as those of measles. Broncho-pneumonia is usually found at autopsy in septic cases where death has occurred later than the third or fourth day, but it is not generally recognisable so early by physical signs.

In septic cases pleuro-pneumonia sometimes occurs early in the disease and at other times late, generally associated with nephritis, but occasionally without it. It is always a serious condition, and not infrequently a direct cause of death. Empyema may follow pleuro-pneumonia or occur with pyæmia or nephritis, but with the latter, simple serous pleurisy is more common. Œdema of the lungs occurs chiefly with nephritis, in which it is the most common cause of death.

Heart.—Cardiac murmurs are frequent at the height of the disease, but both endocarditis and pericarditis are rare. They are oftenest seen in septic cases, and with post-scarlatinal nephritis. Endocarditis may be simple or malignant, and may lead to embolism during convalescence. Some degenerative changes in the cardiac muscle are probably present in all the severe cases. Acute dilatation may result, which is sometimes a cause of death.

Blood.—In all cases there is a rapidly progressing anæmia that lasts into convalescence. The reduction in the red cells in an average case is about one million. The chief interest, however, attaches to the number and character of the white cells. In mild cases there may be only a moderate increase in their number, usually to from 10,000 to 14,000. It is in cases of moderate severity that the characteristic changes are found. In these there is a decided leucocytosis which appears early, attains its maximum about the fourth day, and gradually declines until the normal is reached, which may be not until the third, fourth, or fifth week. The maximum is usually about 30,000 to 35,000; although it may be as high as 75,000. During the first week the polynuclear neutrophiles form from 90 to 95 per cent of these cells; the eosinophiles as well as the lymphocytes are diminished. After the fifth or sixth day, there is a rapid increase in the eosinophiles which attain their maximum, sometimes 20 per cent of the total leucocytes, between the fourteenth and twenty-first days. After the third week they gradually diminish. Exceptionally there is found in convalescence a relative lymphocytosis, which may be as high as 50 per cent. Complications, nephritis excepted, usually show actual as well as relative increase in the polynuclear neutrophiles. In malignant and rapidly fatal cases there is usually a very small proportion of eosinophiles, and little if any leucocytosis, though exceptionally it may be high.

Digestive system.—Functional disturbances are very frequent, and, in fact, are seen in most of the cases, but organic changes are rare. Vomiting is the mode of onset in the majority of cases, but rarely continues throughout the attack. Late in the disease it is a frequent symptom of uræmia. Diarrhœa may be associated with it under both conditions. The tongue is nearly always coated, and clears off in quite a characteristic way, which, with the prominent papillæ, gives rise to the "strawberry" appearance. Catarrhal stomatitis is a very frequent complication, and in many cases of severe membranous angina the same p the buccal cavity.

Nervous system.—Nervous complications and sequel frequently with scarlatina than with most of the infect such severity. Convulsions are frequent at the outset, a dicate a severe attack, though not invariably so. Occur disease, they are usually due to uræmia, and may be a Meningitis may occur as a complication of otitis, in py sometimes with post-scarlatinal nephritis. Paralysis neuritis is rarely seen. Hemiplegia sometimes occurs hæmorrhage, or from embolism secondary to endocarditi with nephritis. Chorea was noted as a sequel in only th reported by Carslaw. In a report of 187 cases of epilep states that it followed scarlet fever in 12 cases. Insanit sionally observed, the usual form being acute mania, wi covery in a few weeks or months.

Gangrene.—Cases of symmetrical gangrene after sc been reported by Wilson and others. The parts genera the buttocks, thighs, and arms, but it may occur almost pathology of these cases is obscure. The process usuall eral places simultaneously, or in rapid succession, and a till death occurs.

Other infectious diseases.—Diphtheria is most frequ may be present even when there is no distinct membran

Scarlatina may also be complicated by measles, va sipelas, and occasionally by variola or typhoid fever. Th often an irregular commingling of those belonging to t They may begin simultaneously, or more frequently one other is subsiding.

Diagnosis.—The characteristic symptoms of scarlet abrupt onset, usually with vomiting, the marked elevat ture, the erythematous condition of the throat, the punct the hard palate, and the enlarged papillæ at the edges tongue, with the appearance of the rash within twenty-f difficulties of diagnosis usually depend upon irregulariti tion. The variations are seen in the mildest and in the m In the former the rash may be of short duration, often and in consequence easily overlooked; or it may be pr certain parts of the body instead of being diffuse. In case the groins, axillæ, and loins should be closely scrutin tate eruption. In very severe attacks also the rash may b may appear late or recede after being fully out, or be hæ irregular blotches instead of a uniform blush. In all stress should not be placed upon the rash alone.

Until we have some exact means of laboratory diagnosis as in typhoid fever, malaria, and diphtheria, an absolute diagnosis will in certain cases be impossible. Sometimes the diagnosis remains doubtful until the end, although occasionally confirmatory evidence may be obtained even in convalescence. Thus, a patient may desquamate in a manner so typical as to leave no doubt as to the nature of the preceding illness; again, the occurrence of a characteristic sequel, such as nephritis in the third or fourth week, may testify strongly for scarlatina as the primary disease; and, finally, the outbreak of undoubted cases among children who have been in contact with the patient is practically conclusive, always provided other sources of infection can be excluded. Desquamation, however, follows so many other eruptions that one should not rely upon it when slight or irregular as an evidence of scarlet fever, but only upon a typical exfoliation upon the hands and feet. It is a point of some practical importance not to oil the skin of a patient when awaiting desquamation for diagnosis, as this alters very much the characteristic appearances. In some puzzling epidemics the length of the incubation may be of material assistance in the diagnosis; where this is regularly more than a week, one may be pretty sure that he is not dealing with scarlet fever.

Scarlet fever with severe throat symptoms and doubtful eruption can be distinguished from diphtheria only by cultures, which should be made early and repeated if the first result is uncertain. Measles is distinguished from scarlet fever by the length of the invasion, the catarrhal symptoms, and the slowly spreading eruption, but most of all by Koplik's spots. Much more difficult is it to distinguish between mild scarlatina and rubella. In rubella the important thing is that, although the rash may be well marked, often covering the body, the constitutional symptoms are few or entirely absent. In scarlet fever with an eruption of the same intensity there is almost invariably a considerable elevation of temperature, usually 102° or 103° F., and a bright red throat.

There are so many skin eruptions which may resemble that of scarlet fever, that it is always hazardous to make the diagnosis of this disease from the eruption alone. This is especially true of sporadic cases occurring in infants; there is seen at this age a great variety of eruptions, usually associated with digestive disturbances, which closely simulate a scarlatinal rash; but most of them are of short duration. A scarlatiniform erythema is occasionally seen after diphtheria antitoxin, also in influenza, typhoid fever, and varicella, which may cause them to be mistaken for scarlet fever, or may lead to the conclusion that both diseases are present. The same is the case with the septic erythema occurring in surgical patients. Belladonna, quinine, and occasionally antipyrine, may produce eruptions more or less closely resembling that of scarlet fever.

This is also true of some cases of urticaria, and of several skin disease. There is little doubt that many of the cases lapsing scarlatina are really examples of recurring erythe as some of the latter are followed by a desquamation which to that after scarlatina. In all doubtful conditions great to be attached to the constitutional symptoms.

Prognosis.—The mortality of scarlet fever varies mu epidemics. In some, nearly all the cases are of a mild mortality may be as low as 3 or 4 per cent; in others, a s nant type prevails, and it may be as high as 40 per cent is, as a rule, more fatal in the youngest infants, becomin advances. This is well shown in two recent epidemics in Infant Asylum. There were—

Under one year	29	cases;	mortality,
From one to two years	37	"	"
" two " three "	28	"	"
Over three years	23	"	"

In the first epidemic the general mortality was 12·5 p second it was 33 per cent, in the same class of children.

The following are the mortality records from vari sources:

Ashby, Manchester Hospital	681	cases;	mortality
Koren, a single epidemic	426	"	"
Bendz, Copenhagen	22,036	"	"
Ollivier, three Paris hospitals for five years	893	"	"
Fleischmann, five epidemics	1,356	"	"

The general mortality of the disease may therefore be from 12 to 14 per cent; it is, however, much higher th young children, as shown by the following figures:

New York Infant Asylum	116 cases	under	5	years;	mortality
Ashby, Manchester Hospital	259 "	"	5	"	"
Bendz	not stated	"	5	"	"
Heubner	136 cases	"	7	"	"
Fleischmann	not stated	"	4	"	"

Under five years of age the average mortality from s therefore, between 20 and 30 per cent.

The fatal cases may be grouped in three classes: firs late nephritis, in which the early symptoms of the diseas erate severity or even mild; secondly, the septic cases, usu with severe throat symptoms and dying most frequently week from exhaustion, or from some complication, such pneumonia, pleurisy, meningitis, or nephritis; thirdly, cases, which are overpowered by the poison of the disea two or three days of the attack.

Prophylaxis.—Even the mildest cases should be isolated for four weeks, and all cases until desquamation is complete. If complications exist, such as otitis, rhinitis, pharyngitis, empyema, or suppurating glands, the quarantine should be continued until these conditions are cured. Patients should not be allowed to mingle with other children for at least a month after all symptoms have subsided, and should be forbidden to sleep with other children for three months. Children in the house who have not been exposed to the disease should be immediately sent away; and those who have been exposed, separately quarantined for at least a week. After recovery, the patient, before mingling with other children, should have at least two disinfectant baths, the entire body being scrubbed with soap and water and then washed in a solution of carbolic acid (1 to 50) or bichloride (1 to 5,000), and every particle of clothing changed. The hair and the scalp should be thoroughly washed and disinfected.

The nurse should be quarantined with the patient, and should not mingle with other members of the family until a complete change of clothing has been made, and hands and face thoroughly disinfected. The nurse and all others in close contact with a severe case should use frequently an antiseptic gargle and a nasal spray. The room should be in that part of the house most easily quarantined, usually on the top floor; during the attack it should be stripped of upholstery, hangings, and carpet, and should be freely ventilated and kept as clean as possible. All dust should be removed with damp cloths which should afterwards be burned; the floor should occasionally be sprinkled with a bichloride solution (1 to 1,000). The presence in the room of vessels filled with antiseptic fluids is of little or no practical value. The same may be said of sheets wet in carbolic or other solutions and hung about the room. Carbolic-acid poisoning has been known to result from this practice. After an attack it should be remembered that the room is probably a greater source of danger than the patient. Smooth walls should be wiped with damp cloths wrung out of a bichloride solution (1 to 2,000). The wood-work should be washed in the same solution and the floor scrubbed with it. After a thorough cleaning, while the floor is still wet and walls damp, the apartment should be fumigated with sulphur, or better with formalin. A simple method of using formalin is by Schering's lamp and tablets. If fumigation is to be efficient the room must be tightly closed, all cracks being stopped with cotton, and larger openings about doors, windows, and fire-places sealed by pasting paper over them. Bedding, cushions, pillows, carpets, etc., should be hung over chairs or upon lines strung about the room. Books should be suspended from covers so that the leaves are exposed. After fumigation, the room should remain closed for twelve hours. After a severe case, the walls should be painted or whitewashed, or if papered, the wall-paper should invariably be renewed and the wood-work repainted. Simply airing a room after an attack is of little or no benefit. An instance is on record of a patient contracting the disease in a room in which the windows had been open constantly for three months. The carpets, bedding, hangings, and upholstery are best disinfected by steam. Where this is impossible, after a severe case the mattress and pillows should be burned. Bedding, blankets, and other articles should be boiled, and afterward exposed to sunlight for a long time out of doors.

The bedclothes, linen, and clothing removed from the patient during an attack, should be put at once into a solution of carbolic acid (1 to 20), or zinc sulphate four ounces, common salt two ounces, and water one gallon, and afterward boiled at least two hours in the same solution. Instead of handkerchiefs, pieces of old muslin, surgeon's gauze, or absorbent cotton, should be used for cleansing the nose and mouth of the patient and burned immediately.

The physician in attendance upon a case should leave his coat and overcoat in an anteroom, and put on a long gown or rubber coat, sufficiently large to cover all his clothing. This should always be worn in the sick-room, and boiled or disinfected when the case is finished. For a single visit the overcoat may be worn in the room, but the clothing should be changed before visits to other children are made. After every visit the physician's hands and face should be thoroughly washed with soap and then with a disinfectant solution. A physician in attendance upon scarlatinal patients should not attend obstetric cases or other patients with recent wounds. The great liability of such cases to contract scarlatina should never be forgotten. If, in emergencies, it becomes necessary to attend such patients, the physician should change all his clothing and disinfect his hands, face, hair, and beard, with the greatest thoroughness.

Schools are the hot-beds for the spread of scarlet fever. The greatest sources of danger are the mild or walking cases in which the disease has not been recognised, and the clothing of patients who have had a severe form of the disease. As a rule, a child should be kept from school six weeks from the beginning of the attack, and the certificate of a physician should be required before re-admission, stating not only that the desquamation is complete, but also that the child is suffering from no sequelæ. Other children in the household should not be allowed to attend schools of any kind during the period of active symptoms; they should be kept at home on the average for a month. This precaution is necessary, first, because they might carry the disease from the child at home; secondly, because otherwise they might themselves attend school while suffering from the disease in a very mild form or during the period of invasion. When the sick child is completely isolated, the danger from the first source is very slight. During severe epidemics it frequently becomes necessary to close all schools.

During desquamation the spread of the disease may be in a measure prevented by the free use of inunctions and warm antiseptic baths. All the excreta from the patient should be disinfected throughout the disease, best by a carbolic solution (1 to 20). If cases of scarlet fever are to be transported, this should be done only in a vehicle which can be easily disinfected. Under all circumstances as few persons as possible should come in contact with the patient.

In general, it is to remembered that the danger is first from the patient, secondly from the room, and thirdly from the nurse. Special attention should always be given to the complete and immediate isolation of the first case which appears in an institution or community, which should apply to mild as well as severe forms of the disease.

Treatment.—There is as yet no specific for scarlet fever. The physician's duty in the average case consists in (1) establishing proper quarantine and the carrying out of adequate means of disinfection; (2) the hygienic care of the patient; (3) directing the diet; (4) watching for complications, especially otitis and nephritis. It should be borne in mind that otitis is rarely accompanied by pain or tenderness, and is recognised only by an examination of the ears; also that severe and fatal nephritis may follow mild as well as severe cases.

Mild attacks require no medicine. Children should be kept in bed for at least a week after the fever has subsided, and upon fluid diet for a period of three weeks. This is an important matter in the prevention of nephritis. During the height of the eruption, the intense itching of the skin may be allayed by sponging with a weak carbolic-acid solution, or by inunctions with vaseline, or by the free use of rice powder. Plenty of fresh air should always be secured in the sick-room. As soon as the fever and rash have disappeared, daily warm baths with soap and water should be used, after which the entire body should be anointed with carbolised vaseline, or boric acid and vaseline, five-per-cent strength, with the two-fold purpose of facilitating desquamation and disinfecting the scales. In case the skin becomes irritated by this treatment, bran baths may be substituted for soap and water.

The temperature does not usually require interference when it only occasionally rises to 104° or 104.5° F. But if there is hyperpyrexia, or a temperature which ranges from 104° to 105.5° F. or over, antipyretic measures are called for. Cold is much safer and more certain than drugs. Sometimes cold sponging is sufficient, but in the great proportion of cases the cold pack or bath is required. The use of cold in the reduction of temperature is especially indicated in septic cases with typhoid symptoms, and in those with pronounced cerebral symptoms.

The nervous symptoms are frequently better controlled by ice to the head and by cold sponging than by medication. Antipyretic drugs may be relied upon to control restlessness and promote sleep, and in mild cases to effect a moderate reduction in temperature. Phenacetine is usually to be preferred.

As soon as the pulse becomes weak or rapid and irregular, or the first sound of the heart feeble, stimulants should be given, no matter at what stage of the disease. In septic, or malignant cases, or in those accompanied by severe angina, adenitis, or cellulitis, alcoholic stimulants should be used freely. Digitalis is especially valuable when the pulse is weak and the tension low. It may be given alone or combined with strychnine; one minim of the fluid extract of digitalis, and gr. $\frac{1}{200}$ of strychnine being the initial doses for a child of five years.

The erythematous sore throat requires no treatment except the use of a mild antiseptic gargle. If there is a profuse nasal discharge, gentle nasal syringing (page 58) with a warm saline or boric-acid solution may be used with the hope of preventing infection of the middle ear. The local treatment of the membranous angina is the same as that of other cases of pseudo-diphtheria.

Milder forms of adenitis require no local treatment. When severe, the glands should be covered with ichthyol, and an ice-bag applied continuously. Poulticing almost invariably does harm. If an abscess forms, early incision should be practised.

The ears of patients with severe throat symptoms should be examined daily in order that there may be no delay in performing paracentesis should this become necessary. Any rise in temperature should direct attention to the ears. The indications for the operation are the same as in other severe forms of otitis.

The physician should be constantly on the watch for the development of nephritis, not only during the febrile period, but also during convalescence. Repeated examinations of the urine are absolutely necessary. These are much facilitated by having a rack of test tubes and the ordinary reagents for detecting albumin in the sick-room, so that the physician may himself examine daily a fresh specimen of urine. The nurse should be instructed to measure and record accurately the twenty-four hours' urine throughout the attack. The treatment of scarlatinal nephritis has been considered in the chapter devoted to Diseases of the Kidney. Diffuse cellulitis of the neck calls for free, early incision as the only means of preventing extensive sloughing.

Sera prepared by means of several different varieties of streptococci have been produced and extensively used without any uniform or striking success. One has lately been produced by Moser (Vienna) concerning whose effects there is much more favourable evidence. Escherich, Bokay, and other reliable Continental observers in their reports, declare that its effects are not less striking than those obtained from diphtheria antitoxin. It is not yet on the market.

During convalescence, tonics, particularly iron and digitalis, are called for. The urine should be frequently examined for a long time; antiseptic gargles and a nasal spray or syringe should be used as long as a purulent discharge from the nose or pharynx continues.

CHAPTER II.

MEASLES.

Synonyms: Rubeola, Morbilli.

MEASLES is an epidemic contagious disease, more widely prevalent than any other eruptive fever; very few persons reach adult life without contracting it. One attack usually confers immunity. It is highly contagious even from the beginning of the invasion, and spreads with great rapidity from the patient to all susceptible persons exposed. The poison, however, does not cling so long to clothing or apartments as does that of scarlet fever. Measles has a period of incubation of from eleven to fourteen days; a gradual invasion of three or four days with symptoms of an acute coryza; a maculo-papular eruption which appears first upon the face and spreads slowly over the body, and which lasts from four to six days. This is followed by a fine bran-like desquamation, which is complete in about a week. The mortality is low, except among infants and delicate children, in whom it may reach 30 or even 40 per cent. In institutions for infants and young children no disease is more to be dreaded than measles, not only on account of its severity, but from the frequency with which, in such subjects, it is complicated by broncho-pneumonia.

Etiology.—The essential cause of measles is as yet unknown. It is generally believed to be due to a micro-organism, but, as in the case of scarlatina, all attempts to isolate it have thus far been unsuccessful. The poison is one which possesses remarkable powers of diffusion, but whose viability is much less than that of most of the pathogenic germs which are known. Only a short exposure is required to communicate the disease, and even close proximity to a patient does not seem necessary. One instance has come under my own observation where measles was apparently conveyed by an exposure of half an hour across a hospital ward, a distance of at least fifteen feet.

Predisposition.—Very young infants do not so readily contract measles, but all other children are extremely susceptible. The disease broke out in a cottage of the New York Infant Asylum which was occupied by twenty-three children, nearly all of them being under two years old; only four escaped, all these being under five months old. In an epidemic reported by Smith and Dabney, 110 unprotected children, between the ages of eight and eighteen years, were exposed and only two escaped.

In the Nursery and Child's Hospital, during the epidemic of 1892, there were 62 children over two years of age; five were protected by a previous attack and escaped; of the remaining 57 children, 55 took the disease. There were also in the institution 113 children under two years old; of this number 78 per cent took the disease; but, although a number were exposed, not one child under six months old contracted measles. The age of the persons affected depends much upon the length of time since the last outbreak of the disease. In an epidemic occurring in the Island of Guernsey, where the disease had not prevailed for many years, all ages were affected, the youngest being twelve days old, and the oldest, a man and wife, each aged eighty years. Somer has reported an instance of an eruption of measles appearing in a child twelve hours after birth; the mother was suffering from the disease at the time. Gautier has collected six additional cases, where measles either existed at the time of birth or developed within a few hours after it.

Except, then, in early infancy, the probabilities are very strong that every child exposed to measles will contract the disease. Occasionally, however, one is seen who seems insusceptible to the poison, no matter how close the exposure.

Epidemics of measles are more frequent and more severe during the winter and spring months. They are least frequent and mildest during the autumn months.

Incubation.—In 144 cases,* in which the period of incubation could be definitely traced, it was as follows:

Incubation of less than nine days	3	cases.
" " nine or ten days	22	"
" " eleven to fourteen days	95	"
" " fifteen to seventeen days	19	"
" " eighteen to twenty-two days	5	"

Thus in 66 per cent of the cases the incubation was between eleven and fourteen days, and in only one case was it less than a week. The constancy of the incubation period is strikingly shown in some epidemics. Thus in the one reported by Smith and Dabney in an institution in Virginia, exactly eleven days after the rash appeared in the first case, the disease developed in twenty children—no cases having occurred in the interval.

Duration of the infective period.—This is much shorter than in scarlet fever, and the average duration may be placed at three weeks. Haig-Brown discharged fifty-eight cases on or before the twenty-ninth day of the disease, and in no instance was measles spread by these children.

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* About twenty-five of these are taken from my own records; the remainder are mainly isolated cases, scattered through medical literature. The incubation is reckoned from the time of exposure to the beginning of the catarrh.

Ransom, however, records one instance in which it was communicated thirty-one days after the appearance of the rash.

Measles is highly contagious from the beginning of the catarrhal symptoms. A case occurred in the Babies' Hospital under my own observation, in which a child conveyed the disease four days before the rash appeared. Ransom reports another precisely similar. An instance has been related to me by Dr. S. W. Lambert, where, of thirteen little girls who were at a children's party, only one escaped measles, the source of infection being a child who showed no rash until the following day; the child who escaped had previously had measles. The period of greatest contagion is still a matter of dispute, the general belief being that it is coincident with the highest temperature, the full eruption, and the most severe catarrhal symptoms.

With the fading of the eruption and the subsidence of the catarrh, the communicability of measles diminishes rapidly. It is relatively feeble during desquamation, and soon after this period it usually ceases altogether. It is generally proportionate to the severity of the catarrhal symptoms, and where these are protracted it is probable that the disease may be communicated for a much longer period than that mentioned.

Mode of infection.—Measles is usually spread by direct contagion, very infrequently through the medium of clothing, furniture, or a third person. Townsend (Boston) records an instance in which one family moved into a tenement house on the same day on which it was vacated by another family in which two children had suffered from measles, one of them fourteen and the other eighteen days previously. The apartments were not fumigated or disinfected, and, although there were two susceptible children in the incoming family, they did not contract the disease. Measles rarely if ever clings to apartments for weeks or months, as does scarlet fever. Many instances are on record in which the disease has been carried by a third person; but, after all, this rarely happens, unless the contact both with the sick and the well child is very close and the interval short. It is very seldom that measles is carried by a physician who takes even ordinary precautions. In a case reported by Girom, the clothing of a patient is stated to have conveyed the disease nineteen days after an attack, but this must be regarded as very exceptional.

Lesions.—The only constant lesions of measles are those of the skin and the mucous membranes, chiefly of the respiratory tract. According to Neumann, the process in the skin is of an inflammatory character, but is more superficial than in scarlet fever. There is congestion, accompanied by an exudation of round cells about the small blood-vessels, and also about the sweat and sebaceous glands, and the papillæ. To this exudation and the œdema, the swelling of the skin is due. It occurs everywhere, but is especially noticeable upon the face.

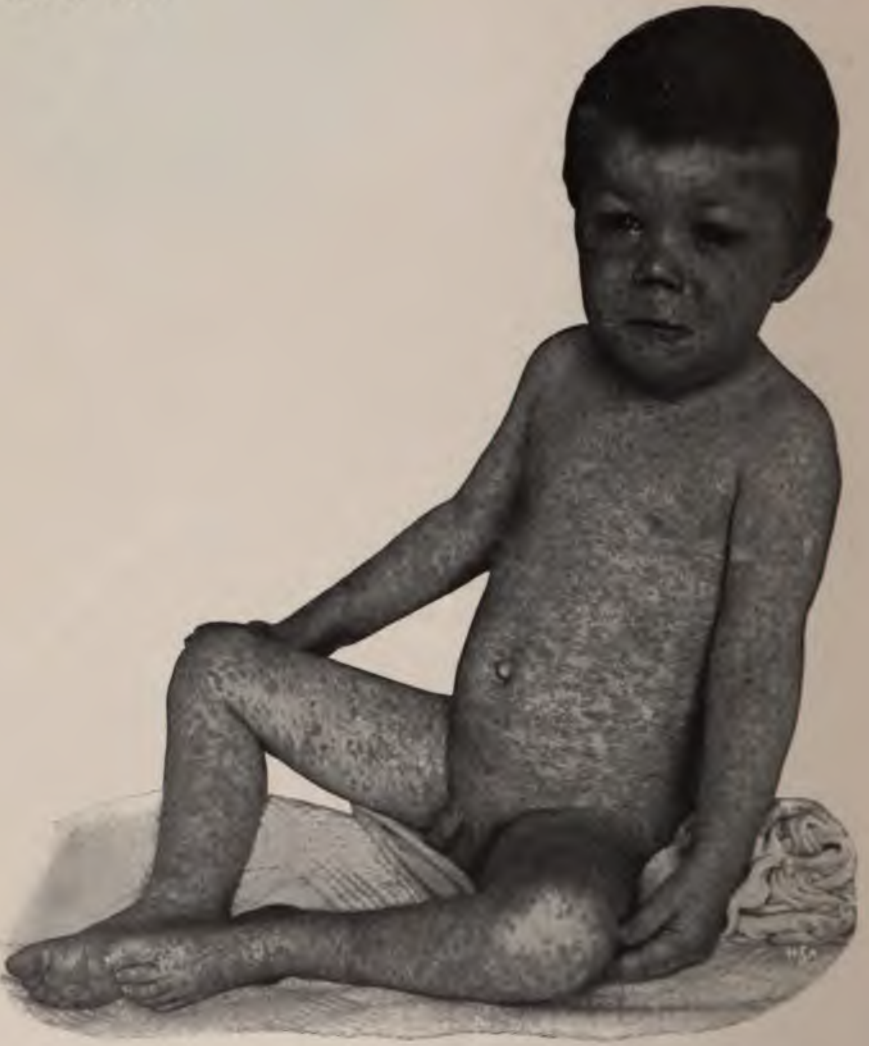
The changes in the mucous membranes are quite as much a part of the disease as are those of the skin. There is a catarrhal inflammation affecting the conjunctivæ, nose, pharynx, larynx, trachea, and large bronchi, which varies in intensity with the severity of the attack. In the most severe forms in infants and in young children, this inflammation extends with great uniformity to the small bronchi, and usually to the air vesicles, causing broncho-pneumonia. In severe cases, the lesion in the pharynx and larynx also, instead of being catarrhal, may be membranous; the larynx being much more frequently involved, and the ears much less so, than in scarlet fever. Freeman has described areas of focal necrosis in the liver similar to those found in diphtheria; they were present in four of twelve cases examined. The lesions of the lungs and of other organs will be more fully considered under Complications.

The bacteria which are associated with the lesions of the respiratory tract are the staphylococcus and the streptococcus, separately or together, and either form may be associated with the pneumococcus (see Bacteriology of Broncho-Pneumonia, page 532). The poison of measles produces conditions in the mucous membranes of the respiratory tract which are especially favourable for the development of these bacteria. They are present in the mouth in great numbers; they may cause pneumonia, otitis, and other local inflammations, and the pneumococcus or streptococcus may produce a general septicæmia.

Symptoms.—*Invasion.*—As a rule, the invasion of measles is gradual, both the fever and catarrhal symptoms increasing steadily up to the appearance of the eruption. The characteristic symptoms of the invasion are those of a severe coryza—suffusion of the eyes, increased lachrymation, photophobia, sneezing, and a discharge from the nose. The hoarse, hard cough indicates that the catarrhal process has involved the larynx and trachea, as well as the visible mucous membranes. Frequently the patient complains of some soreness of the throat, and on inspection there is seen moderate congestion of the tonsils, fauces, and pharynx. On the hard palate are frequently seen small red spots. Much more characteristic are the minute white spots upon the mucous membrane of the cheeks, known as Koplik's sign (see Diagnosis). The constitutional symptoms are indefinite, and may be met with in almost any disease. There is dulness, headache, pains in the back, and the usual symptoms of *malaise;* there is rarely vomiting or diarrhœa. Drowsiness is a frequent symptom, and is regarded by the laity as characteristic.

The exceptional cases in which the invasion is abrupt are puzzling. There may be a sudden accession of fever with vomiting, and even convulsions, as in a case lately under my observation. Not infrequently, when the disease prevails epidemically, the invasion is sudden, with high fever and pulmonary symptoms which are so severe as to mask everything else until the rash makes its appearance, the case up to that time being often regarded as one of primary pneumonia or of influenza. The

PLATE XVI.



Eruption of Measles.

On the face and trunk the eruption is rather more confluent than is usual; on the upper part of the chest, on the lower part of the abdomen, but especially on the left arm, many hæmorrhagic spots are seen. The eruption on the lower extremities and feet is typical in appearance.

duration of the stage of invasion—i. e., from the beginning of the catarrh until the eruption—in 270 cases of which I have notes, was as follows:

1 day or less	35 cases.	6 days	20 cases.
2 days	47 "	7 "	6 "
3 "	64 "	8 "	2 "
4 "	64 "	9 "	2 "
5 "	29 "	10 "	1 case.

From this table it will be seen that the length of the period of invasion varies considerably—more, I think, in infants and very young children (most of these were under three years old) than in those who are older. In the greater number of cases it lasts from two to four days.

Eruption.—The rash usually appears on the third, fourth, or fifth day of the disease—in the largest number upon the fourth day. As a rule, it is first seen behind the ears, on the neck, or at the roots of the hair over the forehead. It appears as small, dark-red spots, which are at first few, scattered, and not elevated, resembling flea-bites. In twenty-four hours the macules are much more numerous, and many of them have become papules. They frequently group themselves in crescentic forms. They are usually separated by areas of normal skin, but where the rash is intense they are frequently coalescent. From the time of its first appearance to the full development of the rash on the face, is usually about thirty-six hours, but may be from one to three days. With a full eruption (Plate XVI) there is considerable swelling of the face, especially about the eyes, and the features are sometimes scarcely recognisable. On the second day of the rash it begins to appear upon the neck beneath the chin, the upper part of the chest and back; on the third day the trunk is covered, and scattered spots are seen upon the extremities. The rash appears last upon the lower extremities, and by the time it is fully out upon them it has usually begun to fade from the face. In mild cases it remains discrete, but in severe ones it is frequently confluent upon the face and upon the extensor surfaces of the extremities. As a rule, it covers the entire body, even the palms and soles.

The eruption fades slowly in the order of its appearance, and there is left behind, in typical cases, a slight brownish staining of the skin, which often remains for a week or more. The duration of the rash is from one to six days, the average being four days.

There are many cases in which the rash does not follow the typical course described: (1) Instead of spreading gradually, the entire body may be covered in a few hours. (2) The rash may be hæmorrhagic. This condition was present in about five per cent of my cases. The whole eruption may be hæmorrhagic, or it may be so only upon certain parts—usually the abdomen or extremities. Under such circumstances small petechial spots take the place of the macules—the "black measles" of the older writers. It is in most cases a bad, but by no means a fatal symptom. I have seen it in several cases that were not especially severe. (3) The rash may be very faint, and of short duration, being scarcely elevated at all. (4) It may consist of very minute papules, closely resembling the rash of scarlet fever. It is to be remembered, however, that the irregular eruptions of scarlet fever much more frequently resemble measles than *vice versa*. (5) It may be very scanty, and late in its appearance; particularly in cases of great severity and hyperpyrexia—the so-called malignant cases. (6) Temporary recession of the eruption may occur at any time during the height of the disease, and is usually due to heart failure. A recurrence of the eruption after it has run its usual course is something which I have never seen; although such cases have been reported, I believe them to be very exceptional.

During the first two days of the eruption, the local and constitutional symptoms increase in severity, both usually reaching their maximum at the time of the full development of the rash upon the face. The skin is swollen, and the seat of intense itching and burning. The eyes are very red and sensitive to light, and there is swelling of the conjunctivæ with an abundant production of mucus or muco-pus, causing the lids to adhere. There is pain on swallowing, also swelling of the glands at the angle of the jaw or in the post-cervical region. The cough is frequent and very annoying. There is complete anorexia, and often diarrhœa. The tongue is coated, and may show at its margin enlarged papillæ, somewhat resembling the "strawberry" appearance of scarlet fever. As the rash fades the temperature declines rapidly, often reaching the normal in two or three days. The catarrhal symptoms now subside, and soon the patient is convalescent. Within a day or two after the fever has ceased, the rash disappears.

Desquamation.—This begins almost as soon as the rash has subsided, and is first noticed on the face and neck, where the eruption first appeared. The nature of the desquamation is invariably fine, branny scales, never in large patches, as in scarlet fever. It is often quite indistinct and may be overlooked. Its usual duration is from five to ten days. It may, however, be prolonged for two weeks. The amount of desquamation varies considerably in the different cases. It is most marked in those in which there has been an intense eruption. There is frequently noticed at this time an odour about the patient which is quite characteristic of measles. During this stage the cough often persists and the eyes remain weak and very sensitive to light, but in other respects the patient usually feels perfectly well.

1. *The mild cases.*—The mildest cases are distinguished by low temperature, which at the height of the eruption usually reaches 102° F., but rarely lasts more than four days. The eruption is often scanty, and is never confluent. The swelling, itching, and other cutaneous symptoms

are wanting, as is also the intense red colour of the skin. The rash is frequently obscure, and, without the other symptoms, hardly sufficient for diagnosis. The catarrhal symptoms are more uniform than the rash, but these are very mild as compared with the usual form. The duration of the rash is shorter, desquamation is scarcely perceptible, and there are no complications.

2. *The cases of moderate severity.*—The course of measles is much more regular in children over three years old than in infancy. In the former, the symptoms of invasion come on gradually, and the temperature rises steadily until the appearance of the eruption, which is in most cases on the third or fourth day of the disease. Figs. 195 and 196 represent the typical temperature curve in average uncomplicated cases. Such a curve was seen in 44 per cent of 173 cases in which careful observations were made. Sometimes the decline in the fever is very rapid, almost a crisis, as in Fig. 195, but more often it falls gradually, as in Fig. 196. In such cases the duration of the fever is from five to nine days, the average being about a week. The other symptoms follow very closely the course of the fever. The maximum temperature is nearly always coincident with the full rash upon the face, at this time usually being in un-

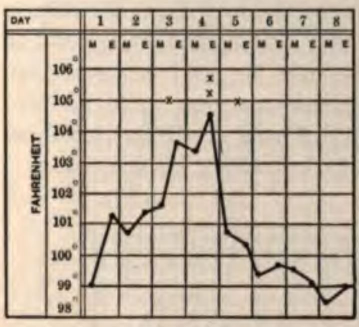


Fig. 195.

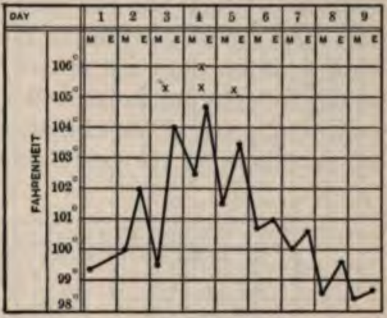


Fig. 196.

Fig. 195.—Temperature curve in uncomplicated measles, showing the gradual rise and critical fall; patient ten years old; × = first eruption; $\overset{\times}{\times}$ = full eruption on the face.

Fig. 196.—Typical curve in uncomplicated measles, with gradual rise and gradual fall; patient three years old.

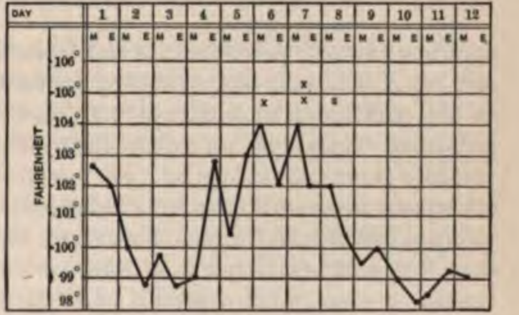


Fig. 197.—A not infrequent temperature curve in measles, showing abrupt invasion, but subsequent course typical; uncomplicated case; patient nine months old.

complicated cases from 103° to 104° F. in older children, and 104° to 105° in infants and young children.

A not very uncommon temperature curve is that of Fig. 197, where the onset of the disease is marked by a sudden rise to 102° or even 104° F., with a fall nearly or quite to normal on the second day, after which the fever rises gradually, as in the first group. This curve was seen in 5 per cent of my cases.

3. *The severe cases.*—In Fig. 198 is shown a type of the disease which is more frequent in infants than in older children, the important features being the late eruption and the continuance of the high fever for several days after the rash has begun to fade. Such a prolonged course and so high a temperature are almost invariably due to some complication, usually broncho-pneumonia. Where the pneumonia goes on to the production of areas of consolidation, the fever usually continues for three and sometimes for four weeks, even though terminating in recovery.

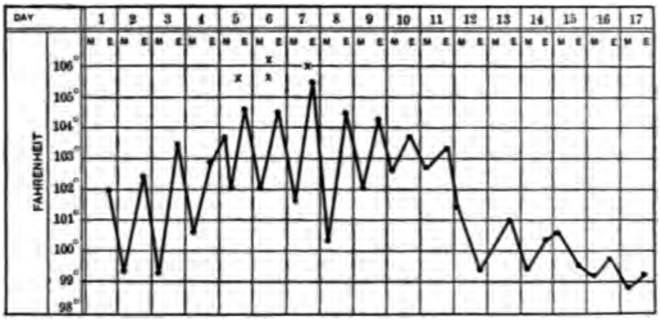


Fig. 198.—Measles with prolonged invasion; continuance of high temperature after full eruption due to severe bronchitis and diarrhœa; child two years old.

Figs. 199 and 200 illustrate two types of the disease which are often seen when measles is complicated by pneumonia. In cases like that shown in Fig. 199 the onset is abrupt with high temperature, prostration, and pulmonary symptoms not unlike those of primary pneumonia. A temperature curve resembling this was seen in 28 of 173 cases. The rash is often late in appearance; it is faint and altogether irregular; it may recede after the first day and reappear after an interval of one or two days. The catarrhal symptoms are not marked, but the whole force of the disease seems to be expended upon the lungs. The diagnosis of these cases presents great difficulties, and very often it would not be made but for the fact that there are other cases of measles in the family or the institution. This form is usually seen in infants, and it is usually fatal.

In other cases marked by a sudden severe onset, the system seems to be overpowered by the poison of the disease itself. There is profound depression, and hyperpyrexia, and the patient may die from toxæmia with cerebral symptoms before the appearance of the rash or just as it is beginning to show itself. Sometimes the pulmonary symptoms are entirely wanting; at others the rash, if it appears, is hæmorrhagic.

In still another group of cases the onset is not violent, and for the first two days the attack may appear to be of only average severity; but there may then develop, often quite suddenly, pulmonary symptoms of such intensity as to cause death within twenty-four hours. The eruption, if seen at all, is faint and not characteristic (Fig. 200).

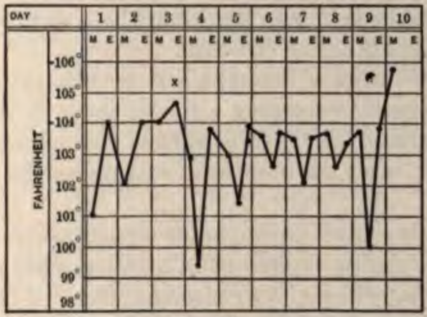


Fig. 199.

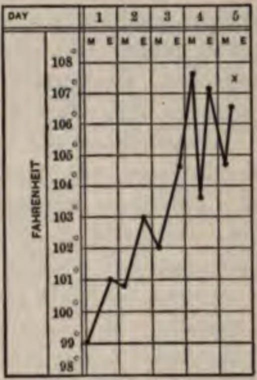


Fig. 200.

Fig. 199.—Fatal attack of measles, complicated by broncho-pneumonia; very severe symptoms from the onset; patient eighteen months old; death on tenth day.

Fig. 200.—Fatal attack of measles, complicated by broncho-pneumonia; early invasion mild, but rapid development of severe symptoms on fourth day; rash on last day; patient eight months old.

A secondary rise in the temperature after it has once fallen to normal was seen in 8 of 173 cases, being due to the development of otitis, ileocolitis, or pneumonia.

Complications and Sequelæ.—The most frequent and most important complication of measles is broncho-pneumonia, and next to this are ileocolitis, otitis, and membranous laryngitis. Most of the others are infrequent; all complications are relatively rare in children over four years old.

Lungs.—The greatest danger in measles arises from pulmonary complications, and the frequency is greatest in children under two years of age. In two epidemics in the Nursery and Child's Hospital, embracing about 300 cases, nearly all in children under three years old, broncho-pneumonia occurred in about 40 per cent of the cases. Of those who had pneumonia, 70 per cent died. Fortunately, such a record as this is never seen outside of asylums or hospitals for young children. Of 2,477 cases, embracing several epidemics of measles among children of all ages, pneumonia occurred in 10 per cent. My own experience in the post-mortem room fully bears out the statement of Henoch, that a certain amount of pneumonia is found in almost every fatal case. Pneumonia is more frequent and its mortality is higher in spring and winter epidemics than in those occurring at other seasons. It may develop at any time from the beginning of invasion until convalescence, but it mostly frequently begins about the time of full eruption.

Lobar pneumonia, although rare, occasionally occurs as a complication in children over three years old. In some epidemics many of the cases of pneumonia are complicated by severe pleurisy, which adds much to the danger from the disease. This form is frequently followed by empyema. Pneumonia is always to be suspected when the temperature continues high after the full appearance of the rash.

Bronchitis of the large tubes, always accompanied by tracheitis, is seen in every case of measles, possibly excepting a few of the very mildest. This is so constant a feature as hardly to be ranked as a complication. In nearly all of the severe cases the bronchitis extends to the medium-sized and smaller tubes.

Larynx.—A mild catarrhal laryngitis accompanies almost every case of measles. Severe catarrhal laryngitis is present in about ten per cent of the cases; it may give symptoms which closely resemble those of membranous laryngitis, and the two are no doubt often confused. (For the points of differential diagnosis see page 493).

Membranous laryngitis is more often seen as a complication of measles than of scarlet fever. It is especially seen in the epidemics of institutions. As a cause of death in older children it ranks next to pneumonia. When it develops at the height of the disease, it is sometimes due to the streptococcus; but when it develops at a later period, it is usually due to the diphtheria bacillus. The streptococcus inflammation is in most cases associated with similar changes in the pharynx or tonsils, but not always. True diphtheria, occurring as a complication of measles, not infrequently begins in the larynx. The streptococcus inflammation may be as serious in this connection as is true diphtheria, from the probability, which amounts almost to a certainty, of the development of broncho-pneumonia. No complication is more to be dreaded than this. The diagnosis between the true and pseudo-diphtheria may sometimes be made by the time of development, but only with certainty by cultures. I once saw in measles, where no false membrane was present in the rest of the larynx, a necrotic inflammation with almost entire destruction of the vocal cords—a condition which may be compared to that seen in the tonsils or epiglottis in scarlatina.

Throat.—A catarrhal angina is part of the disease, and is as characteristic of measles as is the eruption upon the skin. There is acute congestion and swelling of the tonsils, uvula, palate, and pharynx. In a certain proportion of cases, very much less frequently than in scarlatina, the development of membranous patches is seen upon the tonsils and adjacent mucous membranes. These occur in two or three per cent of the cases. They are to be regarded in the same light as similar conditions complicating scarlet fever, with these differences, that in measles there is much greater likelihood of the extension of the disease to the larynx, while extension to the nose and ears is much less probable. True diphtheria, however, may complicate measles, and cases of membranous inflammation of the tonsils or pharynx developing late in measles are usually due to the Klebs-Loeffler bacillus.

Although in most cases the inflammations of the pharynx and tonsils which accompany measles are not serious when they are due to the streptococcus, they are sometimes quite as severe as any that accompany scarlet fever. They may cause death from general sepsis apart from any affection of the larynx.

Digestive System.—Gastric disorders are not more common than in other febrile diseases; but diarrhœa is very frequent, and in summer it may be even more serious than the pulmonary complications. All forms of diarrhœa are seen, from that which results from simple indigestion to the severe types of ileo-colitis. This complication is most often seen in children under two years old. The most severe intestinal symptoms are not usually seen at the height of the primary fever; but, beginning at this time, they often increase in severity, and are most marked in the second and third weeks of the disease.

Catarrhal stomatitis is present in almost every case of measles; less frequently the herpetic form is seen. Ulcerative stomatitis is not uncommon, particularly in institutions. One of the worst complications of measles, but fortunately a rare one, is gangrenous stomatitis, or noma. This usually occurs in inmates of institutions, or in children with bad surroundings who were previously in wretched condition. It is nearly always fatal.

Gangrenous inflammations of other parts of the body are sometimes seen after measles, especially of the ear, the vulva, or the prepuce.

Nervous System.—I have seen convulsions at the onset of measles in but a single case. During the progress of the disease they are not so rare, and may occur in connection with otitis, meningitis, or severe bronchopneumonia—chiefly in infants.

Meningitis is rare, but either the simple or the tuberculous form may occur, more often, however, as a sequel than as a complication. Insanity, usually of a temporary character, occasionally follows measles. In the epidemic of 108 cases reported by Smith and Dabney, insanity was noted three times, all the cases terminating in recovery. Epilepsy and chorea are rare sequelæ.

Ears.—Otitis is a frequent complication in some epidemics; in others it is seldom seen. In one hospital epidemic it was noted in 14 per cent of the cases. This epidemic occurred in early spring and affected very young children, both of which circumstances are favourable for the development of otitis. Usually both ears are affected, but the otitis of measles is, as a rule, less serious than that of scarlet fever.

Eyes.—Simple catarrhal conjunctivitis accompanies nearly every case of measles. In the severe form there is a muco-purulent catarrh, which may attain any degree of severity. In neglected cases, and among children who are poorly nourished, especially in asylums, the disease is apt to extend to the cornea. Chronic conjunctivitis often persists after measles, particularly in the class of children just mentioned.

Lymph nodes.—Swelling of the lymphatic glands of the neck is frequent, but not generally severe, and rarely terminates in suppuration. Chronic enlargement may continue for months, and sometimes the glands may become tuberculous. Similar changes and similar consequences may occur in the glands of the tracheo-bronchial group.

Kidneys.—The infrequency of renal complications in measles is in striking contrast to scarlet fever. Transient febrile albuminuria is not uncommon, but a serious degree of nephritis, either clinically or at autopsy, I have never seen, and literature furnishes but few cases.

Heart.—Both endocarditis and pericarditis have occurred in the course of measles, but they belong to the rare complications. The same may be said of changes in the muscular walls of the heart.

Skin.—As complications, erysipelas, furunculosis, impetigo, and pemphigus have been noted; but all are rare.

Hæmorrhages.—Associated with the hæmorrhagic type of the eruption, severe and even fatal hæmorrhages may occur from the mucous membranes, and the latter are sometimes seen without the hæmorrhagic eruption.

Blood.—There is a leucocytosis of 15,000 to 30,000 beginning soon after infection, even before the invasion, and increasing for four or five days. The number of leucocytes then falls gradually to normal or below during the eruption. A marked leucocytosis at any time points to a complication, but its absence during eruption does not exclude one. The differential count shows the increase to be in the polynuclear neutrophiles.

Other infectious diseases.—Measles in institutions is often complicated by diphtheria. Scarlet fever or varicella occasionally occurs during measles, though it is rare that the two eruptions are exactly simultaneous. Epidemics of measles and whooping-cough frequently occur together or follow each other. The relation of measles to tuberculosis seems to be particularly close. In some cases general or pulmonary tuberculosis follows directly in the wake of measles, which seems to furnish, especially in the lungs, conditions which are favourable for the development of latent tuberculosis. As a late manifestation the most common one is tuberculosis of the bones, occurring as hip-joint disease, caries of the spine, etc. An attack of measles in a child with tuber-

PLATE XVII.

Fig. 1.

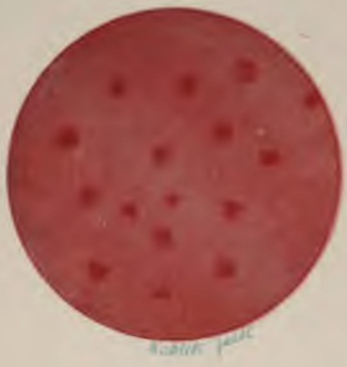


Fig. 2.

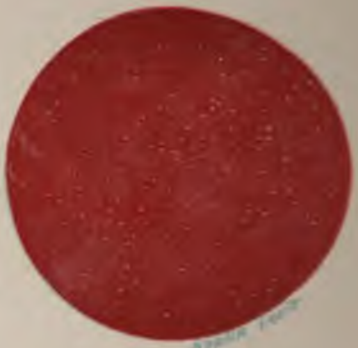


Fig. 3.

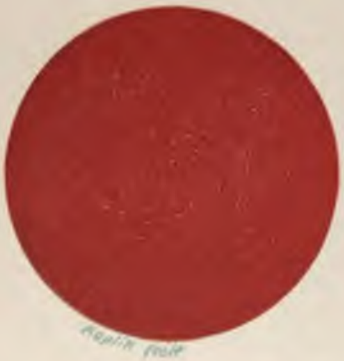
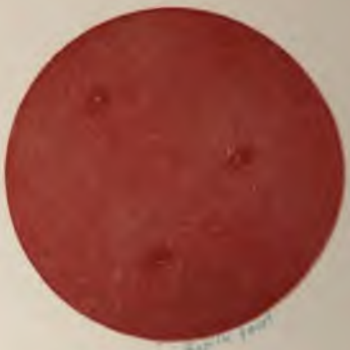


Fig. 4.



The Pathognomonic Sign of Measles (Koplik's Spots).

Fig. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

Fig. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

Fig. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

Fig. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

culous antecedents should, therefore, always be looked upon with apprehension.

Diagnosis.—A sign of the greatest diagnostic value is the buccal eruption. Although it appears that this was described many years ago by Flindt, of Denmark, it is to Koplik, of New York, that the credit belongs of its independent discovery and publication in 1896. It is generally known as "Koplik's sign." The unit of the eruption is a bluish-white speck upon a red ground; only a few of these may be present or the mucous membrane may be fairly peppered with them (Plate XVII). Often they are not seen except by careful search for which strong sunlight is necessary; artificial light is not satisfactory. The spots are best seen on the inside of the cheeks opposite the molar teeth, and in most cases only there; but they may be present on almost any part of the buccal mucous membrane. Their diagnostic value is due to the fact that they are nearly always present, that they are not found in other diseases, and that they usually appear two or three days before the skin eruption. They generally disappear at the time of full eruption.

I have recently had an opportunity to study the value of this sign in two epidemics of measles at the New York Foundling Hospital. Careful notes were kept in the second epidemic of 187 cases. Koplik's spots were unmistakably present in 169 cases, absent in 8, doubtful in 10. In 78 cases, fever, rash, and Koplik's spots were all present at the first observation. In 54 patients the sign was noted one day before the rash; in 25, two days before; in 4, three days before; in 3, four days before; and in 2, five days before. In 2 the spots were not seen until after the skin eruption; in one case they were present without any eruption. As this patient had been exposed and had a prolonged fever, it seems fair to regard the case as one of measles. In only one case was the buccal eruption seen before any elevation of temperature.

These facts, amply confirmed by other observations, indicate that Koplik's sign is of value in enabling us to make a diagnosis from one to three days before it is possible by the skin eruption, also in furnishing a new means of distinguishing measles from the other eruptive fevers, as well as from rashes due to drugs, antitoxin, etc.

Other important symptoms are the coryza, the gradual rise in temperature, and the eruption which appears first upon the neck and face, and slowly extends over the body. Cases which present the greatest difficulties in diagnosis are usually the very severe ones and those in infants.

Prognosis.—This depends upon the age and previous condition of the patient, the character of the epidemic, and the season of the year. Except in children under three years of age, the deaths from measles are few; but in institutions containing young children, no epidemic disease is so fatal.

The general mortality of the disease is from 4 to 6 per cent; but in epidemics in institutions for young children it has, in my experience, ranged from 15 to 35 per cent. The following table gives the figures of an epidemic in one institution in 1892:

From six to twelve months	42 cases;	mortality, 33 per cent.
" one to two years	51 "	" 50 "
" two to three years	27 "	" 30 "
" three to four years	20 "	" 14 "
" four to five years	3 "	" 0 "

In any single case the important symptoms for prognosis are the temperature and the character of the eruption. An initial temperature above 103° F., or one which remains high until the eruption appears, is a bad symptom. So also is one which rises after a full eruption, or which does not fall as the rash fades. The following table shows the highest temperature and mortality in 161 hospital cases:

Highest temperature not over 102°	6 cases;	mortality, 0 per cent.
" " 102° to 103·5°	14 "	" 7 "
" " 104° " 104·5°	49 "	" 16 "
" " 105° " 105·5°	65 "	" 40 "
" " 106° or over	27 "	" 80 "

A favourable eruption is one of a bright colour, covering the body, remaining discrete, and spreading gradually. It is unfavourable for the eruption to appear late, to be very faint, scanty, or hæmorrhagic, or to recede suddenly, as this is usually due to a weak heart.

Of 51 fatal cases, the cause of death was broncho-pneumonia in 45, ileo-colitis in 4, and membranous laryngitis in 2. More than half the deaths occurred during the second week, the earliest being upon the fifth day of the disease.

The ultimate result of an attack of measles may not be evident for some time. Cases in which the temperature persists for two or three weeks without assignable cause after the disease is apparently over, should be watched with the greatest solicitude. The explanation of this is most frequently to be found in the lungs, although the physical signs are often obscure. The condition may be either subacute pneumonia or pulmonary tuberculosis. Even though the attack of measles may not have been in itself severe, seeds are often sown the full fruits of which are not seen until long afterward. Chronic glandular enlargements which may or may not be tuberculous, chronic bronchitis, chronic laryngitis, subacute or chronic nasal catarrh, hypertrophy of the tonsils, and adenoid growths of the pharynx—all are frequent sequelæ.

Prophylaxis.—Measles is often regarded by the laity as so mild a disease that its prevention is thought of little importance, and no effort is made to limit its extension. The great probability that every person at some time in his life will have the disease, is no justification of unnecessary exposure. Although in older children measles is usually mild, this is not so in infants, who should be carefully protected from exposure. Special care should also be taken to avoid the exposure of delicate children or those with a strong tendency to pulmonary disease or to tuberculosis. In institutions it is of the utmost importance to secure prompt and complete isolation of the first case which appears.

The disease being usually spread by the patient and rarely from apartments, it follows that while early isolation is more important, there is not required the same thorough cleansing and disinfection which should follow every case of scarlet fever. In an institution, the ward or cottage from which a case has been removed should be quarantined for at least sixteen days after the appearance of the last case, and absolute security can not be said to exist until the end of three weeks. The same rule should be applied in private families where children who have been exposed should be quarantined apart from the patient, but not sent away. Under ordinary circumstances the quarantine of a case of measles should last three weeks from the beginning of invasion. It should be continued longer if there is pneumonia, otitis, or a nasal discharge.

Thorough cleansing and disinfection of the sick-room should be done before it is again occupied by children, and it should remain vacant at least two weeks. Children should be kept from all schools while the disease is in their homes, chiefly because they are otherwise liable to spread the disease while suffering from the early symptoms of invasion.

Treatment.—Measles is a self-limited disease, and there are no known measures by which it can be aborted, its course shortened, or its severity lessened. The indications are therefore to treat serious symptoms as they arise, and, as far as possible, to prevent complications, which are the principal cause of death.

The sick-room should be darkened, as the eyes are very sensitive to light. Every child with measles should be put to bed and kept there with light covering during the entire febrile period. There can be no possible advantage in causing a child to swelter by thick covering, under the delusion that the disease may be modified thereby. The food should be light, fluid, and given at regular intervals. If the conjunctivitis is severe, iced cloths should be applied to the eyes, which should be kept clean by the frequent use of a saturated solution of boric acid, the lids being prevented from adhering by the application of vaseline or simple ointment. The intense itching and burning of the skin may be relieved by inunctions of plain or carbolized vaseline. The cough, when distressing, may be allayed by heroin, small doses of opium, either in the form of codeine or the brown mixture. The restlessness, headache, and the general discomfort which accompany the height of the fever may be relieved by an occasional dose of phenacetine or antipyrine. As soon as the rash has subsided, a daily warm bath should be given, followed by inunctions to facilitate desquamation and prevent the dissemination of the fine scales.

The important indications to be met in the severe cases are very high temperature, cardiac depression, and nervous symptoms—dulness, stupor, sometimes coma, or convulsions. In some of the cases there is in addition dyspnœa and cyanosis, showing severe acute pulmonary congestion. For the nervous symptoms and high temperature, nothing is so reliable as the cold baths or packs (pages 49 and 50) and the nearly continuous use of ice to the head. I do not think there is any evidence that the use of cold increases the liability to pneumonia; but cold extremities, feeble pulse, and cyanosis, when associated with high temperature, call for the hot mustard bath, although ice should still be applied to the head. The indications for stimulants and the methods of using them are the same as in broncho-pneumonia, which is usually present in cases requiring them.

To diminish the chances of pneumonia, it is necessary that every patient should be kept in bed during the attack, and care exercised to avoid exposure; that the chest should be protected with flannel and rubbed daily with oil. But still more important is it in hospitals and institutions where most of the cases of pneumonia occur, to allow the patients plenty of air space, never crowding them together in small wards. If possible, cases complicated by pneumonia should be separated from simple cases. The pneumococcus and the streptococcus are found in the mouth in such numbers that systematic disinfection of the mouth may prove of value.

The danger of diphtheria as a complication may be greatly lessened if during epidemics of measles in institutions every case receives an immunizing dose of diphtheria antitoxin. This plan has been followed at the New York Foundling Hospital for several years with most striking benefit.

The bronchitis and broncho-pneumonia of measles should be managed as when they occur as primary diseases, since the coexistence of measles furnishes no new indications. The same is true of the diarrhœa, conjunctivitis, and otitis. Membranous laryngitis, pharyngitis, or tonsillitis should be treated like other cases of pseudo-diphtheria. Should cultures show the presence of the diphtheria bacillus, the case should be treated like one of ordinary diphtheria in the same situation.

During convalescence the eyes should be used very carefully for at least several weeks. Should the cough and slight fever persist, with or without physical signs in the chest, the patient should, if possible, be sent away to a warm, dry, elevated district, as the development of tuberculosis is always to be feared. Cod-liver oil should be given continuously throughout the succeeding cool season, and iron, wine, and other tonics according to indications. The cough itself should be treated as when it follows an ordinary bronchitis, creosote being more generally useful than any other drug.

CHAPTER III.

RUBELLA.

Synonyms: German measles; rötheln.

RUBELLA is a contagious eruptive fever which is rarely seen except when prevailing epidemically. It is characterized by a short invasion, with mild, indefinite symptoms, usually lasting but a few hours, and by an eruption which is generally well marked but of variable appearance. The constitutional symptoms are very mild, and the disease rarely proves fatal, not often being even serious. For a long time rubella was confounded with measles and scarlet fever, as the eruption sometimes resembles one and sometimes the other disease. Its identity is now fully established, and, as Strümpell well says, its existence is doubted only by those who have never seen it. The following peculiarities have been stated by Griffith (Philadelphia), who has written more fully on rubella than any other American writer, and to whom I am indebted for many facts in this article:

(1) Rubella is a contagious, eruptive fever, and not a simple affection of the skin; (2) it prevails independently either of measles or of scarlet fever; (3) its incubation, eruption, invasion, and symptoms differ materially from those of both these diseases; (4) it attacks indiscriminately and with equal severity those who have had measles and scarlet fever and those who have not, nor does it protect in any degree against either of them; (5) it never produces anything but rubella in those exposed to its contagion; (6) it occurs but once in the same individual.

Etiology.—Rubella is beyond question contagious, but is decidedly less so than either measles or scarlet fever; so that some observers have doubted its contagion altogether. It can be communicated at any time during its course, but is especially contagious during the early stage. Epidemics usually prevail in the winter or spring. As in the other eruptive fevers, a striking immunity is seen in infants under six months old; but, with this exception, all ages are liable to the disease.

The incubation of rubella varies considerably; the usual period is from fourteen to twenty-one days, although the limits are from ten to twenty-two days.

Symptoms.—*Invasion.*—This is rarely more than half a day, and in many cases no prodromata whatever are noticed, the rash being the first thing to attract attention. In a few cases there are mild catarrhal symptoms, with general *malaise* and slight fever. At other times there may be vomiting, convulsions, delirium, epistaxis, rigors, headache, or dizziness; but all are to be regarded as very exceptional.

Eruption.—Frequently a child wakes in the morning covered with the rash, no symptoms having been previously noticed. It generally appears first upon the face, and spreads rapidly to the whole body, the lower extremities being last covered. Less than a day is usually required for its full development. Exceptionally the eruption comes first upon the chest and back, and sometimes nearly the whole body is covered almost at once. The rash is occasionally observed in the roof of the mouth before it is visible on the face. In a considerable number of cases the entire body is not covered; but the rash is more constantly seen upon the face than upon any other part of the body.

Its character is subject to considerable variation. The eruption is most frequently composed of very small maculo-papules; they are of a pale-red colour, and vary in size from a pin's head to a pea. The spots are usually discrete, but may cover the greater part of the body where it is seen. On the face it is frequently confluent, and often appears here as large, irregular blotches of a red colour. From this description the rash will be seen to resemble that of measles more than that of any other disease. Very often, however, there is a tolerably uniform red blush which bears a close resemblance to the rash of scarlet fever; but even in such cases there will nearly always be found upon some part of the body, usually the wrists, fingers, or forehead, some typical maculo-papules. Between these two extremes all variations are seen. The colour of the eruption is sometimes dark red, and rarely it has been noted to be hæmorrhagic. The degree of elevation above the surface is also variable; sometimes this is so marked as to give to the skin a "shotty" feel, while in others the elevation is scarcely perceptible. The duration of the eruption is usually three days. Occasionally it lasts only two days, and it may last but one; it is rare for it to remain as long as four days. It fades in the order of its appearance, and more rapidly than the eruption of measles. A slight brown pigmentation of the skin sometimes remains for a few days after the rash.

The highest temperature is coincident with the full eruption; this does not usually exceed 101°, and often it is only 100° F. As a rule, the temperature continues but two days, falling as the eruption fades. Very often the fall to normal is abrupt. Rarely more severe cases are seen in which the fever lasts for two or three days, being 101° or 102° F. during the invasion, and rising to 103° F. or more during the full eruption. The other symptoms are in most cases even less marked than the fever. Occasionally catarrhal symptoms resembling a mild attack of measles are present, or a sore throat suggesting mild scarlet fever; but more frequently all these symptoms are absent. The eruption is usually out of all proportion to the other signs of disease.

Swelling of the post-cervical glands is one of the most constant features of rubella. In most epidemics it is seen in nearly all cases; but as a symptom for differential diagnosis it is not of great importance, as it is not uncommon in measles. The glandular swelling is most marked at the height of the disease; it is never very great, and subsides slowly without suppuration. Vomiting and diarrhœa are rare. Swelling and itching of the skin are usually present and sometimes marked. There is no leucocytosis in this disease.

Forchheimer * has described an eruption on the mucous membrane of the throat, or "enanthem," which he believes to be characteristic. It consists of minute, bright, rosy-red points, seen on the uvula and soft palate, rarely on the hard palate. It is present only in the first twenty-four hours.

Desquamation.—This is exceedingly variable. It is sometimes entirely wanting; writers who have observed some fairly typical epidemics have stated that it did not occur. In most cases, however, some desquamation is present, though it may be so slight as to be discovered only by a close examination. It is usually in the form of fine scales over the body and extremities. In a few cases it is more pronounced, and may be in larger flakes or patches.

Prognosis.—There are few diseases so free from danger as rubella. Complications and sequelæ are very seldom seen, and when present are usually of the mildest character.

Diagnosis.—The principal interest attaching to rubella is in its diagnosis. This is a matter of extreme difficulty, and often it is an impossibility. The characteristic thing about the disease is a well-marked eruption with very few other symptoms. Cases so closely resemble mild scarlet fever that the differentiation by symptoms may be impossible; it must be made by the circumstances under which it occurs, especially a prevailing epidemic. Scarlet fever with a low temperature and abundant rash should always be regarded with suspicion; also an abundant rash with little or no desquamation. The longer period of incubation in rubella may be of assistance. Koplik's spots furnish a valuable means of distinguishing measles from rubella. These difficulties in diagnosis can be appreciated only by one who has seen epidemics of measles and scarlet fever in institutions, and has watched the mild course of undoubted cases of these diseases which have there occurred.

It is never safe to make the diagnosis of rubella unless the disease is prevailing epidemically. Sporadic cases in which this diagnosis is made are, I believe, almost invariably instances of mild measles or scarlet fever. The first cases of rubella in an epidemic are usually overlooked. The continued absence in succeeding cases of the characteristic symptoms and complications of measles or scarlet fever should suggest to the physician that he is probably dealing with rubella.

Treatment.—None whatever is required for the disease excepting isolation, which should be complete until the diagnosis is positively determined. The individual symptoms and complications are to be treated as they arise.

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* Archives of Pædiatrics, 1898, 721.

CHAPTER IV.

VARICELLA.

Synonym: Chicken-pox.

VARICELLA is an acute, contagious disease, characterized by a c neous eruption of papules and vesicles and by mild constitutional sy toms, serious complications and sequelæ being very rare. Although l confounded with varioloid, its existence as a distinct disease has l generally admitted for many years.

Etiology.—It is well established that the contagium of the diseas contained in the vesicles, as it may be communicated by inoculation w their contents. The specific poison, however, has not yet been isola Varicella is contracted by exposure to another case or through the dium of a third person. It affects children of all ages, one attack be as a rule protective. It is very contagious, resembling measles in respect. The period of incubation is quite uniformly from fourteen sixteen days.

Symptoms.—Slight fever and general indisposition may be noti for twenty-four hours before the appearance of the eruption, but in m cases the eruption is the first symptom. It usually appears first u the face or trunk, as small, red, widely-scattered papules. The papule most cases come in crops, new ones continuing to appear for three four days, even upon the same part of the body. The earlier ones h generally begun to dry up by the time the later ones appear, so that stages of the eruption may be present at one time in the same regi this being one of its diagnostic features. The papules are at first v small, but gradually increase in size, and are surrounded by an are from one fourth to half an inch in width. Many of them go no furt than this stage, but the majority become vesicular. The vesicles usually flat, and vary a good deal in size—the largest being about fourth of an inch in diameter. The process of drying up generally gins at the centre, which causes a slight depression, giving the vesi a somewhat umbilicated appearance. The areola is most distinct at time of the fully-formed vesicle, and fades as the latter dries. Cru now form, which fall off in from five to twenty days, depending u the depth to which the skin has been involved. In the majority of ca no mark is left, but after the most severe attacks, where the true s has been involved, scars remain, and occasionally there is quite d pitting. Such marks are few in number, and are most likely to occ upon the face.

Sometimes, especially upon hands and feet, the vesicle appears wi out having been preceded by a papule; then there is no areola, and vesicle resembles a drop of water upon healthy skin. In most cases pustules are not seen, but they may develop in consequence of irritation or infection, the result of scratching, or in children who are poorly nourished. Under these circumstances deeper ulceration may occur, lasting for weeks. In rare cases there may be a necrotic inflammation about the site of the pock, a condition to which is sometimes given the name *varicella gangrenosa*. It is not peculiar to varicella, and is described elsewhere under the head of Gangrenous Dermatitis (page 936).

The pocks are usually most abundant over the back and shoulders. In mild cases only twenty or thirty may be found upon the entire body, but in severe cases the skin in certain regions may be nearly covered. The eruption is never confluent. The pocks are usually seen on the hairy scalp, and often on the mucous membrane of the mouth or pharynx —a point of some diagnostic value. In the latter situation the appearance is first as a tiny vesicle, and later as a superficial ulcer resembling that of herpetic stomatitis. Marfan and Halle have described cases of varicella of the larynx. Croupy symptoms were present, and in one case which proved fatal from pneumonia a tiny ulcer was found on the vocal cords.

The temperature is highest when the eruption is most rapidly appearing, this usually being the second or third day. In an average case it reaches only 101° or 102° F., and lasts but two days; in severe cases it may rise to 104° or 105° F., and lasts for four or five days. It falls gradually to normal as the rash fades. The other symptoms are mild and not characteristic.

Complications.—The most important complication is erysipelas, which develops about the pocks, particularly when they are deep and attended with some ulceration. I have known of three fatal cases from this cause. Adenitis, either simple or suppurative, and abscesses in the cellular tissue, are occasionally seen. Nephritis is very infrequent, but a number of cases are recorded. It may occur at the height of the disease, but more often at a later period, like the nephritis of scarlet fever. Varicella is quite frequently complicated by other infectious diseases. In the New York Infant Asylum epidemics of varicella and scarlet fever at one time occurred together, and in at least a dozen children both diseases were seen at the same time.

Diagnosis.—The diagnosis of varicella is usually easy, provided the following points are kept in mind: first, that the eruption comes out slowly and in crops, so that papules, vesicles, and crusts may be seen upon the skin in close proximity; secondly, that the umbilication is due only to the mode of drying up of the vesicle, which begins at the centre; thirdly, the appearance of the pocks upon the mucous membranes, and the history of exposure. It is distinguished from urticaria and other forms of skin disease by the presence of fever.

Treatment.—Although it is usually a trivial disease, isolation of cases of varicella should be enforced in schools and in institutions containing many infants. In the home, unless the other children are delicate or in poor condition, quarantine is unnecessary. The disease may probably be conveyed as long as the crusts are present, hence isolation should be maintained until they have fallen off. In most cases constitutional symptoms of the disease are so mild as to require no treatment.

Locally, the itching, when annoying, may be allayed by sponging with a weak solution of carbolic acid or the use of carbolized vaseline. When the crusts have formed, zinc ointment or vaseline containing two per cent ichthyol should be applied. Care is necessary to keep the skin clean, and in the case of infants, to prevent scratching. In severe cases the urine should invariably be examined.

CHAPTER V.

VACCINIA—VACCINATION.

Vaccinia (cowpox) is a febrile disease induced in man by inoculation with the virus obtained either directly from the cow (bovine virus) or from a person who has been inoculated (humanized virus). The disease is not contagious in the ordinary sense of the term, but is communicated by inoculation either accidental or intentional.

The nature of the protection against smallpox which vaccination affords is even now but imperfectly understood. The fact, however, remains one of the best attested in medical history. Its effect when systematically practised is graphically shown in the accompanying chart (Fig. [illegible]). It is the imperative duty of the physician to see to it that every young infant is vaccinated.

Re-vaccination.—Regarding the duration of the protective power of a single vaccination, positive statements are impossible. Nearly all writers are agreed that vaccination should be done in infancy, again at puberty, and a third time at about the age of twenty or twenty-five. Many also insist upon re-vaccination at about the seventh year. It is a safe rule when smallpox is prevalent to vaccinate every person who has not been successfully vaccinated within five years.

Choice of Lymph.—The substitution of bovine for humanized virus is now well-nigh universal. It has precluded the possibility of transmitting syphilis and greatly lessened the chances of other forms of infection. A further advance has lately been made by the introduction of "glycerinated" lymph. As now prepared, the lymph is taken from the calves under the most rigid aseptic precautions and emulsified with glycerin. The few saprophytic bacteria present soon die, so that when properly prepared the glycerinated virus is practically sterile.* It should

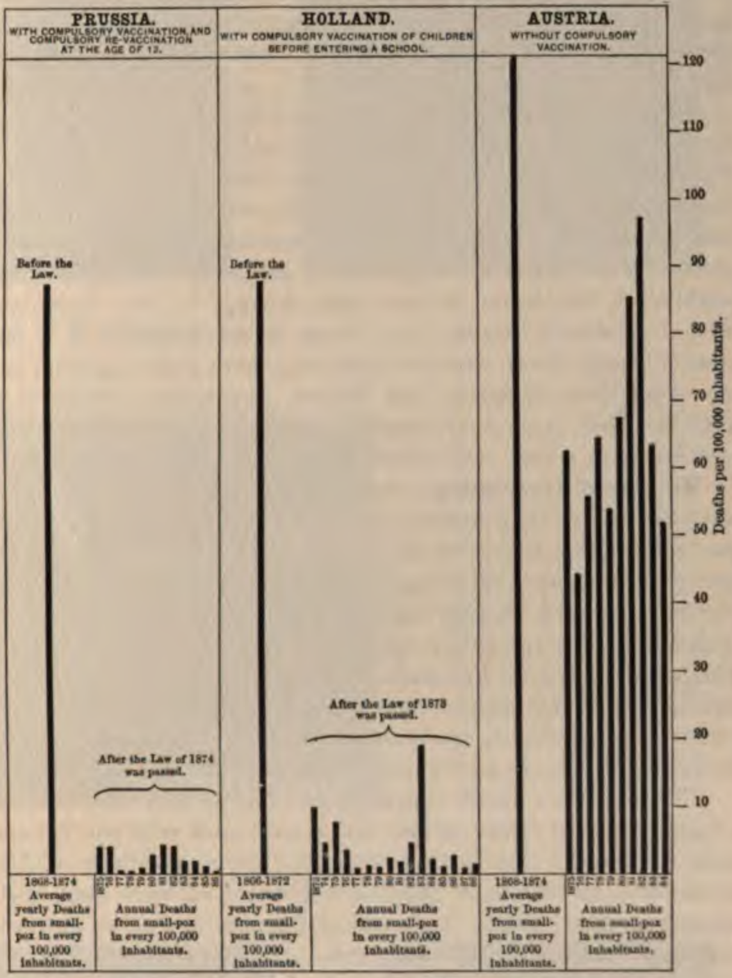

FIG. 201.—Table showing the protective power of vaccination. (Carsten.)

not be distributed until it has been carefully tested for pathogenic organisms of all kinds, particularly the tetanus bacillus. It is preserved and distributed in capillary tubes hermetically sealed; these are much safer than quills or ivory points, which may easily become contaminated by handling. After the lymph has been taken, the calves are killed in order to make certain that they are free from disease. The practical advantages of glycerinated lymph are so great that it has been officially adopted by the Governments of the United States, Great Britain, Germany, and many other countries.

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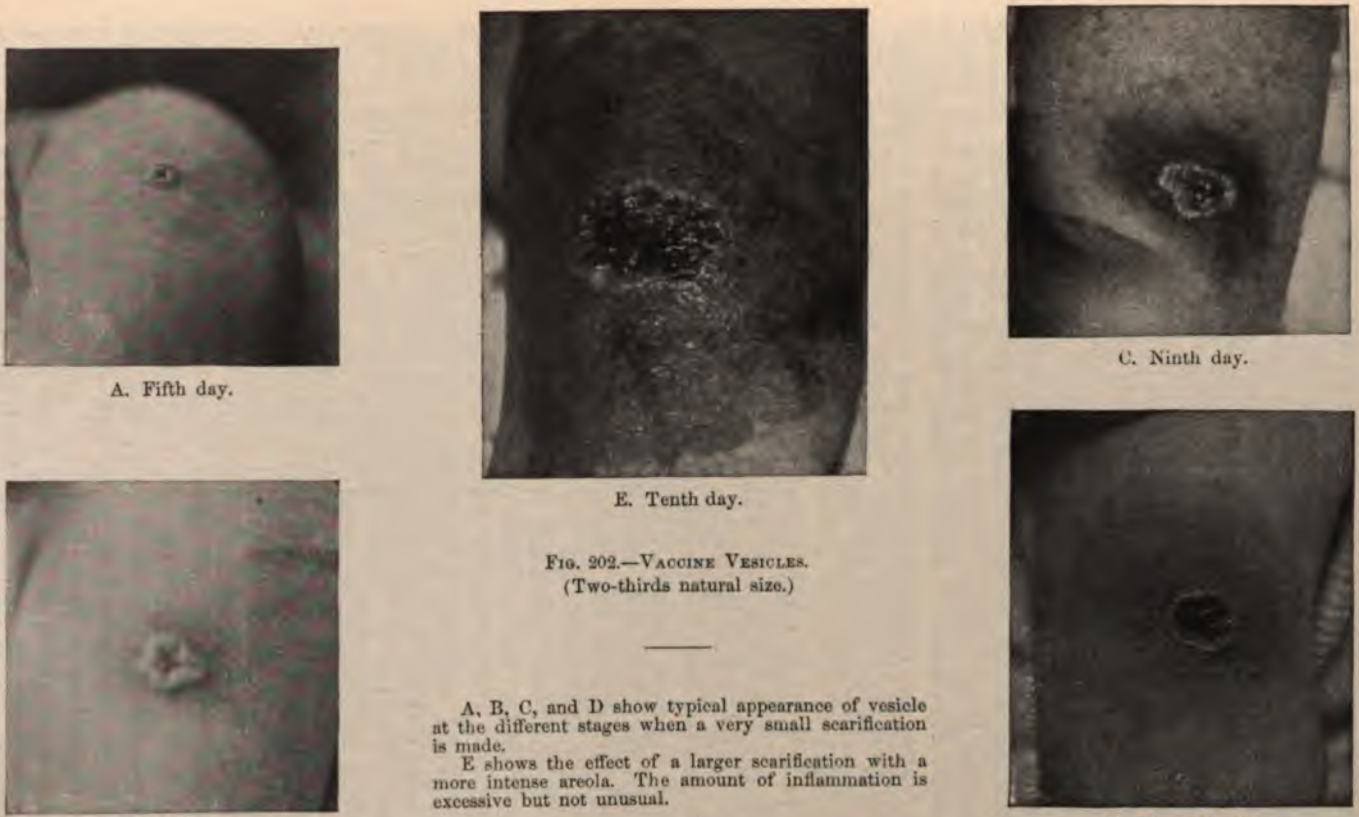
* Reliable glycerinated lymph is prepared by the New York Health Department, Mulford & Co., and Parke, Davis & Co. For an excellent paper on Clinical Aspects of Vaccination, see Fielder, Medical News, March 30, 1901. On Vaccination Infections, see Kubin, Medical Record, April 6, 1901.

Time for Vaccinating.—In selecting a time for vaccination, the child's age and general health must be taken into consideration. It is pretty well established that the constitutional disturbance is much less in infancy than in later childhood, and less in very young infants (under one month) than in those of five or six months. A good rule for general practice is to vaccinate every healthy infant as soon as its nutrition is established, this being in most cases during the first three months of life. In delicate infants or in those whose nutrition is a matter of great difficulty, those who are syphilitic, those suffering from eczema or any other form of active skin disease, vaccination should be deferred until the child is in good condition, unless it is likely to be exposed to smallpox. As a rule, vaccination should be avoided during dentition.

Methods of Vaccinating.—In my experience it is better to vaccinate in one place rather than to make two or three inoculations. If more than one is made they should be at least an inch apart. Either the leg or the arm may be chosen; in young infants it is usually easier to protect the vaccine sore upon the leg than upon the arm; in children old enough to run about, the arm is to be preferred, as being more easily kept at rest. The point selected for inoculation should be either the outer aspect of the left calf, about the junction of the middle with the upper third of the leg, or, if the arm is chosen, the insertion of the left deltoid. The skin should be washed with soap and water, dried, and then washed with alcohol.

The New York Health Department supplies with each tube of lymph, a needle, a bit of rubber tubing, and a tooth-pick with one flat end. The needle should be sterilised in an alcohol flame, and three or four small scratches made not more than one-eighth of an inch long, just deeply enough to draw blood. The ends of the capillary tubes are broken off, one end inserted in the rubber tube, and the lymph blown out of the tube upon the broad end of the tooth-pick, then applied to the scratched surface and rubbed in for a full minute. The wound should not be covered until dry; this usually requires from fifteen to twenty minutes. It may then be covered with a sterilised bandage, or isinglass plaster moistened in boiled water. If thoroughly dried no dressing is necessary. The limb should not be washed for twenty-four hours.

The Normal Course of Vaccinia.—The course of a proper vaccination-pock is quite uniform, and one which does not follow this course should not be considered protective. The wound heals and nothing is noticed until the third or fourth day, when a red papule makes its appearance.



A. Fifth day.

B. Seventh day.

E. Tenth day.

C. Ninth day.

D. Eleventh day.

FIG. 202.—VACCINE VESICLES.
(Two-thirds natural size.)

A, B, C, and D show typical appearance of vesicle at the different stages when a very small scarification is made.

E shows the effect of a larger scarification with a more intense areola. The amount of inflammation is excessive but not unusual.

Usually in twenty-four hours more a small vesicle appears which enlarges until the sixth or seventh day, reaching its full development about the ninth day. Its shape and size depend somewhat upon the inoculation (Fig. 202). The vesicle is usually from one-fourth to one-half inch in diameter; it is of a pearly-gray colour and has a depressed centre. During the next two days an areola forms about the vesicle extending from it a variable distance, usually one or two inches into the healthy skin. Its size depends upon the intensity of the infection. This areola is normally of a bright red colour and accompanied by some induration. It is generally at its height on the ninth or tenth day. The vesicle usually dries down to a firm, dark crust which remains from one to three weeks and falls off, leaving a bluish scar which fades to white, becoming somewhat honey-combed. When the process is at its height some constitutional disturbance is usually present; there may be loss of appetite, fretfulness, and general indisposition, and the temperature is usually elevated from one to three degrees. The lymph nodes in the groin or axilla may be tender and swollen. These symptoms generally last for three or four days.

If in a young infant the first inoculation is unsuccessful, at least three trials should be made with good virus, and in the event of further failure, after a year vaccination should be repeated. A failure to inoculate does not mean insusceptibility to smallpox, as is often popularly believed, but most frequently arises from the fact that the virus is inert. I have known one case in which the seventh, and another in which the thirteenth, inoculation was successful after previous failures; occasionally there are seen children who can not be inoculated at all.

Constitutional symptoms, as previously stated, may be absent in very young infants; but in others there is quite constantly present a fever which runs a fairly regular course. It usually begins on the fourth or fifth day, is remittent in type, and rises gradually, reaching its highest point with the full development of the vesicle. At this time it varies from 101° to 104° F., falling gradually to normal. The duration of the fever in cases running the usual course is four or five days. Accompanying the fever there may be anorexia, restlessness, loss of sleep, slight indigestion, and other symptoms of a general indisposition.

Both the local and the general symptoms are sometimes more severe. This may depend upon the susceptibility of the child, the lymph being pure and the vaccination properly done. The original vesicle may be much larger than usual, and small secondary vesicles may form in the neighbourhood (Fig. 202). In very rare instances a generalized eruption of true vaccine vesicles occurs with marked fever and other general symptoms of corresponding severity. Single vesicles may be produced on distant parts of the body as a result of auto-inoculation, usually by scratching. Where eczema of the face is present, inoculation is not infrequently carried thither. Most of the very sore arms and legs, however, are due to infection from pyogenic bacteria contained in the lymph, or to their accidental introduction at the time of vaccination or subsequently. In the milder cases, the swelling and other evidences of local inflammation are more marked than in a normal vaccination; a drop or two of pus forms beneath the scab, and when the latter comes away an excavation is left which heals in two or three weeks. Or, the inflammation may extend more deeply into the connective tissue, to be followed by more extensive suppuration or sloughing, leaving an ugly ulcer an inch or more in diameter which slowly fills by granulation in from five to eight weeks. Sometimes the period of incubation is unduly prolonged, so that the vesicle does not form until the twelfth or fourteenth day, although its subsequent course may be normal. In other cases, the incubation is shorter than usual, and the vesicle may appear as early as the third or fourth day.

Much has been written about the so-called "raspberry excrescence" which not very infrequently takes the place of a proper vesicle. It is of a dark-red colour, elevated, smooth or slightly granular, not sensitive, having no areola and no constitutional symptoms. It generally persists for two or three weeks, and slowly disappears, leaving no scar. It is usually the result of virus of feeble activity, and if it gives any protection it is very slight. Such cases should always be re-vaccinated, and in my experience re-vaccination is usually successful.

Complications and Sequelæ.—Post-vaccine eruptions are many and of great variety. The most frequent is a general roseola, sometimes resembling scarlet fever, but much oftener measles, and usually occurring at the height of the local process. Other eruptions seen are urticaria, various forms of erythema, and, rarely, purpura. Other complications are chiefly from mixed infection. Syphilis and tuberculosis are practically excluded by modern methods of procuring the lymph. Tetanus can result only from carelessness or neglect of suitable precautions in preparing the lymph; proper legal restrictions regarding its production should in the future make this impossible. The most common form of local infection is cellulitis, which may terminate in suppuration or sloughing at the site of vaccination, and sometimes may cause suppuration of the neighbouring lymph nodes. In rare cases, general septicæmia or pyæmia may follow. Impetigo contagiosa sometimes occurs. Erysipelas may develop at any time before the vaccine sore is entirely healed; I saw it once as late as the sixth week. Pneumonia and nephritis may be associated with any of the more serious complications. Latent tuberculosis may become active after vaccinia, and a child who is subject to eczema is liable to a recurrence. In a delicate child a condition of malnutrition is often intensified if the vaccinia is at all severe.

The mortality of vaccination is stated by Voigt, from careful statisties drawn from German sources, to have been 35 in 2,275,000 cases, including both primary and secondary vaccinations. Of the deaths, 15 were due to erysipelas, 8 to gangrene, 5 to cellulitis, 3 to "blood poisoning," and 4 to other causes. The occurrence of tetanus after vaccinia has already been mentioned. With proper precautions in preparing lymph it will not occur. In fact, nearly all the deaths are from causes which are preventable.

Treatment.—The whole purpose of treatment is to prevent infection. The first essentials are a clean limb, pure virus, and a clean needle; the next, to allow thorough drying of the wound before the clothing touches it. After this nothing is necessary until the vesicle forms. Then the important thing is to prevent scratching and the irritation of the clothing. All vaccine shields are objectionable. For an infant nothing is better than the sterilized bandage, which can be kept in place by sewing to the stocking or sleeve of the shirt. Any constriction of the limb is injurious. For older children the simplest dressing is a pad of sterile gauze fastened to the limb by two pieces of adhesive plaster. Should the vesicle rupture and discharge serum, it should be kept clean and dry by dusting daily with boric acid. When the local symptoms are at all severe the limb should be kept at rest. An infected vaccination wound, like any other infected wound, requires careful surgical treatment; disastrous results often follow the use of poultices and other applications much in vogue in domestic practice.

CHAPTER VI.

PERTUSSIS.

Synonym: Whooping-cough.

PERTUSSIS is a contagious disease which prevails epidemically and in most large cities endemically. Although it may affect persons of any age, it is generally seen in young children, and as a rule it occurs but once in the same individual. While in later childhood pertussis may be ranked as one of the milder infectious diseases, in infancy it is one of the most fatal. Its principal complications are broncho-pneumonia and convulsions. Pertussis is characterized by catarrhal and nervous symptoms. The catarrh affects the mucous membrane of the respiratory tract, and is probably due to a specific form of infection. It is accompanied by a hyperæsthetic condition of this mucous membrane. The most prominent nervous manifestation is a peculiar spasmodic cough which occurs in paroxysms, and from which the disease takes its name. The cough is no doubt of reflex origin, from an irritation which has been located by different writers in various parts of the respiratory tract. In addition to these conditions, there is present in pertussis a marked irritability of the nervous system, which in infancy often shows itself by convulsions.

Etiology.—Everything that is known of pertussis suggests a microorganism as its cause. Present evidence, moreover, points strongly to a bacillus, first described by Eppendorf, afterward more fully by Jochmann and Krause. An important recent contribution to this subject has been made by Dr. Martha Wollstein from the laboratory of the Babies' Hospital. She not only confirmed previous observations as to the constant presence of this organism from a study of thirty cases of pertussis, but obtained characteristic agglutination reactions with the blood of children suffering from the disease. The bacillus belongs to the influenza group, and in many points resembles Pfeiffer's bacillus.*

Proximity to a patient is all that is required to communicate the disease, and even close proximity is not necessary. There seems to be no doubt that the disease may be contracted in the open air.

Predisposition.—Fully one half the cases of pertussis occur during the first two years of life. The following are the statistics of Szabo (Buda-Pesth), showing the ages at which the disease was met with in 4,591 cases, comprising the records of one clinic for thirty-four years:

Under one year	1,028 cases.	Three to four years	904 cases.
One to two years	1,008 "	Four to seven years	803 "
Two to three years	659 "	Over seven years	189 "

Pertussis thus shows a stronger tendency to affect young infants than does any other contagious disease. A number of cases are on record in which it has occurred during the first month, and one has recently come to my notice where a child twelve days old was attacked, whose mother was suffering from the disease. The disease is nearly twice as frequent in the winter and spring as in the summer and autumn. Epidemics of pertussis often occur at the same time with or follow those of measles.

The susceptibility to pertussis is very great, and is equalled only by that to measles. Biedert reports that of 401 children exposed during an epidemic in a certain village, 366, or ninety-one per cent, took the disease.

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* The bacillus is found in the mucus expelled after the typical paroxysm. This should be received in a sterile dish and washed several times in sterile or peptone water. Examined in smears, the organism appears as a short, plump, ovoid bacillus lying singly or in clumps between the pus and epithelial cells. It decolourizes when stained by Gram's method. It grows best on blood-agar plates. It is non-motile. According to Wollstein the bacillus agglutinates with the blood of pertussis patients in dilutions as high as 1–200, and occasionally 1–500. This reaction was not found until the third week of the attack and was present as late as three months. The bacilli were present in greatest numbers after the cough had continued for about two weeks, but were very numerous throughout the paroxysmal stage, being found as late as the eighth week of the disease.

Infective period.—Pertussis may be communicated
ginning of the catarrhal stage; it is more contagious
later. There seems little doubt that it is contagio
spasmodic stage and possibly longer. Quarantine is
for two months, and in many cases for a longer time.
of the contagion is the patient, rarely the room or th
pertussis may be carried by a third person, this is ve
one has been in very close contact with the patient,
without change of clothing to another child.

Incubation.—The very gradual onset of pertussis
sible in the majority of cases to fix the exact date, and
the definite duration of the period of incubation. I
could best be determined it has usually been from
days, or about the same as in measles. If, after an
days pass without the development of a cough, the pr
strong that the disease has not been contracted.

Lesions.—The only constant lesion of pertussis con
inflammation of varying intensity, which affects the
of the larynx, trachea, and bronchi, and sometimes th
pharynx. If the child dies during a paroxysm, eithe
convulsions, the brain is found intensely congested an
of punctate hæmorrhages, or even larger extravasa
always show emphysema if the attack has been sev
The other pulmonary lesions are due to complicatio
quent of which is broncho-pneumonia. Catarrhal e
are not infrequent.

Symptoms.—The symptoms of pertussis are usually
stages—the catarrhal, the spasmodic, and the stage of

The catarrhal stage continues on the average for a
though cases show considerable variation on this poi
whoop almost from the very beginning of the disease,
cough for three or four weeks before a typical whoo
symptoms in the beginning are indistinguishable fro
nary attack of subacute tracheo-bronchitis, and unless
exposure to pertussis no suspicion is excited. After fiv
ever, the cough, instead of abating as in an ordinary
creases in severity and occurs in paroxysms. At fir
and there are only two or three a day, but they gra
frequency and severity until the typical whoop is h
the beginning of the spasmodic stage. During the first
symptoms of a mild grade of catarrhal inflammation of
and larynx, and often there is a slight elevation of te

The spasmodic stage.—In a typical paroxysm of a
child, who can usually foretell it, will often run for su

the mother or the nurse, or seize a chair with both hands. There now occurs a series of explosive coughs, from ten to twenty in number, coming in such rapid succession that the child can not get its breath between them; the face becomes of a deep red or purple colour, sometimes almost black; the veins of the face and scalp stand out prominently; the eyes are suffused, and seem almost to start from their sockets; there follows a long-drawn inspiration through the narrowed glottis, producing the crowing sound known as the whoop; and then another succession of rapid coughs follows and another whoop. In a single severe paroxysm, which lasts two or three minutes, the child may whoop half a dozen times; with the final paroxysm a mass of tenacious mucus is usually brought up. In a young child vomiting is almost certain to follow, if food has been recently taken. Epistaxis sometimes occurs with nearly every severe paroxysm, but in most cases the bleeding is slight. After a severe attack the child is at times so exhausted as to be hardly able to stand; there is profuse perspiration; his mind is confused, and he may be completely dazed. In infants the attack may result in a degree of asphyxia requiring artificial respiration. Those old enough to describe their sensations tell of a sense of impending suffocation, the suffering from which is almost indescribable.

The number of severe paroxysms or "kinks" in twenty-four hours varies, according to the severity of the case, from half a dozen to forty or fifty. There are always many more of a milder form. Paroxysms are often excited by eating or drinking anything cold, by a draught of air, or by imitation; they are usually more frequent during the night than the day, and in a close room than in the open air.

In less severe cases no paroxysms of the grade above described may occur, and no typical whoop may be heard throughout the attack; but the paroxysmal nature of the cough which continues until the plug of mucus is expelled, the watery eyes, and the vomiting which follows a paroxysm, stamp the disease as pertussis. In young infants the whoop is frequently not marked. The child sometimes coughs until it is asphyxiated, and yet no whoop occurs. The paroxysms are also modified by intercurrent disease, especially by attacks of pneumonia or severe bronchitis. At such times they usually become less frequent and less typical, and may be absent for several days, returning as the complication subsides.

The seat of the irritation which produces the cough has been variously located by different observers: some have thought it to be in the nose, others in the trachea, the bronchi, or the larynx. It is very probable that it may not always be in the same place and that the infectious catarrh, which is really the most important element in the disease, may vary in its intensity and location in different cases. The weight of evidence seems to be that in the great majority of cases the source of irritation is in the larynx or trachea. From laryngoscopic examinations made during the disease, Von Herff found the mucous membrane of the larynx to be swollen and congested, and occasionally the seat of small hæmorrhages or superficial ulcers. He states that the frequency and severity of the paroxysms corresponded with the degree of laryngitis, and he found that a paroxysm could always be excited by irritating the mucous membrane between the arytenoid cartilages. During a paroxysm he observed that there was a collection of mucus on the posterior laryngeal wall, the removal of which had the effect of shortening the paroxysm.

Rossbach made laryngoscopic examinations, with negative results so far as the larynx was concerned, but he states that a plug of mucus could always be seen in the lower trachea for one or two minutes before the paroxysm occurred. There is little doubt that this collection of mucus is the exciting cause of the paroxysm, as it is a familiar clinical fact that the paroxysm always continues until this is dislodged.

The average duration of the spasmodic stage is about one month. It increases in intensity for the first two weeks, remains stationary for about a week, and then gradually diminishes in severity. The course and duration of this stage are, however, subject to wide variations. In mild cases it may last only a week; in severe cases, especially in the winter season, it may continue for three months, at times almost subsiding, but lighting up again with all its previous severity with every fresh attack of cold. After it has entirely ceased the whoop may return with an attack of bronchitis, and continue for a month or more. This is not to be regarded as a true relapse of pertussis. The habit of the paroxysmal cough once established, it tends to recur with every slight bronchitis, often for months afterward.

The stage of decline.—Gradually the severity of the paroxysms abates, the whoop ceases, and the cough resembles more and more that of ordinary bronchitis. This stage usually continues about three weeks, but may be prolonged indefinitely in the winter months.

Complications.—*Hæmorrhages.*—The hæmorrhages of pertussis are mechanical, and depend upon the intense venous congestion which accompanies the paroxysm. Epistaxis is the most frequent variety, and occurs in a considerable proportion of the severe cases, in a few with almost every severe paroxysm, but it is rarely severe enough to require local treatment. Hæmorrhages from the mouth may have their origin either in the pharynx or the bronchi, the blood being brought up by the cough; such hæmorrhages are usually small. Conjunctival hæmorrhages are less frequent, and are usually slight, although I have seen the entire conjunctiva covered. In a case under my observation there was bleeding from both ears with every severe paroxysm, for more than a week. This child had previously suffered from scarlatinal otitis, with perforation of the drum membrane. Small extravasations into the cellular tissue beneath the eyes are occasionally seen, giving an appearance somewhat like an ordinary "black eye." Intracranial hæmorrhages are not frequent, but many examples have been recorded, and they may be severe enough to produce death. They are usually meningeal, very rarely cerebral; according to their extent and location they may produce hemiplegia, monoplegia, aphasia, facial paralysis, or disturbances of sight, hearing, or sensation; in addition, there may be convulsions or rigidity, but rarely complete coma. The extravasations are usually small, and the symptoms which they produce disappear at the end of a few weeks. Fatal cases with autopsies have been reported by Cazin, Marshall, and others. In almost every instance these hæmorrhages have occurred as a direct result of the severe paroxysms. Purpura hæmorrhagica as a sequel of pertussis was twice seen at the New York Infant Asylum.

Respiratory system.—The most serious complications of pertussis are connected with the lungs. By far the largest proportion of deaths is due to pulmonary complications, usually broncho-pneumonia. This is more frequent in winter and spring than in the summer months, and is especially to be dreaded during infancy. In later childhood lobar pneumonia is occasionally seen. Pneumonia rarely begins before the second week of the disease, and most frequently develops at the height or toward the close of the spasmodic stage. The physical signs present no peculiarities; the cough changes somewhat in character during the pneumonia, and the whoop may not be heard. The prognosis of the pneumonia is bad, because of the debilitated condition of the children at the time of its occurrence. A great danger is from the supervention of convulsions, this being a frequent mode of termination. As there is always considerable emphysema the rapidity of breathing is frequently out of proportion to the temperature, which often is only moderately elevated. If the child escapes the dangers of the acute stage, death may still occur from exhaustion, owing to the protracted course which the disease frequently runs (see page 551).

Bronchitis of the large tubes is present in almost all the severe cases, and is not of itself serious. Bronchitis of the small tubes has the same dangers and the same complications as broncho-pneumonia.

Vesicular emphysema has been present, I think, in every case which I have seen upon the post-mortem table; a certain amount of it, no doubt, occurs in every severe case. It is produced by the forcible cough of the paroxysm. In very severe cases interstitial emphysema is also found. Northrup has reported a remarkable instance of this complication. Rupture of the air-blebs which form on the surface of the lung may lead to emphysema of the cellular tissue of the mediastinum, and the air may find its way along the great vessels into the neck, and finally into the subcutaneous cellular tissue of the entire body. Cases of general subcutaneous emphysema have been reported by Croker and by Hodge, both of which ended fatally, one in three and one in eight days from the beginning of the emphysema. In the great majority of the cases vesicular emphysema is not permanent.

Digestive system.—During the summer, infants with pertussis are almost certain to suffer from diarrhœa; it may be only an occasional symptom, or the attack may be severe and prolonged, resulting in the development of ileo-colitis. The intestinal complications may be almost as serious in summer as are those of the respiratory tract in winter. Vomiting is even more frequent than diarrhœa, and while it may be distressing at any age, it is especially so in infancy. So frequently does the taking of food excite vomiting, that the nutrition of these patients often becomes a matter of the greatest difficulty, and in fact the most serious problem in the management of a case. Malnutrition and even marasmus may follow, or the general resistance of the child may become so reduced by lack of food that it falls a ready prey to pneumonia.

Nervous system.—There may be convulsions, coma, paralysis, aphasia, disturbances of sight or hearing, and in rare cases even of the mental condition. The most serious of these complications are convulsions. They are much more frequent in infancy than later, and particularly in those who are rachitic, in whom they are often fatal. Convulsions are of course more common in severe attacks, but they may occur suddenly where there has previously been no cause for anxiety. They are especially to be dreaded if pneumonia is present. The attack of convulsions may be the culmination of the extreme degree of nervous irritability which accompanies the paroxysm, it may be due to asphyxia, or to an intracranial lesion; if the latter, there is usually meningeal hæmorrhage. This is to be suspected if there are continued convulsions for several hours, with general rigidity or hemiplegia.

Disturbances of sight are not infrequent in severe cases; usually these are transient, but there may be blindness lasting two or three days or even weeks. The transient symptoms depend most likely upon circulatory changes that occur in the brain during the paroxysm, while those which last for two or three weeks are probably due to meningeal hæmorrhage. Disturbances of hearing are rare. The different forms of paralysis occurring with pertussis may likewise be transient or permanent. They are to be explained in the same way as the disturbances of the special senses. The most common form is hemiplegia.

Albuminuria is not infrequent, being found in 66 of 86 examinations by Knight. The quantity of albumin is rarely large, and it may be accompanied by a few hyaline casts. Both are probably the result of circulatory disturbances in the kidney. Other complications of pertussis are hernia, prolapsus ani, and ulcer of the frenum linguæ.

Diagnosis.—The only constant features of pertussis are the course of the disease and its communicability. In many cases the typical whoop is never heard. There are no symptoms by which a positive diagnosis can be made in the catarrhal stage; but a cough not accompanied by fever or physical signs, which steadily increases in severity for two weeks, in spite of treatment, and which occurs chiefly at night, is always suspicious. When, in addition, the cough begins to come in paroxysms, accompanied by suffusion of the face and occasionally by vomiting, there can be little doubt even though no whoop is heard. If the disease is prevalent the diagnosis is practically certain. Mild cases which do not go even as far as the symptoms mentioned are most puzzling. But if there is a history of exposure, if the cough continues from four to six weeks, little influenced by treatment, and if other cases follow, the disease must be pertussis. Without evidence of communicability, however, one may be in doubt even after the disease is over. In early infancy any cough may have more or less of a spasmodic character, and a fairly typical whoop is often heard in the course of an ordinary bronchitis. I have several times seen abortive or very short attacks in one member of a family of children, the others having the disease in a typical form. Occurring by themselves such cases cannot be recognised.

Irritation of the pneumogastric or recurrent laryngeal nerve from enlarged tracheal or bronchial lymph nodes, whether of a simple or tuberculous character, may give rise to a spasmodic cough, which in certain cases may be indistinguishable from pertussis. The prolonged duration of these cases is sometimes the only diagnostic point; but the paroxysms are usually not so severe as in true pertussis, and the course is generally less typical.

The presence of leucocytosis may be an aid to diagnosis in some doubtful cases.*

Prognosis.—The most important factor in the prognosis of the disease is the age of the patient. After the fourth year it is indeed rare that either a fatal result or serious complications are seen; but during infancy, and particularly during the first year, there are few diseases more to be dreaded. This is especially true on account of the connection of whooping-cough with the three most fatal conditions of infantile life—broncho-pneumonia, diarrhœal diseases, and convulsions. Fully two thirds of the deaths from whooping-cough occur during the first year of life. The prognosis is very much worse in infants under three months than in those who are older and consequently have more resistance. It is better in the summer than in the winter, because broncho-pneumonia is then less frequent. It is particularly bad in delicate infants, in those who are rachitic, in those who are prone to attacks of bronchitis, in those who have suffered previously from pneumonia, and in those with a strong tendency to tuberculosis.

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* Frohlich and Meunier first called attention to the leucocytosis accompanying pertussis, far exceeding that of any other afebrile disease of the respiratory tract. It appears in the early part of the convulsive stage, and disappears slowly with improvement. The count is usually between 15,000 and 25,000, although 51,000 has been recorded. The differential count shows an increase in the lymphocytes at the expense of the neutrophiles. The leucocytosis is little influenced by complications, and even during broncho-pneumonia the lymphocytes may continue to be in excess.

The exact mortality of whooping-cough it is difficult to state in figures. During the first year of life it is probably not far from twenty-five per cent, although it diminishes rapidly after this time. In foundling asylums and hospitals for infants it is to be ranked among the most fatal diseases, and in some epidemics the mortality in such institutions is as high as fifty per cent.

Fully two thirds of the deaths during whooping-cough are from broncho-pneumonia; the next most frequent cause is diarrhœal diseases. Convulsions may be the mode of death in either of the above conditions, or may occur apart from them. During the first year, death often results from marasmus, the child having been reduced by the prolonged disease. Occasionally death is due to asphyxia following a severe paroxysm, to intracranial hæmorrhage, or to general emphysema.

As a predisposing cause of tuberculosis, pertussis is second only to measles. In both diseases tuberculosis develops in much the same way and from practically the same causes.

Prophylaxis.—Pertussis is a contagious disease, and a child suffering from it should be isolated from other children whenever this is possible. Children with pertussis should never be allowed to attend school, and needless exposure should always be avoided.

Young infants, delicate children, and those with a predisposition to tuberculosis, should be most carefully protected against exposure, since it is in them chiefly that the disease is likely to be serious. As it is from the patient that the disease is nearly always contracted, there does not exist the same necessity for the fumigation and disinfection of apartments as after other contagious diseases. In institutions, however, this should always be practised, and in private houses if the room is subsequently to be occupied by an infant.

It is as undesirable as it is impossible to confine a child with pertussis to a single room during the attack; all those persons for whom exposure would be dangerous should therefore be sent away from the house. Quarantine should continue for at least six weeks, or until the spasmodic stage is over.

Treatment.—We have as yet no specific remedy for pertussis. The important thing in most cases is the hygiene or general management of the case; fully half of the cases seen in practice require nothing more. Much harm is done by indiscriminate drugging.

General measures.—Fresh air is important throughout the attack. It is almost invariable that the paroxysms are fewer while patients are out of doors, and more frequent when they are in close rooms. Older children with pertussis may go out even in winter except on stormy, raw, or windy days. With infants and delicate children, the outdoor treatment in cold weather so enthusiastically advocated by some writers should be used with the greatest caution. It should certainly not be permitted if the patient has even the slightest amount of bronchitis. My own experience is that during the winter in a climate like that of New York or New England, the class of patients just referred to are better off indoors, taking their airing, if at all, in their rooms. In warm weather or in a mild climate all children should be kept in the open air as much as possible.

A change of climate is desirable when the cough is unduly prolonged, also for delicate children in winter. A warm place at the seashore is one which is most likely to be beneficial. The improvement following a sea voyage is often very marked, surpassing even a residence at the seashore.

The rooms occupied by children suffering from pertussis should be frequently changed, thoroughly aired, and occasionally fumigated. The daily use in the room of one of the small formalin lamps is of decided benefit. A change of rooms, clothing, bedding, etc., sometimes exerts a marked influence on the course of very prolonged attacks, the inference being that continued re-infection takes place. Such a change should be made twice a week, and it is of special importance in hospitals, where many children quarantined in a ward seem to cough interminably.

Careful feeding and attention to the bowels are matters of the greatest importance; with infants particularly, chronic indigestion and abdominal distention have a very marked effect in increasing the frequency of the paroxysms. Feeding is difficult since vomiting occurs so easily. In most cases it is necessary to repeat the meal in a short time, if the first one has been vomited. Children over two years old should in all such cases be kept upon a fluid diet, chiefly of milk. For infants, milk should be diluted, and in many instances it should also be partially peptonized. Any medication which causes disturbance of the stomach should be omitted. In severe cases the child's strength should be kept up by the judicious use of alcoholic stimulants.

Local treatment.—This may be effected by insufflations of powder into the nose, by local applications to the larynx, or by inhalations.

The first two methods have been advocated, in the belief that the cough is due to an infectious catarrh having its seat in the nose or larynx. For insufflation, quinine or benzoic acid is preferred, mixed with some finely divided, inert powder, such as bicarbonate of sodium, talcum, or coffee; these are used with the powder insufflator once or twice daily. Local applications to the larynx may be made by means of a spray or swab. Resorcin and carbolic acid, each in a one-per-cent solution, are most used. These applications are made once or twice daily. I have never seen from any of the above methods the beneficial results claimed, and I believe them to have been exaggerated. The application of cocaine to the larynx should never be employed in young children on account of the danger of poisoning.

Inhalations are of much more value. They are useful to modify the catarrh by allaying irritation, facilitating the expulsion of the mucus, and possibly as antiseptics. Those most employed are carbolic acid, creosote, and cresolene. In my experience creosote is the best. These substances may be used upon cotton in a respirator, or vapourized over an alcohol lamp (page 60). The possibility of absorption should not be forgotten, and the urine should be watched. Where the paroxysms are frequent and of great severity, chloroform may be used to ward off convulsions or prevent dangerous asphyxia. In such conditions O'Dwyer used intubation with striking benefit. The tube entirely overcomes the glottic spasm which is the chief cause of suffering and danger. O'Dwyer's plan was to have the tube worn constantly until the severity of the disease had passed. With the rubber tubes now in use the difficulty in getting rid of the tube subsequently is not great.

Internal medication.—Of the innumerable drugs which have been recommended for this disease, four possess undoubted advantages over all others—viz., quinine, belladonna, bromoform, and antipyrine. Quinine should not be used for infants and seldom for young children on account of its tendency to upset the stomach. For older children full doses are required to be of much benefit—i. e., twelve to fifteen grains daily to a child of five years. In giving belladonna it is important to begin with a small dose and gradually increase both its frequency and size until the physiological effects of the drug are produced. To an infant two years old, one fourth of a minim of the fluid extract may be given every four hours as an initial dose, gradually increasing to every two hours; if atropine is used, gr. $\frac{1}{800}$ may be given in the same way. Although belladonna usually has a decided influence in reducing both the frequency and the severity of the paroxysms, it causes many unpleasant symptoms, and its effects must be closely watched.

Bromoform has considerable value, but it is by no means a specific. A convenient method of administration is to drop it upon sugar. When prescribed in emulsions or mixtures these should be carefully shaken before each dose, or the patient may be poisoned by getting the greater part of the drug in the last few doses. The dose at two years is from one to three drops, at five years two to four drops from three to five times a day. In full doses it must be used with caution.

Antipyrine has been in my experience more generally useful than any other single drug. It may be given with safety, even to young infants, in considerably larger doses than are ordinarily employed. For a child six months old the initial dose may be one grain every three hours; later this may be given every two hours. For a child two years old the initial dose may be two grains repeated every four to six hours, gradually increasing up to two grains every two hours. Should pneumonia develop, the antipyrine should be discontinued.

Nearly all drugs which allay nervous irritability have a certain amount of effect in controlling the paroxysms of pertussis; codeine, chloral, and trional are useful where the night attacks are so severe as to prevent sleep. A combination of the bromide of sodium with antipyrine is often better than the latter given alone. Heroin, although in use but a short time, promises to be a valuable addition to our therapeutics. I do not believe that any form of internal medication or local treatment shortens pertussis; but, inasmuch as the disease is self-limited, great benefit to the patient results from the reduction of the number and the diminution of the severity of the paroxysms.

In establishing the value of any method of treatment, it should be remembered that the number of cases in which the disease is considerably shorter than the average is large, and also that almost any method of treatment if employed after the attack has reached its height will be thought beneficial, as the natural tendency is then to improve. The value of any particular line of treatment is to be judged in a given case only by its effect in reducing the number and severity of the paroxysms. This ought to be evident in the case of drugs within two or three days, and can only be determined by keeping a careful record of the number of severe paroxysms day and night. No drug succeeds equally well in all cases.

In a mild case, where the number of paroxysms does not exceed eight or ten during the day, where there is no vomiting and the general health is not affected, it is not usually advisable to continue the administration of any drugs throughout the disease. A single dose of antipyrine or codeine at night may be all that is necessary. All cases in infants must be watched with great care and the parents warned of the possible dangers which may supervene suddenly, even in the course of mild attacks. For severe cases antipyrine should be given to diminish the frequency and the severity of the paroxysms, and inhalations of creosote used if much catarrh is present. All the fresh air possible should be allowed. For older children the same plan of treatment may be followed, or quinine or belladonna may be substituted for the antipyrine.

As these drugs are given solely for the purpose of diminishing the frequency and severity of the paroxysms, their continuous use should be deferred until the symptoms are sufficiently severe to greatly disturb the child, the benefit at this period being more striking than if they are begun early and used continuously.

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* Manchester Chronicle, 1885.

Incubation.—In forty-eight collected cases in which the incubation was definitely determined, it varied between three and twenty-five days. It was less than fourteen days in only four cases, and in twenty-six of the forty-eight cases it was between seventeen and twenty days. In three cases of my own in which it could be definitely fixed, the incubation was nineteen days in one case and twenty days in two cases. The average period of incubation, then, may be stated to be from seventeen to twenty days.

Symptoms.—In the milder cases the local symptoms are the first to attract attention; in those which are more severe there are frequently prodromal symptoms of from twelve to forty-eight hours' duration—anorexia, headache, vomiting, pains in the back and limbs, and fever. Soltmann has reported a case ushered in by convulsions. The initial temperature in a mild attack is 100° to 101° F.; in a severe one, from 102° to 104° F.

Of the local symptoms, the pain usually precedes the swelling; it is increased by movement of the jaws, by pressure, and sometimes by the presence of acid substances in the mouth. It is usually referred to the posterior part of the jaw just below the ear. The swelling may begin simultaneously in both parotids, but more frequently one side is involved a day or two in advance of the other. It usually reaches its maximum on the third day, often on the second, remains stationary for two or three days, and then subsides gradually. The degree of swelling varies with the severity of the attack. When it is marked, the patient may be so changed in appearance as scarcely to be recognisable; it fills the lateral region of the neck between the jaw and the sterno-mastoid muscle and extends forward upon the face to the zygomatic arch, so that the centre of the tumour is usually the lobe of the ear. The other salivary glands may swell simultaneously with the parotids, or several days later, even after the parotid tumour has disappeared. Occasionally swelling of the submaxillary or the sublingual glands occurs before that of the parotid, and in rare instances these may be the only glands affected.

As a rule, the parotid of both sides is involved.. Of 282 cases both sides were affected in 215. When one side alone is involved, it is the left a little more frequently than the right. The interval between the swelling of the two sides may be a week, or even five or six weeks, but usually it is only two or three days.

The salivary secretion is usually very much diminished, and the dry mouth causes great discomfort. An exceptional instance has been reported by Simon, in which a distressing salivation occurred, the secretion amounting to six or eight ounces daily.

Although as a rule the patient is not seriously ill, mumps may in rare cases produce most alarming and even dangerous symptoms. The temperature may for several days reach 104° F. or more, deglutition may be extremely difficult, pressure on the jugular veins may lead to venous hyperæmia of the brain, causing headache and sometimes delirium; there is sometimes great prostration and the symptoms of the typhoid condition. These severe attacks are nearly always in children over twelve years old.

The constitutional symptoms of mumps usually last from three to five days; the swelling continues on an average a little less than a week. If the case has been a severe one, slight swelling may continue for two weeks or even longer. Relapses, in which the opposite side from the one first affected is involved, are quite frequent, occurring in about ten per cent of the cases.

Complications and Sequelæ.—In childhood the complications are few and usually unimportant; but in adolescence they are occasionally serious. Orchitis is exceedingly rare in childhood; of 230 cases observed by Rilliet and Barthez, this was seen in but 10, and only 3 of these cases were under fifteen years, and no case under twelve years old. When orchitis occurs it is generally toward the end of the second or the beginning of the third week; it is usually marked by an accession of fever, sometimes by a chill; if severe, nervous symptoms may be present. The body of the testicle and not the epididymis is generally affected. The acute symptoms continue for three or four days, and the entire duration of the attack is about a week; although the testicle is often enlarged for some time afterward, and atrophy of the organ may follow.

In females, congestion and swelling of the breasts, ovaries, or labia majora may occur; and, although these complications are all very rare, most of them have been observed even in young children.

Nephritis has in a few instances followed mumps, sometimes coming on as late as four or five weeks after the attack. Single cases have been reported by Croner, Isham, Henoch, and others. Nervous sequelæ are more frequent, but even these are rare. Jaffrey has reported a case of multiple neuritis with typical symptoms, occurring three weeks after an attack. Facial paralysis three weeks after mumps has been reported by Hillier, apparently due to an extension of inflammation from the gland to the seventh nerve.

Pearce * has collected an interesting series of forty cases of deafness following mumps, in which there was no sign of otitis, the symptoms coming on suddenly with vertigo, a staggering gait, and often with vomiting. In most of the cases the deafness was unilateral and the loss of hearing was permanent. The cause assigned was disease of the auditory nerve, the seat of the trouble being in the labyrinth. Toynbee has reported an instance of hæmorrhage into the labyrinth. Otitis media is rarely seen.

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* Manchester Chronicle, 1885.

Suppuration of the parotid gland occurs in about one per cent of the cases, and is probably due to accidental infection. Gangrene and sloughing of the parotid were observed twice by Demme in 117 cases; both of these proved fatal. Pneumonia, meningitis, endocarditis, and pericarditis have been observed as complications of mumps, although all are extremely rare.

Prognosis.—In the great proportion of cases mumps is a mild disease, and terminates in complete recovery in a few days. In young children complications are infrequent, and those which occur are rarely severe.

Diagnosis.—Mumps is most likely to be confounded with acute swelling of the cervical lymph nodes. In a parotid swelling, the lobe of the ear is near the centre of the tumour, which extends backward to the sterno-mastoid muscle and forward upon the face as far as the zygomatic arch, embracing the angle and ramus of the jaw.

A swollen lymph node is usually entirely below the ear and behind the jaw, not extending upon the face. The tumour is generally smaller and more circumscribed if only a single node is involved, and it comes on much more slowly than does mumps. When only the submaxillary or sublingual glands are affected, the diagnosis from swollen lymph nodes is sometimes impossible except by the course of the disease. Mumps is characterised by the rapidity with which the swelling occurs, and by its relatively short duration.

Treatment.—The disease is self-limited and the individual symptoms rarely distressing, so that in most cases very little treatment is required. If constitutional symptoms are present the patient should be kept in bed, and if there are none he should be confined to the house. The gland should be protected by cotton or spongio-piline, and if the pain is severe heat should be applied or the gland painted with belladonna. The diet should be liquid, on account of the pain produced by mastication. The mouth should be kept clean by the use of some antiseptic mouth-wash. The general symptoms and complications are to be treated according to the indications presented. Cases of mumps occurring in schools or institutions should be quarantined for three weeks, and in private practice where there are susceptible persons. Fumigation and disinfection after an attack are unnecessary.

CHAPTER VIII.

DIPHTHERIA.

UNTIL within the last few years it was customary to class as diphtheria all diseases characterised by the production of a false membrane upon the mucous membranes of the throat or air passages. In the following pages the term *diphtheria* will be limited to those cases in which the Klebs-Loeffler bacillus is present, the others being grouped under the head of false or *pseudo-diphtheria.*

Diphtheria may then be defined as an acute, specific, communicable disease due to the bacillus of Klebs and Loeffler. It is usually characterised by the formation of a false membrane upon certain mucous membranes, especially those of the tonsils, pharynx, nose, or larynx. Like other pathogenic organisms, however, this germ acts with varying intensity, and may cause inflammation of all degrees of severity, from a mild catarrhal angina to the most serious membranous inflammation; but to all alike the term diphtheria should be applied. In its mild form it may be almost without constitutional symptoms; but in its severe form it is attended by great general prostration, cardiac depression, and anæmia, it is frequently complicated by pneumonia and nephritis, and it may be followed by localised or general paralysis; it then constitutes one of the diseases most to be dreaded in childhood.

Etiology.—*The Bacillus Diphtheriæ.*—This was first described by Klebs in 1883, and during the following year it was isolated by Loeffler and shown to be pathogenic. It varies considerably in size and shape even in the same culture. In a specimen it occurs singly or in pairs, sometimes in chains of three or four; the bacilli may lie parallel, but frequently two form an acute or an obtuse angle (Plate XIX, 3, 4, and 5). They are straight or slightly curved, and sometimes branching; they may be swollen or club-shaped at their ends.

Distribution and mode of communication.—In most large cities diphtheria prevails endemically, with periods in which outbreaks of considerable severity are observed. In the country it prevails chiefly as an epidemic. The disease is often introduced into remote districts in some inexplicable manner, and before its nature is recognised a large number of persons may be exposed, and an epidemic results.*

Diphtheria does not arise *de novo.* Every case has its origin in a previous case either directly or remotely. The bacilli may enter the body through the inspired air; they may be taken into the mouth with toys or other articles upon which they have lodged, or by kissing, and sometimes by accidental inoculation. As a rule, the bacilli first gain a foothold upon the mucous membrane of the tonsils, nose, or larynx.

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* The following is an example of the way in which diphtheria may be introduced: In the country branch of the New York Infant Asylum, consisting of a somewhat isolated community of about five hundred persons, chiefly children, there had been no case of diphtheria for several years. The first case was one of membranous laryngitis, rapidly proving fatal in two days. The case was regarded at that time as evidence of the existence of a primary non-diphtheritic membranous croup. In the course of the next few weeks there developed a number of cases of typical diphtheria. On investigation, it was discovered that the nurse who had charge of the child first affected had been a few weeks before in attendance upon a case of diphtheria. During the five years following, cases of diphtheria occurred in the institution every year.

Direct infection is the cause in the great majority of the cases. There is no proof that the bacilli are contained in the breath of a person suffering from the disease. They are present in great numbers in the saliva and mucus from the mouth and nose, often being distributed by sneezing and coughing, and also in pieces of membrane which are discharged; they are not present in the urine or fæces. The most contagious cases are those of pharyngeal diphtheria on account of the amount of discharge which accompanies them. The least contagious are those in which the membrane is limited to the larynx and lower air passages.

Direct infection may occur from persons convalescent from diphtheria, whose throats still contain virulent bacilli, or from persons suffering from a mild form of the disease, which is not recognised as diphtheria. In the latter way it is often spread in schools. It has been repeatedly shown that a person may harbour virulent bacilli in his nose or throat, and may even communicate the disease to others, without himself suffering from diphtheria at any time.

The length of time during which a patient with diphtheria may convey the disease to others is somewhat uncertain. Transmission is possible so long as virulent bacilli remain in the throat; these are frequently found two weeks after the membrane has disappeared and the patient is regarded as entirely well, and in a few cases they are found five or six weeks or longer after recovery.

Indirect infection is not uncommon, and may occur from the bed or clothing of the patient, from the carpet, furniture, wall-paper or hangings of the room, from toys or picture-books, from dishes, feeding bottles, or drinking-cups, from swabs and brushes used for local applications to the throat, from spoons and tongue-depressors, and from surgical instruments with which tracheotomy or intubation has been done. Diphtheria may be carried by a third person, but rarely except by one who has been in close contact with the patient—either the physician or nurse. The frequency of diphtheria in physicians' families bears witness to the great danger of infection in this manner.

Bacilli may retain their virulence for an indefinite period. Both Park and Loeffler found cultures in blood-serum to be virulent after seven months; Roux and Yersin, bacilli in dried membrane to be virulent after twenty weeks; and Abel, upon a child's toy after five months.

Domestic animals may in rare instances be carriers of infection, and in the case of pigeons, at least, they may themselves suffer from the disease. Diphtheria has been repeatedly spread by milk, but very rarely through the contamination of a water supply.

Predisposing causes.—Local conditions in the throat influence very largely the occurrence of diphtheria. An important predisposing cause is the existence of a chronic catarrhal inflammation of the mucous membranes of the nose and throat, so frequently found in children suffering from adenoid growths of the pharynx or from enlarged tonsils. These adenoid growths, the tonsillar crypts, and the cavities of carious teeth, may harbour the bacilli for a considerable time both before and after an attack. The condition of the mucous membranes of the nose and pharynx in other acute infectious diseases furnishes a marked predisposition to diphtheria. This is most striking in the case of measles and scarlet fever; it is seen less frequently in typhoid fever and influenza.

The two sexes are about equally liable to the disease. Children under ten are much more often affected than those who are older, the greatest susceptibility as regards age being between the second and fifth years.

While diphtheria is seen throughout the year, it is more frequent during the cold than the warm months.

The incubation of diphtheria is short. In most of the cases in which it could be definitely traced it has been between two and five days. The virulence of the bacillus varies much in different cases and in different seasons, and while it is frequently true that persons infected from a mild type of the disease have a mild attack, and those infected from a malignant one a severe attack, there is no certainty that such will be the sequence. Dr. W. H. Park informs me that, out of many hundreds tested in the laboratory of the New York Health Department, by far the most virulent bacillus was obtained from the throat of a boy who had what was clinically a very mild form of tonsillar diphtheria.

The immunity conferred by one attack of diphtheria is of comparatively short duration, amounting probably to a few months only. Instances have recently been reported where a second attack occurred within two months of the first, although antitoxin was used.

Lesions.—The essential lesions of diphtheria consist not in the production of a membrane, but, as long ago pointed out by Oertel, and more recently by Babes, Sidney Martin, and others, in certain acute degenerative changes in the cells of the body caused by the diphtheria toxins. These changes are seen particularly in the epithelial cells of the affected mucous membranes, the heart muscle, the kidney, the liver, the central and peripheral nervous system, the spleen, and the lymph glands; the most characteristic being those of the nerves and the liver. There are other lesions which are the result of the action of other organisms, especially the streptococcus pyogenes and the pneumococcus, either alone, together, or in conjunction with the diphtheria bacillus. The most important lesions due to these organisms are broncho-pneumonia and nephritis; but there may be found in the blood, and in many of the organs of the body, the evidences of the invasion of these bacteria—i. e., a streptococcus septicæmia, less frequently a general pneumococcus infection.

Distribution of the diphtheria bacillus in the body.—Unlike many other pathogenic organisms, the diphtheria bacillus is not in most cases widely distributed throughout the body. It is found in great numbers on the surface of the affected mucous membranes and in the false membrane itself, particularly in its superficial portion, but it does not invade deeply the subjacent structures.

The frequency with which the diphtheria bacillus and other organisms are found in the blood and viscera is shown in a series of 209 autopsies studied by Councilman, Mallory, and Pearce, of Boston, in 1901. The following table shows the percentage of cases in which the different bacteria were found by culture:

	Heart's blood.	Liver.	Spleen.	Kidneys.
Diphtheria bacillus...........	6 per cent.	20 per cent.	12 per cent.	19 per cent.
Streptococcus.................	20 "	30 "	27 "	28 "
Staphylococcus aureus........	2·5 "	4 "	3 "	8 "
Pneumococcus................	1·5 "	2·5 "	1·5 "	5 "

In this series, 153 cases were pure diphtheria; 56 were complicated by measles or scarlet fever or both. The streptococcus was much oftener found in the viscera in the complicated cases; otherwise there was little difference in the two groups of cases.

The diphtheria toxins.—The wide-spread effects seen in diphtheria are due to the action of certain substances called *toxins* which the diphtheria bacillus produces during its growth on mucous membranes. They are very diffusible, readily entering the lymphatic circulation and the blood, and through these channels may affect the entire body. In susceptible animals there may be produced by the injection of these toxins all the characteristic lesions of diphtheria except the membrane, as well as the essential symptoms of the disease, even including paralysis. For the production of the membrane living bacilli are required.

"Catarrhal" diphtheria.—The routine practice of making cultures from diseased throats has established the fact that catarrhal inflammation may often be the only result of diphtheritic infection. Although to the naked eye there were only the ordinary changes of a simple inflammation, Oertel found the characteristic degenerative changes in the epithelial cells, varying in degree with the severity of the process.

The diphtheritic membrane.—The membrane in diphtheria is most frequently seen upon the mucous membrane of the tonsils, soft palate, uvula, pharynx, nose, larynx, trachea, and bronchi; less frequently upon the mouth, lips, œsophagus, conjunctivæ, middle ear, stomach, and genital organs. It may also affect fresh wounds, notably a tracheotomy wound, or any abraded cutaneous surface. The gross appearance of the membrane varies greatly (Plate XVIII). It is most frequently of a gray or mouse-colour, but it may be pearly white, yellow, green, and sometimes almost black. It is composed of fibrin, cells, granular matter, and bacteria. Its consistency varies with the relative proportions of the different elements. When made up chiefly of fibrin it is firm and retains its form, often being discharged as a complete cast of the nose, larynx, or trachea. When the amount of fibrin is small the membrane is soft, friable, and sometimes granular. It is more closely adherent upon the mucous membranes covered with squamous epithelium, as in the pharynx and upper air passages, than upon those covered with columnar and ciliated epithelium, as in the lower air passages.

The microscopical examination shows the fibrin to be sometimes granular, but usually in the form of a network, inclosing in its meshes small round cells and epithelial cells in various stages of degeneration. On the surface and in the superficial layer there is usually found quite a variety of bacteria including diphtheria bacilli. Beneath this is a cellular layer containing little or no fibrin, in which also the diphtheria bacilli are usually found. In the deepest parts of the false membrane and in the mucous membrane itself they are few in number or absent.

Characteristic changes, which are similar in all the affected mucous membranes, are found in the epithelial cells, which undergo marked degeneration with fragmentation of their nuclei; the mucosa is infiltrated with leucocytes. The infiltration with small round cells is variable in degree in the different mucous membranes; in some it extends deeply into the submucous and even the muscular layers, while in others it is very superficial. Marked evidences of degeneration are seen also in the cells infiltrating the deeper layers. In places the epithelium is detached, in others the line between the false membrane and the granular mucous membrane is scarcely distinguishable.

The seat and the distribution of the membrane.—This varies somewhat with the age of the patient, the season, and the peculiarity of the epidemic.

My own records show that the larynx is involved in about 40 per cent of the cases in children under three years. In general the statement may be made that the younger the child the greater the liability of the disease to attack the larynx; also when the larynx is affected, the greater the tendency to spread to the trachea and bronchi. The larynx and lower air passages are rather more frequently attacked in winter than in summer.

The tonsils are the most frequent and usually the earliest seat of the diphtheritic membrane; it may form here a tough, leathery patch, partially or completely covering and very adherent to them; or the disease may affect only the tonsillar crypts, so that the gross lesion may resemble that of ordinary follicular tonsillitis. There is in most cases only moderate swelling, but it may be so great that the tonsils are in contact. The surrounding cellular tissue is infiltrated with inflammatory products.

The membrane covering the pharynx and uvula is also usually very adherent and intimately blended with the mucous membrane. The uvula is swollen and œdematous. Membrane may be seen only upon the fauces and uvula, or the posterior and lateral pharyngeal walls may be covered down to the level of the cricoid cartilage, but generally not below this point. If the posterior pharyngeal wall is covered, the membrane is apt to extend into the rhino-pharynx, and may fill the entire pharyngeal vault, covering the posterior portion of the velum and extending into the posterior nares. The adenoid tissue of the vault is frequently the part most affected.

The nose may be involved secondarily to the rhino-pharynx, or infection may be through the anterior nares; if the latter, it is not infrequently the only part involved. Many cases classed as nasal are really rhino-pharyngeal. The membrane in the pure nasal cases is usually thick and tough and often separates *en masse*. Both sides are generally involved, but it may be unilateral.

The observations of Councilman, Mallory, and Pearce have shown that it is very common for the accessory sinuses of the nose, especially the antrum of Highmore, to be involved in fatal cases. It seems highly probable that infection of these parts explains the remarkable persistence of diphtheria bacilli in the nose which is occasionally seen.

The epiglottis is swollen to three or four times its normal thickness, and the aryteno-epiglottic folds are œdematous. The anterior surface of the epiglottis is rarely covered by membrane; but its lateral borders and posterior surface, and the aryteno-epiglottic folds are involved in most of the severe pharyngeal cases (Plate XVIII, C). This lesion is associated with pharyngeal rather than with laryngeal diphtheria.

The lesions which extend most deeply are thus seen in the tonsils, uvula, pharynx, and epiglottis. But even here there is very rarely deep or extensive sloughing.

The lesions of the larynx, trachea, and bronchi are similar to the above, although much more superficial. The interior of the larynx may be completely covered, the membrane coating the true and false vocal cords and lining the ventricles of the larynx. The membrane in the larynx is not usually very adherent, and it frequently separates and is coughed up in large pieces or even as a cast. That covering the epiglottis and the aryteno-epiglottic folds is very adherent, like that in the pharynx. Catarrhal laryngitis is not an uncommon complication of pharyngeal diphtheria.

In a considerable number of cases the membrane stops abruptly at the lower border of the larynx. In the trachea it is generally loosely attached, and often it is found at autopsy entirely separated from the mucous membrane. It is almost invariably associated with membrane in the larynx. Usually the membrane in the bronchi is continuous with that in the trachea. Occasionally I have seen the trachea and larger bronchi passed over and found membrane only in the larynx and smaller bronchi. As a rule, the bronchi of both sides are affected, and to the same degree. I once saw a case of laryngeal diphtheria in which membrane was found only in the bronchi of one lung. The above exceptions are to be explained as accidents in the mechanical transportation of bacilli.

The extent of the membrane varies greatly in different cases. It may stop at the bifurcation of the trachea or at the bifurcation of the primary bronchi; but if it goes beyond this point it is likely to extend to the minutest subdivisions. Exceptionally a very tough fibrinous membrane forms in the trachea and bronchi, of sufficient thickness and consistency to be expelled as a cast, reproducing almost the entire bronchial tree.

The inflammation of the mucous membrane of the larynx, trachea, and bronchi is very much less severe and more superficial in character than that of the pharynx, tonsils, and upper air passages.

The buccal cavity is very seldom covered by the membrane; but in the worst cases of pharyngeal disease it may line the cheeks, cover the lips, gums, and more or less of the hard palate, but rarely the tongue. It usually occurs in patches rather than as a continuous membrane. In one case I saw the membrane on the lower lip, extending on to the face, though the buccal cavity was free. It is not common for the diphtheritic membrane to spread down the digestive tract. In 127 autopsies studied by Councilman, Mallory, and Pearce, in which the extent of the membrane was carefully noted, it was found twelve times in the œsophagus, five times in the stomach, and once in the duodenum. The amount of membrane varied from small striations on the folds of the stomach or œsophagus to a complete covering. The accompanying changes consist in infiltration, hæmorrhage, and cell degeneration. In the intestines there is often found a hyperplasia of the lymphoid elements—solitary follicles and Peyer's patches—with changes similar to those in the lymph nodes elsewhere in the body, but nothing else that is characteristic.

The writers just referred to found otitis, usually double, in 60 per cent of 144 autopsies; although in less than one third of the number was the complication recognised during life. Mastoid disease is infrequent. Otitis is usually the result of direct extension from the pharynx. It may be due to the diphtheria bacillus alone, to the streptococcus alone, or more frequently to both combined; occasionally the pneumococcus is found. Conjunctival diphtheria is rare and probably due to accidental infection rather than extension through the lachrymal duct. Before the advent of antitoxin, it almost invariably resulted in destruction of the eye; but a number of cases successfully treated have now been reported, and one has recently come under my own observation. Diphtheria may attack any muco-cutaneous surface, especially the anus, prepuce, or female genitals; any abraded cutaneous surface, or recent wound, most frequently the tracheotomy wound of the neck. The diphtheria bacilli have been found in pure culture in superficial abscesses.

Visceral lesions.—The visceral lesions * of diphtheria are due partly to the action of the diphtheria toxins and partly to the invasion of the body with other organisms, especially the streptococcus. It is to experimental diphtheria that we owe our most accurate knowledge of the former changes, for in human diphtheria the large proportion of all the fatal cases show infection with other organisms, particularly the streptococcus, to a less degree the pneumococcus or staphylococcus. The frequency with which these bacteria are found at autopsy in different organs has been already stated.

The visceral lesions of diphtheria consist in wide-spread areas of cell degeneration similar to those which have already been described as occurring in the epithelial cells of the affected mucous membranes, together with hæmorrhages due to changes in the blood-vessels and possibly in the blood itself.

The lymph nodes of the cervical region are the most constantly and the most seriously affected. Similar but less marked changes are seen in the tracheo-bronchial and the mesenteric groups, and in the lymph nodules of the mucous membrane of the stomach and intestine. There are degenerative changes in the cells of the nodes most affected, with marked infiltration with leucocytes and frequently small hæmorrhages. The cellular tissue in the neighbourhood of the cervical nodes is often extensively infiltrated with cells. The process in the lymph nodes usually terminates in resolution, rarely in suppuration.

The changes in the spleen are quite constant. The organ is swollen, sometimes very much so, and deeply congested. Hæmorrhages are often seen beneath the capsule; the spleen pulp is soft, the follicles are large, and cell degeneration is quite constantly observed similar to that which takes place in the lymph nodes.

There are frequently small hæmorrhages beneath the capsule of the liver, and sometimes these are seen throughout the organ. There are found scattered through the liver, areas of necrotic hepatic cells which are peculiar to this disease; some of these areas are infiltrated with leucocytes.

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* For an exhaustive study of the pathological anatomy of diphtheria, see monograph of Councilman, Mallory, and Pearce (Boston, 1901); being a study of 220 fatal cases.

The kidneys are involved in almost all fatal cases except where death occurs early from laryngeal stenosis, also in nearly every severe case which terminates in recovery. Acute degeneration of the epithelium of the tubes and the tufts is seen in less severe cases and those of shorter duration, and is the direct result of the action of the toxins in the blood. In the more severe and protracted cases there is acute diffuse nephritis of variable type and intensity. There is no form of inflammation which is peculiar to diphtheria; in some cases the interstitial changes predominate, in others the glomerular changes. Welch mentions hyaline changes in the glomerular capillaries and small arteries as the characteristic feature of the nephritis of diphtheria.

In children dying suddenly in the early stage of the disease, cardiac thrombi are occasionally found. They may form rapidly only a short time before death, or slowly during several days when the circulation is very feeble. Portions of these thrombi may be carried into the pulmonary or systemic circulation, causing embolism in any of the arteries of the extremities, the lungs, or other viscera. Even in the early fatal cases the heart muscle may be seriously affected; in the later ones this is almost constant. The changes consist in a toxic myocarditis, the left ventricle being most involved.

Degeneration of the arteries, especially of the endothelial layer, is occasionally seen, and there may be infiltration of the adventitia. The arteries of any of the viscera may be the seat of hyaline degeneration.

Lesions of the brain are rare; both hæmorrhage and embolism may be met with. In the spinal cord and membranes multiple hæmorrhages occasionally occur. The characteristic lesion, however, consists in degenerative changes which are found to some degree in nearly all the more severe cases which have been examined. These affect the ganglion cells of the anterior horns, the anterior and posterior nerve-roots, and sometimes the pyramidal tracts and columns of Goll. In some cases of paralysis induced in animals, lesions practically identical with those of ordinary poliomyelitis have been seen. Some recent writers (Katz and Crosz) are of the opinion that the cord lesions are primary and the degeneration of the spinal nerves secondary. However, the general opinion still prevails that certainly the less severe cases of diphtheritic paralysis are due to peripheral rather than to central lesions. Degenerative changes have been found also in the pneumogastric, spinal accessory, hypoglossal, motor-oculi, and in the cardiac nerves. These nerve degenerations produced by the diphtheria toxin constitute one of the most striking lesions of diphtheria. (See Multiple Neuritis.)

In infants and young children broncho-pneumonia is found at autopsy in fully three fourths of the cases, and in a large proportion of these it is the cause of death. It is well-nigh constant in cases of diphtheritic bronchitis of the finer tubes, and is usually present where the membrane has extended to the bifurcation of the trachea. The largest factor in the production of pneumonia is the aspiration of diphtheria bacilli and streptococci from the upper air passages; an important part is also played by the pneumococcus and the influenza bacillus. These organisms may be present in many combinations.

With laryngeal stenosis, some emphysema is invariably present, and usually it is of the vesicular variety. In extreme or protracted cases of stenosis there may be interstitial emphysema. Rupture of some of these blebs may lead to the escape of air into the cellular tissue of the mediastinum or of the neck, which may result in the production of a general emphysema of the subcutaneous cellular tissue.

Blood.—According to the studies of Ewing, Morse, Billings, and others, there is found in all severe cases of diphtheria a reduction in the number of red cells to the extent of 500,000 to 2,000,000. There is a nearly proportionate reduction in the hæmoglobin, this amounting to from 12 to 28 per cent. While the hæmoglobin falls coincidently with the number of red cells, it is regained much more slowly. Leucocytosis is generally present, and usually proportionate to the severity of the attack, but is occasionally wanting in the most severe as well as in some of the very mildest cases. The increase in the leucocytes is in the polynuclear forms. Engel has noted the frequent presence of myelocytes, especially in fatal cases, the proportion of these in some instances reaching 16 per cent of the white cells. In his observations, every case in which the myelocytes exceeded 2 per cent, proved fatal.

Symptoms.—The clinical picture of diphtheria is one which presents wide variations, depending upon the principal location of the disease, its severity, and its complications. For practical purposes the following seems the simplest grouping that can be made:

1. The mild cases, in which there is either no membrane, or the amount of membrane is small and limited to the tonsils or to the nose, with few or none of the constitutional symptoms which follow absorption of the diphtheria poison. These cases partake essentially of the character of a local disease.

2. The severe cases, which are of two kinds: first, those in which there are marked evidences of constitutional poisoning from diphtheria toxins; and, secondly, those with laryngeal stenosis. The first form is usually accompanied by an extensive formation of membrane in the pharynx and sometimes in the nose. The larynx may be involved secondarily to disease in the pharynx or nose, or it may be primarily affected.

3. The cases of mixed infection or the septic cases. In very many of the cases of the two preceding groups streptococci are found in the throat, but they are not in sufficient numbers or of sufficient virulence to modify the course of the disease. In the cases to which the term

mixed infection is applied, in addition to the constitutional symptoms of diphtheritic toxæmia and the local conditions which usually attend it. there are marked evidences of a general septicæmia, usually due to the streptococcus. In these cases the symptoms of inflammation are especially prominent, not only in the pharynx but sometimes in the lymph glands and cellular tissue of the neck, which may be followed by suppuration or sloughing. This form is frequently complicated by broncho-pneumonia even without laryngeal disease, and sometimes by severe nephritis.

Cases without membrane.—During an epidemic of diphtheria in a family or an institution, cases are frequently seen which present the clinical evidences of only a catarrhal inflammation of the nose or pharynx, and yet cultures show the presence of the diphtheria bacillus. Such cases may be examples of simple catarrhal inflammation with the accidental presence of the diphtheria bacillus; or the inflammation may be caused by infection with the diphtheria bacillus, but not of sufficient intensity to lead to the production of a membrane. The latter is the view of pathologists, and the one to which clinicians must, it seems, inevitably come. However, a membrane has so long been regarded as a *sine qua non* of this disease that the existence of diphtheria without it, is something which the clinician finds it hard to grasp.

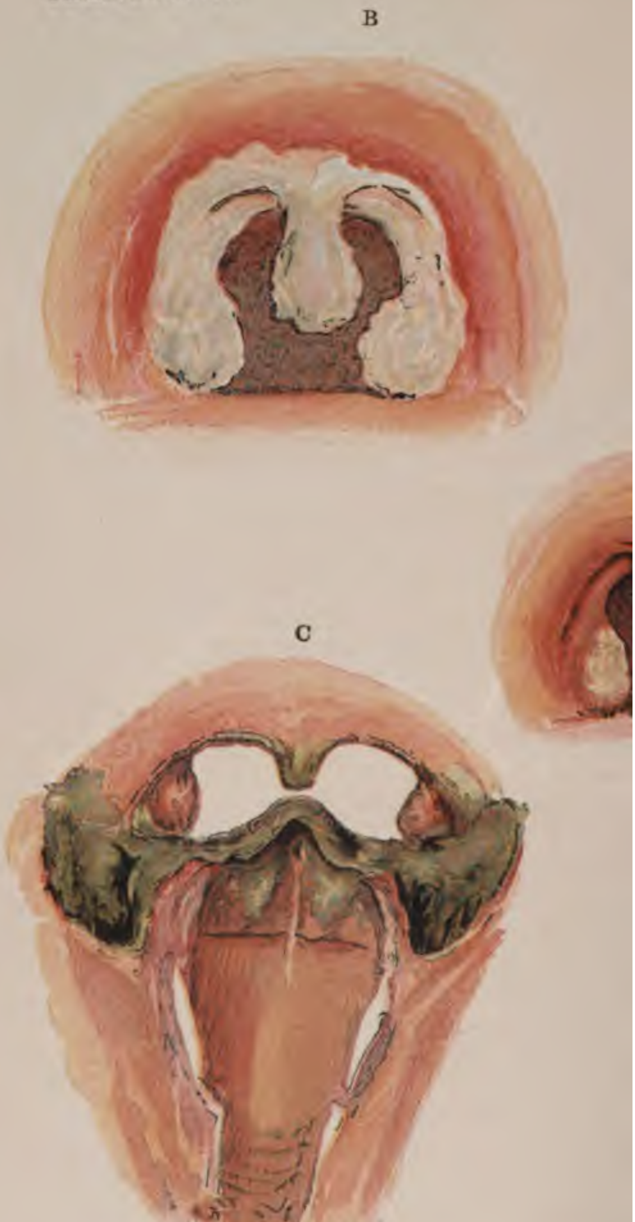
Catarrhal diphtheria may be either pharyngeal or nasal. In the pharyngeal cases there are present the usual appearances belonging to a catarrhal inflammation of moderate severity, often accompanied by swelling and tenderness of the cervical lymph glands.

The nasal cases, in my experience, have been most frequent in infants or very young children. Constitutional symptoms may be wanting or so slight as to be overlooked. The only striking thing is a persistent nasal discharge which may be serous and frothy, purulent or bloody. It is usually copious, often excoriating the upper lip and sometimes continuing for three or four weeks before any other symptoms are observed. I have known it to be mistaken for a syphilitic coryza. Such cases can be recognised with certainty only by cultures. Clinical evidence of their true character is sometimes afforded by the appearance of visible membrane in the nose or pharynx, by the development of croup, or by the fact that they cause diphtheria in other children.

Catarrhal diphtheria is not in itself serious, but it may be followed, particularly in young children, by laryngeal diphtheria, or, after it has existed for a time, pharyngeal diphtheria may develop in its usual form. Cases like those just described are to be distinguished from others in which bacilli, either of the virulent or the non-virulent variety, are found without any evidence of inflammation.

Cases with a small amount of membrane.—Tonsillar diphtheria.—The exudation is usually limited to the tonsils (Plate XVIII, A), and

PLATE XVIII.



THE DIPHTHERITIC MEMBRANE.

A. Typical tonsillar diphtheria.
B. Severe pharyngeal diphtheria (fatal case).
C. Pseudo-diphtheria. The specimen is seen from behind, t
having been laid open, and shows an extensive membrane involv
the entire lower pharynx, but extending into the larynx only a
also seen upon the posterior surface of the uvula and soft palate.
partially covered. The colour of the membrane is not characte
theria, as the same appearance is often seen in true diphtheri
septic type.

may partake of the character of either follicular or croupous tonsillitis; sometimes there is a slight extension to the faucial pillars or to the pharynx. These cases are quite common, and in some epidemics most of those seen are of this variety. They are more frequent in older children and adults than in infants and young children.

The onset is accompanied by a little soreness of the throat; the initial temperature is from 101° to 104° F.; but the symptoms are often not severe enough to keep the patient in bed. If seen early, the throat shows slight redness, followed by a gray film, and later by a gray or white deposit upon the tonsils. It may start as a small patch which enlarges, or as small, isolated spots which coalesce or remain separate. Until it disappears the membrane generally remains of its original colour. It is generally quite adherent, and can not easily be removed with a swab; usually it is sharply defined, but with a somewhat irregular outline. In many cases the patch is not larger than the finger nail. The inflammatory changes in the pharynx are slight; a faint red areola is frequently present at the border of the patch. The lymph glands behind the jaw may be slightly swollen. There is no nasal discharge and very little increase in the saliva or mucus from the pharynx. Some constitutional symptoms are present, but they are never severe. The temperature commonly continues above the normal while the membrane lasts, its usual range being from 100° to 102° F. The membrane remains from three to seven days—a shorter time if antitoxin is used. It is very often a matter of surprise that so small an exudate is so persistent. The urine is generally normal. The parents are loath to believe that strict quarantine is necessary in so mild an illness; and where the membrane is only upon the tonsils, even after the disease has run its course, the physician may be lead to doubt the diagnosis of diphtheria.

In many cases one with experience can usually make an accurate diagnosis from the clinical symptoms alone; but there are many others in which the diagnosis from ordinary tonsillitis is impossible, even by the most practised observers, except by cultures. When diphtheria bacilli are found in these mild cases the question often arises whether they may not be the non-virulent form. Park tested forty such cases, and found the bacilli to be virulent in thirty-five and non-virulent in five. In twenty of the forty cases the clinical diagnosis was follicular tonsillitis.*

Severe cases.—The clinical picture of diphtheria is so modified by the use of antitoxin that those who see it given regularly and early can have but little conception of the horrors of this disease when not thus influenced. The onset in severe cases may be gradual, even insidious.

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* From one of these mild cases was obtained a bacillus whose virulence so greatly exceeded that obtained from any other case of diphtheria, that its cultures were used for the preparation of toxins for injecting horses. It was by means of these powerful toxins that the strongest antitoxin was produced.

There is then a slight indisposition for a day or two, ar soreness of the throat; the temperature may be but little times less than 100° F. The symptoms may steadily incr for four or five days, until the maximum is reached. At disease begins abruptly with vomiting, headache, chilly s temperature of 103° or 104° F. Occasionally, the first attention is the swelling of the cervical lymph glands, v great that mumps is suspected. The abrupt onset is mo young children than in those who are older.

The membrane upon the tonsils resembles that of the viously described, but, instead of remaining limited to th spreads to the fauces, the lateral wall of the pharynx rhino-pharynx, and the posterior nares. The rapidity membrane extends is in direct proportion to the severit In some cases it may cover all the parts mentioned in tv from its first appearance; in others this may require seve the nose is first affected there is an abundant discharg mucus, occasionally tinged with blood, which may con before any membrane is visible.

When a severe case is fully developed there is a ver charge of mucus from the mouth and nose. The tonsils cial ring, and the pharynx are covered with membrane (F which is at first gray and gradually becomes darker, dirty olive-green colour. Membrane is sometimes seen in patches in the mouth. There is obstruction to nasal the swelling of the palate, the tonsils, and the tissues of ynx; the mouth is half open, the breathing noisy, the the lips are fissured and bleed readily. Occasionally la orrhages occur which may necessitate plugging the nares are generally blocked by the swelling and the false mer charge excoriates the upper lip, and frequently has a fet ing the second week there may be regurgitation of flu nose, owing to paralysis of the palate. The lymph glar of the jaw swell rapidly; in severe cases they are very there may also be extensive infiltration of the cellul them, although this is more characteristic of the case fection.

The constitutional symptoms usually increase stead tension of the membrane. In the most severe cases the whelmed with the poison, and all the evidences of inte present by the third day of the disease. This is shown lar weakness and prostration, by a feeble, rapid pulse, an of complete apathy or stupor, sometimes alternating lessness. It is more frequent for the constitutional sym gradually, and not to reach their height before the fourth or fifth day. The pulse becomes rapid, weak, and compressible, sometimes irregular; and there is a great and steadily increasing anæmia. The course of the temperature is irregular, and bears no constant relation to the severity of the other symptoms. Its usual range is from 101° to 103°, but in some of the worst cases it may never go above 101° F. It fluctuates irregularly with the development of complications, and sometimes without apparent cause. By the second or third day the urine regularly shows the presence of albumin, and by the end of the first week the quantity is often large. Granular and hyaline casts, and occasionally blood in small quantities, are also found. The amount of urine secreted is not noticeably diminished, and dropsy is rare. There is complete anorexia, and often vomiting and diarrhœa are present; in some of the cases they are prominent. Nervous symptoms are seen in all the very severe cases. There may be dulness and apathy, but more frequently, owing to the discomfort arising from local symptoms, there is extreme restlessness and excitement, sometimes followed by delirium.

At any time during the first week, but not often after that time, symptoms may arise indicating that the disease has extended to the larynx. The first signs of laryngeal invasion usually appear from the second to the fifth day of the disease. These are at first hoarseness, a croupy cough, and slight dyspnœa. In the severe cases these symptoms steadily increase until all the signs of laryngeal stenosis are present. The symptoms of diphtheria of the larynx, whether it begins there or follows disease of the pharynx, have already been described in the chapter on Diseases of the Larynx.

The local process in the pharynx seems to be a self-limited one, even when no antitoxin is used. It usually reaches its height by the fifth or sixth day, and after that the appearances do not change materially for two or three days. From the seventh to the tenth day, in favourable cases, the diphtheritic membrane begins to loosen and separate from its attachment. It hangs loosely from the palate or uvula, and can often be pulled away in large masses. The detachment is frequently rapid, and in two or three days from the time when the first improvement is seen, the tonsils and pharynx may be almost free from membrane. The mucous surface left behind is of a bright red colour and bleeds easily. The separation of the membrane in the nose and rhino-pharynx takes place more slowly. From the former it may disintegrate gradually or come away *en masse*. With the disappearance of the membrane the local symptoms abate rapidly—the discharge ceases, the swelling of the lymph glands subsides, deglutition becomes easy and natural, and nasal breathing is re-established. When antitoxin is given the local process passes through similar stages, but much more rapidly.

Simultaneously with these changes in the throat the constitutional symptoms improve, but much more slowly. Convalescence is often protracted. The anæmia and muscular weakness, and, most of all, the feeble heart action, may persist for weeks.

Instead of the usual course just described, the diphtheritic membrane may persist for two or three weeks. In rare cases relapses occur, the membrane forming again after it has entirely or partially disappeared.

The early course of the disease in the fatal cases often does not differ from that of the severe cases which end in recovery, except in the malignant form, which kills in twenty-four or forty-eight hours, and which is very rare. In very young children death is most frequently due to broncho-pneumonia, usually accompanying diphtheria of the larynx and bronchi. It may also be due to progressive asthenia the result of diphtheritic toxæmia, or to heart failure, which may come early or late; rarely to nephritis.

Pneumogastric paralysis.—This usually follows severe types of infection, and is seen not only in cases in which no antitoxin is given, but also when it is administered late or in too small doses. In such circumstances the early toxæmia may be neutralised and the local disease in the larynx and trachea controlled; yet so susceptible are the nervous tissues to the action of the diphtheria toxin, that injury sufficient ultimately to produce death may still have been done. This is most frequently through the action of the toxin upon the pneumogastric nerves.

Pneumogastric paralysis may come on at any time in the course of the disease, but seldom earlier than the end of the second week. By this time the throat has usually cleared off entirely, and the patient is considered convalescent. The physician has ceased his frequent visits and looks in only once a day to satisfy himself that all is going well. The symptoms relate to the stomach, the heart and the respiration. Usually the first thing to attract notice is that the patient refuses food and vomits occasionally, afterward persistently, without apparent cause. If the pulse is carefully observed it is found to be much slower than previously, being only 80 or 90 when it was formerly 120 or more. It is also weaker, compressible, and often somewhat irregular. The face is pale, often slightly cyanotic, and moderate dyspnœa may be noticed. There are frequent attacks of severe abdominal pain which comes in paroxysms, and is usually referred to the epigastrium. These symptoms in most cases gradually increase in severity for two or three days, but sometimes develop with such intensity that death occurs within twelve or twenty-four hours. The later symptoms are a continuance of the abdominal pain and vomiting; there is a feeling of great precordial oppression and distress accompanied by dyspnœa; the respiration is shallow and often rapid; the face is either pale or cyanotic; the extremities, cold; the pulse, slow, irregular and intermittent, becoming rapid on the slightest exertion. The heart sounds are weak, the muscular quality is absent, and the rhythm much disturbed. There may be no murmurs. There is great restlessness, but the mind is entirely clear. Death usually results from syncope, which may come quite suddenly, often from so slight exertion as turning over in bed or attempting to take food.

Not all the cases are so severe. In the milder forms of the condition there is some palpitation, an irregular pulse, slight dyspnœa, and occasional syncopal attacks, but of no great severity. Such symptoms may come and go for several days and then disappear; but more frequently they prove to be the beginning of the more serious form of the complication.

The time of occurrence of pneumogastric paralysis varies considerably. It may be as late as the third or fourth week. The late cases are generally associated with some other form of post-diphtheritic paralysis.

Sudden heart failure may be seen late in diphtheria quite apart from the symptoms just described. It may occur with few or no premonitory symptoms; as when a child falls dead after walking across a room, or suddenly sitting up in bed, or from some other muscular effort, or possibly as a consequence of passion or excitement. I knew of one little girl who was considered well enough to go coasting and who died suddenly after the effort.

The explanation of heart failure during or after diphtheria is therefore not always the same. When it occurs at the height of the disease it is sometimes due to cardiac thrombosis, probably always associated with changes in the muscular walls. When it occurs late and follows some sudden muscular effort or excitement without premonitory symptoms of any sort, it is probably the result of changes in the muscular walls—a toxic myocarditis. When prodromal symptoms are present, and particularly when accompanied by vomiting, abdominal pain, and disturbed respiration, it is probably the result of a toxic neuritis affecting either the pneumogastric or the cardiac nerves, and is to be regarded as a form of post-diphtheritic paralysis. In many cases, no doubt, changes are present both in the nerves and in the myocardium. The other forms of diphtheritic paralysis which may result fatally, are discussed in the chapter on Diseases of the Peripheral Nerves.

Cases of mixed infection or septic diphtheria.—The symptoms are usually severe from the outset. The exudation in these cases may be of a yellow, or dirty-gray, or olive colour, sometimes being almost black from the presence of blood. The membrane is usually extensive, covering the entire pharynx, often extending to the nose and the middle ear, and occasionally spreading to the buccal cavity. There is great swelling of the tonsils and uvula, and it is often impossible to obtain a view of the pharynx; all the evidences of inflammation are usually more marked than in the severe uncomplicated cases. Sometimes the inflammation is of a necrotic character, and there may be extensive sloughing of the tonsils, the uvula, or the soft palate. The nasal discharge is generally abundant, and often very offensive. There is marked swelling of the cervical lymph glands, and frequently extensive infiltration of the cellular tissue of the neck, so that the head is thrown back to relieve the pressure upon the larynx and trachea. The swelling sometimes forms a distinct collar, reaching from ear to ear and filling out the whole space beneath the jaw. The pressure upon the jugular veins leads to congestion and swelling of the face and congestion of the brain.

The general symptoms are those of a severe septicæmia. The temperature is usually higher than in simple diphtheria; it follows no regular course, but is generally high and sometimes fluctuates widely from 102° to 106° F. In the cases characterised by such high temperature there is frequently found a general streptococcus or pneumococcus infection, usually the former. The pulse is weak, rapid, and compressible. The peripheral circulation is poor, the extremities are often cold, there is extreme muscular prostration, and both vomiting and diarrhœa are frequent. There may be excitement, restlessness, and active delirium, or dulness, apathy, and stupor. Nephritis is very frequent and is often severe; the urine contains a large amount of albumin and casts of all varieties, but rarely blood. In a large proportion of the children under three years old broncho-pneumonia develops. Severe symptoms continue for from two days to a week; the patient may die from the sudden invasion of the larynx, or there may be suppression of urine and uræmic convulsions; but more frequently the cause of death is asthenia or broncho-pneumonia. Death usually occurs while the local disease is at its height. Occasionally it comes later from heart failure, after the signs of local improvement have begun.

Those who manage to escape the dangers of the acute period have still others to encounter. Among the latter may be mentioned: extensive sloughing in the throat or of the cellular tissue of the neck, which may be followed by severe or even fatal hæmorrhage, diffuse suppuration of the same region, late nephritis, pneumonia, or pleurisy, and finally paralysis of the heart or respiration.

Complications and Sequelæ.—Most of the complications of diphtheria have already been mentioned either under the head of Lesions or Symptoms. It only remains to consider their clinical association.

Otitis occurs particularly in the rhino-pharyngeal cases, and is sometimes due to the diphtheria bacillus alone, but more often to mixed infection. The type of inflammation is often a severe one, and it may be accompanied by necrotic changes in the drum membrane which resemble those of scarlet fever.

Broncho-pneumonia is the most frequent complication in young children. It occurs especially in laryngeal cases, and in those of a septic type whether the larynx is involved or not. Other pulmonary complications are infrequent. Pleurisy with a serous effusion may occur in connection with severe nephritis, and empyema in septic cases. Emphysema is a complication of laryngeal diphtheria; it is nearly always vesicular, sometimes interstitial, and may become general, extending into the cellular tissue of the neck and afterward that of the entire body. Pericarditis, endocarditis, and meningitis are all very rare and are seen chiefly in septic cases of the most severe type. Myocarditis is much more frequent, and is present to a greater or less degree in nearly all severe cases, although in but a small proportion of these does it give rise to distinct symptoms. It is closely connected pathologically with degeneration of the cardiac nerves, and it may be a cause of sudden death at any time during the acute period of the disease or during convalescence.

Thrombosis and embolism are among the less frequent complications. If cerebral, they may cause hemiplegia, aphasia, and sometimes convulsions; if peripheral, they usually affect one of the lower extremities, where they may cause sudden pain, numbness, and coldness of the limb, followed by partial paralysis, œdema, and sometimes even by gangrene. Thrombosis of the pulmonary artery or of the heart may be a cause of sudden death, the symptoms being dyspnœa and præcordial distress, with pallor or cyanosis. Both thrombosis and embolism are associated with a very feeble action of the heart, and generally they are preceded by degenerative changes in its muscular walls.

Hæmorrhages are usually nasal, and while in most cases they are not serious, they may necessitate plugging of the posterior nares. Bleeding from any other mucous membrane may occur, but it is rare except from the mouth. Subcutaneous hæmorrhages are infrequent, and are evidence of a very high degree of diphtheritic toxæmia. They usually occur as small petechial spots, but are sometimes extensive. They may be seen upon almost any part of the body, most frequently upon the abdomen and lower extremities; but the most extensive extravasation I have ever seen was in the neck, reaching from the clavicle almost to the ear and covering nearly one lateral half of the neck.

Albumin is present in the urine of almost every case of moderate severity, usually depending upon acute degeneration of the kidneys. Acute nephritis is most frequently seen in septic cases. It then usually develops at the height of the local disease, but may come during convalescence. Albumin and casts are found in the urine, but rarely is there dropsy or signs of uræmia. Less frequently a more severe form of inflammation occurs, with dropsy, scanty urine, or even suppression, vomiting, and all the usual symptoms of acute uræmia. This complication may be a cause of death.

Functional disturbances of the stomach are present in most of the severe cases, but lesions of the mucous membrane are rare. While diarrhœa is often seen without intestinal lesions, the latter are of frequent occurrence. The most characteristic form of inflammation is a follicular ileo-colitis, which, however, seldom goes on to ulceration. It is extremely rare that the membranous form is seen, and then it is almost always associated with the presence of other bacteria, not with diphtheria bacilli.

Diphtheria is usually followed by a severe and often persistent anæmia which may continue for weeks. Pneumonia, nephritis, and cardiac disease may first show themselves during convalescence, and so be ranked as sequelæ. The most important sequel of diphtheria, however, is post-diphtheritic paralysis, already discussed in the chapter on Multiple Neuritis.

Diagnosis.—The diagnosis of diphtheria rests upon two kinds of evidence—clinical and bacteriological. In mild cases and in the early stage only bacteriological evidence can be relied upon. However, the clinical manifestations of the disease are important and should not be ignored. It is in most cases possible to say from clinical symptoms that a case is one of diphtheria; but it is never possible to say from symptoms alone that a case is not diphtheria. Cultures, therefore, are of the greatest assistance, and should if possible be made in every case. They are necessary in the mild cases in order that a correct diagnosis may be made and proper quarantine regulations enforced; otherwise a case might be dismissed as simple tonsillitis and no precautions taken.

The mere presence of diphtheria bacilli in the throat does not prove that a person has diphtheria any more than the presence of the pneumococcus in his saliva proves that he has pneumonia; but where diphtheria bacilli are associated with clinical evidences of inflammation of the throat or nose the diagnosis may be regarded as established. Again, the case may be one of diphtheria and the bacilli not found at the first examination, although found subsequently. In using antitoxin one must, in perhaps the majority of cases, be guided by clinical symptoms alone, not waiting for the result of the bacteriological examination. It is therefore important that both methods of diagnosis should be employed.

1. **The Clinical Diagnosis.**—Not much importance can be attached to the mode of onset; for diphtheria may begin in many different ways. The presence of a nasal discharge, especially if abundant, ichorous and tinged with blood, the early development of the symptoms of croup, the rapid enlargement of the cervical lymph glands, and the early appearance of albumin in the urine—all point strongly to diphtheria. Later symptoms which are especially diagnostic are marked anæmia, progressive asthenia, intense toxæmia often with a low temperature, very

feeble pulse which is sometimes slow, sometimes rapid, sudden attacks of syncope, nasal hæmorrhages, nasal regurgitation from paralysis of the soft palate, contagion, and, finally, the development of paralysis of the muscles of the throat, eye, or extremities, with paralysis of the heart or respiration.

The membrane of diphtheria generally appears first upon the tonsils, usually as a gray film which gradually becomes more dense and white, and often has the look of being plastered on. The colour of older membrane is gray, greenish-yellow, brown, sometimes black. Beginning as a small patch, it soon covers the tonsils. It frequently affects one tonsil twenty-four or thirty-six hours before the other, and occasionally it is confined to one side. In exceptional cases it begins in the crypts of the tonsil and appears as isolated dots, which may coalesce to form a continuous patch like that already described, or it may remain isolated like the exudate of an ordinary follicular tonsillitis. More important is the fact that the membrane spreads from the original seat, and also the manner of its spreading. If it extends beyond the tonsils to the walls of the pharynx, the faucial pillars, and the uvula, it is almost surely diphtheria. The same is true of doubtful patches on the tonsils or fauces followed by symptoms of croup. The rapidity of the spreading varies much in the different cases, depending upon the intensity of the infection; but the gradual extension, as shown by observations made at intervals of six or eight hours, usually settles the diagnosis in the primary cases. However, if the throat symptoms complicate measles or scarlet fever the above rules do not apply. Most of the membranous inflammations of the throat seen in these diseases are not due to diphtheria. This is particularly true of those which occur at the height of the primary disease. Those which develop at a later period are often due to diphtheria.

In pure diphtheria there is a notable absence of œdema of the faucial pillars and uvula, so common in throat inflammations due to cocci. In fact, whenever there are seen in the throat evidences of a very high degree of inflammation, it usually points either to mixed infection or to false diphtheria.

Primary membranous inflammation of the larynx may always be safely regarded as diphtheria; but if there is no visible membrane, the diagnosis is rendered positive only by a bacteriological examination. This may be true of many nasal cases where the only symptoms are a discharge of the character previously described. Such cases may continue for weeks with no symptoms other than the discharge, especially in infants.

The most characteristic clinical differences between diphtheria and other inflammations accompanied by an exudation upon the throat or in the nose—i. e., pseudo-diphtheria—are shown in the following table:

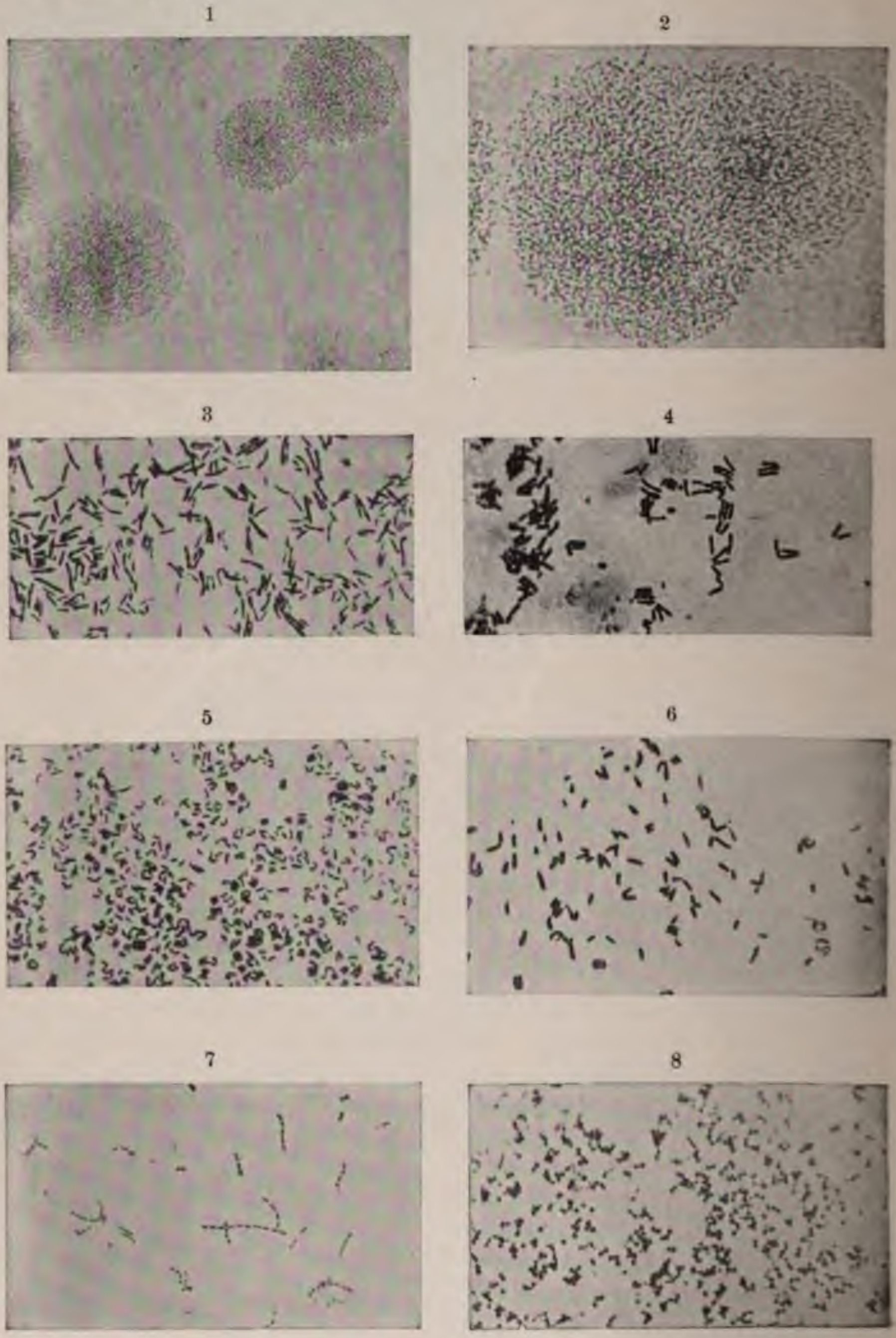
DIPHTHERIA.	PSEUDO-DIPHTHERIA.
1. Often a history of exposure, or presence of an epidemic.	1. Usually none.
2. Onset often gradual, with low temperature and slight constitutional symptoms.	2. Onset usually abrupt, with high temperature and quite marked constitutional symptoms.
3. Previous attacks rare.	3. Often a history of repeated attacks.
4. Often begins in the larynx.	4. Seldom if ever does so when primary.
5. If pharyngeal, shows a strong tendency to extend to the larynx.	5. This tendency is much less marked.
6. Primary cases frequently severe.	6. Rarely severe unless secondary, particularly to measles or scarlet fever.
7. When it complicates measles or scarlet fever it often develops late—after the primary fever has subsided.	7. Usually occurs at the height of the primary disease.
8. Occasionally limited to the nose (membranous rhinitis).	8. Doubtful if ever so.
9. Albuminuria the rule except in the mildest cases.	9. Rarely seen in primary cases, and sometimes not in the secondary form even though the symptoms are severe.
10. Nasal regurgitation from paralysis of the palate in the second week or later.	10. Never seen.
11. Toxic symptoms common; asthenia, great anæmia after the fourth or fifth day; later, sudden heart paralysis, respiratory paralysis, or post-diphtheritic paralysis of throat, eyes, or extremities.	11. Septic symptoms frequent, especially when secondary, but the peculiar toxic symptoms are never seen.
12. Usually less evidence of inflammation of mucous membrane and in surrounding parts.	12. Often evidence of intense inflammation.
13. A membrane on the tonsils with patches on the uvula or elsewhere in the pharynx is usually diphtheria; doubtful patches on the tonsils followed by croup almost invariably diphtheria.	13. It is never possible to say by the appearance of the membrane alone that the case is not true diphtheria.

It is seldom difficult to distinguish diphtheria from any other disease; but the exudation upon the pharynx or tonsils may be confounded with thrush or herpes. The appearance of the tonsils on the second or third day after tonsillotomy has been performed, may easily be mistaken for diphtheria by one who is unfamiliar with the appearance of the wound.

Diphtheria of the mouth may be mistaken for herpetic or ulcerative stomatitis; but, as a rule, it is seen only in the worst cases of pharyngeal diphtheria. Diphtheria of the mouth alone is so rare that it may be ignored.

It is sometimes difficult to distinguish cases of scarlet fever in which the throat symptoms are severe and appear early, from cases of primary diphtheria. In many of these cases the eruption appears late, and is

PLATE XIX.



DIPHTHERIA BACILLI AND THEIR ASSOCIATES.

1 and 2, colonies of diphtheria bacilli under a low and a high power; 3, 4, 5, characteristic diphtheria bacilli × 1,000; 5, showing the short even-stained diphtheria bacilli; 6, pseudo-diphtheria bacilli; 7, streptococci from a serum culture; 8, streptococci from a smear directly from the throat. (After Park.)

not characteristic. Much importance is to be attached, as pointing toward scarlet fever, to a prevailing epidemic, a history of exposure, a sudden onset with severe symptoms, vomiting, prostration, very high temperature, and to a very active inflammation in the pharynx. In all cases with a sudden onset, in which from the throat symptoms one is inclined to make a diagnosis of diphtheria, the possibility of scarlet fever should not be forgotten; and one should never omit to examine the patient thoroughly for an eruption. The diagnosis of primary diphtheria of the larynx has already been considered (page 495).

2. **The Bacteriological Diagnosis.**—*The technique.*—In many cases an immediate diagnosis may be reached by the examination of a cover-glass smear from the throat. This method, although often valuable, is not adapted for general use, as bacilli directly from the throat are much less typical than those from cultures, and the chances of contamination are much increased. Furthermore, the mouth often contains bacilli which somewhat resemble the diphtheria bacillus.

In taking a culture from the throat, the tongue should be depressed and the tonsils, pharynx, or other seat of visible membrane rubbed firmly with a swab, which is then rubbed over the surface of the culture-medium in the tube or on the plate. In laryngeal cases the culture should be taken from the posterior wall of the pharynx, and in nasal cases from the nostril. The tube or plate is then placed in an incubator for twelve or fourteen hours * and kept at a temperature of about 100° F. (37° C.), at the end of which time the colonies (Plate XIX, 1 and 2) may be examined. Examination, in the great majority of cases, shows either a great number of diphtheria bacilli (Plate XIX, 3, 4, and 5) and a few cocci, or only cocci in pairs or short chains (7 and 8); exceptionally, the cocci and bacilli may be present in nearly equal numbers. A definite opinion should not be given without examining several colonies.

The reliance to be placed upon bacteriological diagnosis.—The diphtheria bacillus will almost invariably be found: (1) if there is visible membrane in the pharynx; (2) if the culture is made during the period in which the membrane is forming; (3) if no antiseptics have been applied shortly before using the swab; (4) if the culture has been made with sufficient care to avoid contamination.

The diphtheria bacillus sometimes disappears early; hence cultures made while the membrane is loosening may be negative. If the membrane has disappeared, or if none has been present, it is not infrequently necessary to go into the tonsillar crypts with a probe or spoon to discover bacilli. It is therefore important in all cases to consider the duration of the disease before drawing a conclusion from a negative culture. If the case is one of laryngeal disease without pharyngeal exudation, an early culture is negative in nearly half the cases; although a little later bacilli may be coughed up and found in the pharynx in abundance. A single negative culture should never be taken as conclusive.

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* In the laboratory of the Babies' Hospital we have found that the rapid method of staining cultures at the end of five or six hours can usually be depended upon, but that it is not always reliable where the result is negative. In every case it is wise for control to make an examination of individual colonies at the expiration of the usual time. However, the rapid method is of great advantage, as the saving of time is of so much importance in the administration of antitoxin.

For diagnostic purposes, all bacilli present in suspicious throats, having the morphological and cultural characteristics of diphtheria bacilli, are to be regarded as virulent.

Non-virulent bacilli resembling the diphtheria bacillus.—There may be found in throats a form which corresponds in every other characteristic with the diphtheria bacillus, but which lacks virulence as shown by animal tests. Also, another form, which, though in many particulars resembling the diphtheria bacillus, differs from it in being shorter, plumper, and more uniform in size, and in producing an alkali in broth cultures; to this the term *pseudo-diphtheria bacillus* * (Plate XIX, 6) has been given. It is more frequently seen than the form just described and like it is non-virulent. Both these forms are rare in throats where a suspicion of diphtheria exists.

The presence of virulent bacilli in the throats of healthy persons.—That virulent bacilli may be harboured for an indefinite period in the throat or nose of a healthy person is proved by many observations. In Escherich's well-known case, the throat of an apparently healthy nurse, under whose care a number of cases of diphtheria had developed, was found to contain numerous virulent bacilli which remained for weeks. In a case observed by Park, virulent bacilli were found for months in the nose of an apparently healthy infant, and this child communicated diphtheria, it was believed, to two other members of the family, without itself ever suffering from the disease. These cases are to be regarded as very exceptional. However, the presence of bacilli in the nose or throat of a child who has recently been exposed to diphtheria is very common. The New York Health Department made observations upon forty-eight children in fourteen families in which one or more cases of diphtheria had occurred, and where no attempt at isolation had been made. In one half these cases bacilli were found, and animal tests showed them to be virulent in every one of six cases tested, although four of the children did not develop diphtheria. Of the entire number, forty per cent subsequently developed diphtheria. My own experience in two institutions where diphtheria has been endemic, fully confirms the observation that bacilli of all degrees of virulence are very frequently found in the noses or throats of such exposed children, although a large proportion of them never develop the disease. Outside of institutions and infected tenement houses, however, such a condition is extremely rare.

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* An unfortunate term, as this bacillus has nothing to do with the form of angina classed as pseudo-diphtheria, which is generally due to the streptococcus.

Summary.—1. The discovery in the throat of a case of suspected diphtheria, of bacilli having the appearance of the Klebs-Loeffler bacillus, may be regarded as conclusive evidence of diphtheria.

2. Cultures may yield negative results late in pharyngeal cases, and often do early in laryngeal cases; but in no instance is a single negative culture to be regarded as conclusive.

3. Both the appearance of the throat and the stage of the disease should be considered in connection with the bacteriological report.

4. Virulent bacilli are frequently found in the noses or throats of children exposed to diphtheria, apart from all throat lesions. Such a finding is not in itself evidence that these persons have diphtheria, but, inasmuch as they may infect others and as a considerable proportion of them subsequently develop diphtheria themselves, they should be regarded with suspicion and if possible kept under observation.

5. Non-virulent bacilli are occasionally, and virulent bacilli are very rarely, found in the throats of healthy persons when there is no history of exposure to diphtheria.

6. The presence of diphtheria bacilli, associated with marked evidences of catarrhal inflammation of the mucous membrane, is evidence of diphtheritic infection.

Prognosis.—Many possibilities exist, and even the mildest case must be regarded as serious and carefully watched, since we can never know when unfavourable symptoms may develop.

The factors to be considered in the prognosis of any given case are: the age and previous condition of the patient; the extent of the membrane and the rapidity with which it is spreading; the degree of diphtheritic toxæmia as shown by the condition of the pulse and the nervous symptoms; whether or not the membrane has invaded the larynx; and the presence or absence of complications, especially nephritis and broncho-pneumonia; but of more importance than any or all these things is whether antitoxin is used and when it is administered.

The following figures are from the Report of the Health Department of Chicago of cases treated from October 5, 1895, to February 28, 1899:

		Died.	Mortality.
Injected 1st day	355	1	0·27 per cent.
" 2d day	1,018	17	1·67 "
" 3d day	1,509	57	3·77 "
" 4th day	720	82	11·39 "
" later	469	119	25·37 "
Totals	4,071	276	6·77 "

In all these cases the diagnosis of diphtheria was confirmed by cultures.

Diphtheria mortality is highest during the first two years of life, from its strong tendency to invade the larynx and lower air passages, and from the frequency with which broncho-pneumonia occurs as a complication. Those whose experience with this disease does not antedate the introduction of antitoxin can scarcely appreciate the results previously obtained. Of eighty-five consecutive cases under twenty-six months of age observed in the New York Infant Asylum, in a period extending over two years, the mortality was 68 per cent; in over two thirds of the fatal cases the disease involved the larynx. In diphtheria hospitals, where most of the mild cases included in the above statistics would probably not have been admitted, the mortality in children under two years formerly varied from 60 to 80 per cent; in private practice it ranged for this age from 30 to 60 per cent.

It can not be too often emphasised that the danger from diphtheria is not over when the throat has cleared. The most frequent causes of death after this time are broncho-pneumonia and cardiac or pneumogastric paralysis.

Prophylaxis.—In no infectious disease, smallpox alone excepted, can so much be accomplished in the way of prevention as in diphtheria.

Public funerals of children dying from diphtheria should invariably be prohibited. Schools should be closed whenever the disease is epidemic. Children from families where diphtheria exists should not be allowed to attend school, nor mingle in any way with other children, for the reasons that they may, while healthy, be the carriers of the disease; and, what is even more important, that they may be themselves suffering from diphtheria in an early stage or in a mild form.

In every large city, hospitals for diphtheria patients should be established, not only for the poor, but with private rooms for cases developing in hotels or other places where isolation is impossible. Every city should be provided with a steam disinfecting plant, where carpets, blankets, bedding, etc., can be sent from the sick-room for disinfection.

Quarantine.—Not only every undoubted case of diphtheria, but every suspected case, should be immediately isolated. Quarantine for the latter should continue until the diagnosis is settled either by a bacteriological examination or by the course of the disease. Positive and suspected cases should not be isolated together. The quarantine in every instance must be complete. If possible, cultures should be taken from the throats of all exposed children. Those containing diphtheria bacilli should be quarantined like cases of diphtheria, for they may be equally dangerous; they should use gargles and sprays, and the nose and throat should be closely watched.

Bacteriology has furnished some very definite data from which the necessary duration of the period of quarantine may be determined. In this the physician is to be guided by the time that the bacilli remain in the throat, for the patient is to be considered as dangerous while they persist. This point was investigated by the New York Health Department in 605 cases: In 304 of these the bacilli had disappeared by the third day after the membrane was gone; and in 301 they persisted for a longer time—in 176, for seven days; in 64, for twelve days; in 36, for fifteen days; in 12, for twenty-one days; in 4, for twenty-eight days; in 4, for thirty-five days; and in 2, for sixty-three days. Many of the cases in which the bacilli have persisted for an unusual time have been those of nasal diphtheria; in some of these it is doubtless owing to the fact that the nasal sinuses, especially the antrum, have been invaded. While it is unquestionably true that in a certain number of cases these persistent bacilli are non-virulent, the opposite has been frequently shown. Of 15 cases in which the virulence was tested, virulent bacilli were found in 9 at periods varying from eight to twenty-five days after the membrane was gone. Tobiesen found that of 46 patients leaving the hospital under ordinary rules, virulent bacilli were present in 24 at the time of their discharge. If no culture tests can be made, quarantine should be continued in mild cases for ten days, and in severe cases for three weeks, after the membrane has disappeared. The danger after this period in either instance is very slight; for even where virulent bacilli are found long after the membrane has disappeared, their number is usually small. The rules above given should be followed with children returning to school or mingling with other children, and adults who are thrown into close contact with children.

Treatment of suspected cases.—During an epidemic of diphtheria, especially in an institution, every sore throat and nasal discharge should be looked upon with suspicion, and isolated pending the result of a bacteriological examination, even though no membrane is present. All such patients should be separated from the other inmates of the home or institution, and while waiting for the results of the bacteriological examination or for positive symptoms, antiseptic gargles or sprays should be used. If there are patches on the tonsils or any other visible membrane, the case should be treated as true diphtheria, in order that no time may be lost. If the bacteriological examination shows the disease not to be true diphtheria, the patient may be released from quarantine in two or three days, provided the throat symptoms disappear. It is, of course, important that the conditions laid down with reference to bacteriological diagnosis shall have been fulfilled. Should symptoms continue, however, a second culture should be taken.

Immunisation of persons exposed.—When a case of diphtheria occurs in a family or an institution, every child that has been exposed should receive an immunising dose of antitoxin. This rule is not followed in practice as regularly as it ought to be. There is no doubt that for a limited time—from three to four weeks—the serum confers almost complete protection.

One need not hesitate to immunise persons of any age and in almost every condition, even newly born infants and pregnant women.

The dose for immunisation is from 100 to 1,000 units, the former being that required for an infant under one month, and the latter for a child of twelve or fourteen years; for one from two to ten years the usual dose is 700 units. If the exposure is continuous, as in an institution, the dose should be repeated every three or four weeks. A nurse in charge of a diphtheria case should receive 1,000 units.

Diphtheria so often complicates scarlet fever and measles, particularly in institutions and in hospitals for contagious diseases, that special consideration should be given to such patients. It is practically impossible by cultures to separate with absolute certainty all cases in which diphtheritic infection is present, from others; the only safe rule is to immunise every child admitted to a scarlet-fever or measles hospital, and in institution epidemics of either of these diseases to immunise every child attacked. This rule has been followed for some years at the New York Foundling Hospital with the most striking benefit.

Nurses and physicians.—As diphtheria is contracted, not from the breath of the patient or the air of the room, but by receiving the bacilli into the mouth or air passages, all possible means should be taken to destroy the bacilli discharged, and to secure absolute cleanliness in everything about the sick-room. Nurses should never be allowed to eat or sleep in the sick-room, and an antiseptic gargle should be used four or five times a day. The hands should be kept clean, and only such dresses worn as can be readily washed and disinfected. It is the nurse who is most likely to contract the disease, on account of the continued exposure.

The physician should take the same precautions as in scarlet fever. A pocket tongue-depressor should not be used for the examination of the throat, but a spoon which is kept in a solution of carbolic acid, 1 to 40.

The sick-room.—The carpets, hangings, upholstered furniture, everything in fact not necessary for the patient's welfare, should be removed, especially books, toys, cushions, etc. The room should be a large one, if possible with an open fireplace, well ventilated, and fresh air should be allowed in abundance. The floor should be washed once a day with a solution of bichloride, 1 to 2,000, and dusted often with cloths moistened in the same solution. All handkerchiefs, bed-linen, and clothing removed from the patient should be treated as in a case of scarlet fever. Pieces of membrane and other matters discharged from the patient should be put into a solution of carbolic acid, 1 to 20, or of bichloride, 1 to 1,000. Old muslin or absorbent cotton should be used to cleanse the nose and mouth of the patient and burned immediately. All vessels for the reception of expectoration or other discharges should contain bichloride, 1 to 2,000. The bed-linen should be very frequently changed, and everything kept scrupulously clean. In the room should be a large bowl of carbolic acid, 1 to 40, or some similar solution for cleansing the hands, and a tray of the carbolic solution for spoons, syringes, or other things used in the treatment of the patient. All spoons, cups, or other dishes used by the patient should be carefully sterilised by boiling. No milk or other food should be allowed to stand about the room. There is no objection to the hanging of sheets moistened in carbolic, bichloride, or other disinfectant solutions before the door, but neither this nor hanging them about in the sick-room is to be regarded as having any value in disinfecting the air of the room. They create a false sense of security, and often lead to the neglect of thorough cleanliness.

Disinfection of apartments after an attack should be done as after scarlet fever.

Treatment.—*General measures.*—It is important in every case that there should be plenty of fresh air in the room throughout the attack. Where it is possible, it is desirable to have two rooms for the patient, so that he can be changed from one to the other every day, giving time for thorough cleanliness and airing. Hospital patients should never have less than 1,000 cubic feet of air space, and if possible 1,200 should be allowed. Even in mild cases the patient should be kept in bed throughout the entire attack, and in severe cases this should be continued for some time during convalescence.

Nursing infants may be fed on breast milk obtained by a breast pump, but should not be put to the mother's breast. The feeding of older children should be managed very much as in other cases of severe illness. Milk is the main reliance; it should usually be diluted, and for younger infants partially peptonised. The greatest difficulty in feeding is seen in the latter part of the disease, when the patients are septic and have a strong aversion to food, when vomiting is easily excited and when swallowing is difficult on account of the swelling and pain. It is then that gavage (page 64) is most valuable. This is much more successful with children under three years old than is rectal feeding. In older children the tube may be passed through the nose.

Stimulants.—These should be begun as soon as the depressing effects of the poison of diphtheria are shown upon the pulse and general condition of the patient. In most cases, therefore, they are not needed until the third or fourth day; in a few they may be required from the outset, and in some they may not be required at all. The indications for alcoholic stimulants are marked prostration, a feeble pulse, and a weak first sound of the heart. In regard to the quantity, half an ounce of whisky or brandy in twenty-four hours is enough to begin with, for a child four years old. This should be diluted with at least eight parts of water. In very severe cases two or three times as much may be given; but more than this, except for a short period, is seldom wise. The excessive doses often used surely endanger the kidneys. The method of administration should be the same as in other severe acute diseases (page 51). Other heart stimulants than alcohol, though inferior to it, are of value. Probably the most useful one is strychnine, which should be given as in pneumonia. Camphor and carbonate of ammonia are valuable for rapid effect in syncopal attacks, and digitalis in other cases where the pulse is weak and arterial tension low, but it is not wise to give it in large doses. In cases of threatened heart paralysis occurring late in the disease or during convalescence, morphine and strychnine may be used hypodermically. Full doses must be given and repeated every two to four hours, so that the child may be kept under their influence.

Except for stimulation or the control of special symptoms such as vomiting or diarrhœa, all internal medication should be omitted; for there is yet wanting proof that drugs influence the course or the result of the disease.

Local treatment.—Since the introduction of antitoxin, opinion has undergone a decided change with reference to local treatment. While it should not be entirely abandoned, still it is of secondary importance; and under conditions where it can be carried out only with great difficulty and the use of force it is often wise not to attempt it systematically.

The purpose of local treatment, it is now generally agreed, should be cleanliness, and not the destruction of bacilli. Cleanliness of the nose, mouth, and pharynx is important, inasmuch as one of the chief dangers of the disease is the aspiration of bacteria contained in the abundant secretions of these parts, into the larynx and bronchi. Our aim should therefore be to keep the parts as clean as possible without too severely taxing the strength of the child.

For cleansing the nose and pharynx only syringing can be depended upon. Nasal syringing is indicated when there is much nasal discharge, whether membrane is visible in the anterior nares or not. In septic cases with a profuse fetid discharge it may be necessary to syringe the nose, no matter how strongly the child resists. Whether it shall be done, will depend upon the condition of the patient's strength and his pulse. The purpose in syringing is not so much to clear the nose, from which absorption is slow and imperfect, as to flush the rhino-pharynx, from which absorption is always very active. Only bland solutions should be employed, such as a common-salt solution, one per cent, or a boric-acid solution, one to four per cent strength. For some cases, the piston syringe and the method described on page 59 may be used; but for most cases a fountain syringe possesses manifest advantages, and it is rather more convenient for hospital purposes. Irrigation of the pharynx is best done with the fountain syringe, and is of especial value where there is much swelling or abundant discharge. All solutions should be used as warm as can be borne, and in sufficient quantity to irrigate the parts thoroughly, a few such irrigations being much better than a great many partial ones. By a skilful nurse syringing can in most cases be done with comparatively little disturbance to the child.

Slight nasal hæmorrhages may necessitate less frequent syringing, and a free hæmorrhage may require it to be discontinued. Astringent solutions of alum, supra-renal extract, lemon juice, etc., are often beneficial in such cases, but they must be used carefully. In children who are old enough gargles should be used. A solution of boric acid, listerine, or Dobell's or Seiler's solution much diluted, may be employed.

In cases with a moderate nasal discharge it is usually sufficient to syringe three or four times a day; but in severe septic cases, with very abundant discharge, syringing should be repeated as often as every two hours during the day and every four hours at night.

External applications to the throat have practicaly no effect upon the disease, but are often useful to relieve pain and tension in the swollen lymph-glands. Poultices should never be employed. As a continuous application, only cold is to be advised, generally by means of an ice bag well protected to prevent wetting the clothing.

The treatment of pneumogastric and other forms of post diphtheritic paralysis has been considered in the chapter on Multiple Neuritis.

The Serum Treatment.—This has been the outcome of a long series of experiments in which many men have had a share; but it is to Behring pre-eminently that the credit belongs for the development of the principles of serum-therapy.

Regarding the nature of the antitoxin and its mode of action much is as yet unknown. It is produced by the cells of the body under the stimulus of the diphtheria toxin. It is intimately combined with the globulin of the blood, and is itself possibly a globulin. The action of the antitoxin is two-fold: it directly neutralises the toxin produced by the diphtheria bacillus which is present in the blood; it also has some effect upon the bacilli themselves the nature of which is not understood, but it induces a condition in the blood which inhibits the growth of the bacilli, and thus arrests the membranous inflammation which the bacilli excite.

Properly prepared, it will keep without deterioration for from three to six months; but after one year it loses somewhat its antitoxic properties. It should be kept in a cool, dark place, and after a bottle has been opened it should be used within a few days. Antitoxin is now prepared in a dry form, which is to be preferred only when it must be kept for a very long time.

The strength of the serum is measured in antitoxin units, the unit being an arbitrary one, viz., the amount of antitoxin which will protect a guinea-pig weighing 250 to 300 grams against one hundred times the fatal dose of diphtheria toxin. The improvements in the production of the serum have thus far consisted in increasing its strength. Behring's serum first used contained but one unit in each cubic centimetre. At present there can be obtained from most manufacturers a serum containing 500 antitoxin units in each cubic centimetre. This concentration is of immense advantage and has to a large degree done away with the unpleasant symptoms, such as pain, localised œdema, etc., which were formerly so frequent.

Method of administration and dosage.—In selecting an antitoxin syringe one should be chosen which holds at least 5 c. c., which can readily be disinfected by boiling and whose needles are not too large. The smallest needle through which the serum will flow is the best. Before making the injection, the skin should be thoroughly cleansed with alcohol; the needle should invariably be boiled and the whole syringe either boiled or rinsed with alcohol. Care should be taken to see that all air is expelled from the syringe. The seat of injection is not a matter of great importance; my own preference is for the cellular tissue of the abdomen. After the injection is made the puncture should be covered by adhesive plaster.

The dose of antitoxin required in a given case is always somewhat problematical. It is desirable to give enough to neutralise the diphtheria toxin present in the blood, and that is always an unknown quantity, depending upon the stage of the disease, the severity of the attack, the extent of the membrane, and to some degree upon the age of the patient. Convinced now of the essential harmlessness of the serum, the tendency everywhere has been to use larger and larger doses, a practice which has been fully justified by the results obtained. For a child over two years old an initial dose for a severe attack, including all laryngeal cases, should not be less than 7,000 or 8,000 units; repeated in from six to eight hours, provided no improvement is seen. Children under two years should receive from 5,000 to 6,000 units. Cases of exceptional severity, in older children, should receive from 10,000 to 15,000 units, to be repeated in from six to eight hours if the progress of the disease is unfavourable. Mild cases should receive from 3,000 to 5,000 units as an initial dose, a second being rarely required.

In cases receiving antitoxin late, even though the symptoms may not seem particularly severe, the dose should be increased in proportion to the length of the illness—i. e., if three days ill, three times the ordinary dose should be given.

Only serum from a trustworthy manufacturer should ever be used. The sera chiefly used in this country are those of the New York Health Department, Mulford & Company, and Parke, Davis & Co., all of which, I believe, are reliable. The most concentrated serum which can be obtained should be selected.

All experience shows that the results are greatly modified by the time of its administration. The serum can not undo the serious damage already done to the cells of the body, and this at the time of injection may be so great that death will result. In very mild cases, with older children, one may wait for the result of a bacteriological examination, but never in a severe case and never in a young child. In the group of severe cases should be placed every one which at the first visit shows a pharyngeal exudate covering more than the tonsils, also all cases with symptoms of laryngeal invasion, and all with an exudate on the pharynx and a profuse nasal discharge. If in a doubtful case twelve hours' observation shows that the membrane has spread from its original seat, no further delay is admissible. Experiments have shown that after a fatal dose of diphtheria toxin, an animal can usually be rescued if the antitoxin is administered within forty-eight hours, but rarely after that time. In human diphtheria marked benefit usually follows injections made as late as the third day; but after this time the value of the serum diminishes very rapidly, and although striking examples of benefit are sometimes seen after later injections, they can not be depended upon. On the other hand, in very severe or in malignant cases irreparable harm may be done during the first twenty-four hours of the attack.

The effect upon the diphtheritic membrane is usually noticeable within twenty-four and often in twelve hours; it first stops spreading, and soon begins to soften and loosen. The swelling of the mucous membrane subsides and the local disease abates, very much as when the disease runs its usual course. The striking thing after the use of antitoxin is the rapidity with which these changes take place, and the abrupt transition from an advancing to a retrograde process. The subsidence of the inflammatory conditions in the larynx and trachea is quite as marked as in the pharynx. The symptoms of stenosis, even when severe, often diminish in a few hours, making operation unnecessary in a very large number of cases where previously it seemed inevitable. The membrane loosens rapidly in the larynx and trachea, sometimes necessitating the frequent removal of the intubation tube, where operation has been performed. Improvement is also shown by the cessation of the nasal discharge, the re-establishment of nasal respiration, and the diminution in the swelling of the glands of the neck.

The effect upon the constitutional symptoms is not less striking. In favourable cases there is seen, often in twelve hours, a fall in temperature and improvement in the pulse and in the nervous symptoms.

The limitations of antitoxin.—It is important that these should always be kept in mind. The serum must be given early, for if given late it can not undo the mischief already done by the diphtheria toxin.

Cases of great severity often pass the period when recovery is possible, before the antitoxin is given. This period may in some cases be four days, in others it may be less than twenty-four hours. The tissues most susceptible to the diphtheria toxin are probably those of the nervous system, the heart, and the kidneys; and the consequences of its action may be seen in the production of nephritis, in heart failure at the height of the disease, or in later paralysis of the heart, respiration, or voluntary muscles, in spite of the fact that antitoxin is given at a period early enough to avert death from local disease in the larynx or bronchi. Again, antitoxin is of no value in cases of streptococcus septicæmia. The early arrest of the inflammation excited by the diphtheria bacillus is unfavourable to the spread of streptococcus infection, yet sometimes the latter gains such headway or is of such intensity as to involve almost the entire body. Against the phlegmonous inflammation of the throat or the cellular tissue of the neck, broncho-pneumonia, and nephritis, antitoxin is powerless; and just in proportion to the severity of these inflammations are negative results seen.

Eruptions and other unpleasant effects.—Some transient œdema usually follows the injection. In a few hours there may be seen a general erythema; this, however, is rare and usually of short duration. The most important eruptions are seen between the eighth and fourteenth days. They follow from ten to twenty per cent of the injections made, and appear to be quite independent of the amount of serum used. The exact cause is not known. The most common eruption is urticaria. This is often intense, very annoying, and may nearly cover the body. It may be accompanied by a slight rise of temperature; it usually lasts for two or three days, however, it is rarely severe for more than twenty-four hours. Various forms of erythema are occasionally met with. In two or three instances I have seen hæmorrhagic eruptions, generally in the neighbourhood of the large joints, and always in children suffering from extreme malnutrition. In a few cases a moderate swelling of some of the joints has been observed, and very exceptionally a transient albuminuria. One occasionally meets with patients who seem unusually susceptible to serum injections, and in whom even small immunising doses cause headache, muscular pains and general malaise so that they feel quite wretched for several days. All of the above symptoms except the urticaria are rare, and should not for an instant deter one from using antitoxin when indicated.

Real and alleged dangers from antitoxin injections.—In the few cases where sudden death has followed antitoxin injections, the evidence that antitoxin was the cause of death is not conclusive. In some of these patients the autopsy has revealed a status lymphaticus not before suspected. In this condition the shock of so slight a thing as a needle puncture might produce death.

That so very few alleged instances of serious harmful results have occurred among the great numbers of injections which have now been made, is sufficient to establish the fact that the serum itself is essentially harmless.

The unfavourable effects upon the heart, the kidneys, and the blood, attributed to antitoxin, have not been proved. In a disease like diphtheria, where the heart and kidneys are often and seriously affected, and where cardiac and renal symptoms in many cases are suddenly manifested, it is impossible to say, even when such symptoms follow the injection of serum, that they are not due to the original disease. They were seen with great frequency before antitoxin was known. Observations regarding the effect of the serum upon the blood were made by Billings, upon twenty-nine cases of diphtheria. He found the reduction both in the hæmoglobin and the red cells to be much less than the average found in cases of diphtheria of similar severity not treated by the serum.

At the present time, no evidence has been adduced as to the danger or injurious effects of diphtheria antitoxin which should deter any one from its use. Those which have been reported are to be regarded in the light of accidents for which the antitoxin itself can not be held responsible.

Results with antitoxin treatment.—Since 1895 the serum has been tested on so extensive a scale as the prevalence of diphtheria all over the world has made possible with results so uniformly good that it seems quite unnecessary any longer to cite statistics in proof of the value of this remedy. No tables of figures are so convincing to an individual as personal experience, and by this argument one by one the opponents of antitoxin have been converted.

The beneficial effects of the remedy may be summed up in the following statements: (1) The percentage mortality from diphtheria in hospitals both in Europe and in America has been reduced to a little more than one third the previous figure; (2) the proportion of cases now requiring operation for laryngeal stenosis has been reduced to about one half; (3) the mortality after tracheotomy has been reduced to one half, and that after intubation to about one third the former figure; (4) but even more convincing is the effect of the serum treatment upon the actual diphtheria mortality of cities and countries where it has been used.

In the first of the subjoined tables is given for a period of years the actual number of reported deaths from diphtheria and membranous croup, irrespective of the growth in populaton; in the second one the number of deaths in each 10,000 of population. These figures can not be open to the question which is sometimes raised concerning percentage mortality statistics.

Table Showing Annual Deaths from Diphtheria and Croup, 1887 to 1900 (inclusive).

	1887	1888	1889	1890	1891	1892	1893	1894	1895	1896	1897	1898	1899	1900
London...........	1,579	1,812	2,075	1,877	1,174	2,182	3,484	2,861	2,479	2,793	2,328	1,842	2,041	1,558
Berlin	1,392	1,195	1,210	1,601	1,106	1,342	1,637	1,416	987	559	546	664	655	563
Paris	1,585	1,729	1,706	1,659	1,361	1,403	1,266	1,009	435	444	268	256	336	291
New York....... (Manhattan and Bronx)	3,056	2,553	2,291	1,783	1,970	2,106	2,558	2,870	1,976	1,763	1,591	843	960	1,121
Chicago	1,405	1,297	1,509	1,261	1,358	1,548	1,467	1,406	1,632	1,098	774	680	917	797
Boston...........	410	589	683	462	285	481	546	878	654	572	456	185	304	537
Philadelphia.....	858	523	727	748	1,362	1,707	1,238	1,452	1,398	1,201	1,514	1,154	997	1,064
Brooklyn........	1,453	1,885	1,467	1,283	1,180	1,137	878	1,660	1,454	1,310	998	745	744	673
Denver	68	120	109	277	175	89	106	71	40	19	43	34	31	14
Germany.... ... (266 towns over 15,000)	10,970	10,142	11,919	11,915	10,484	12,365	16,557	13,790	7,611	6,262	5,208	5,220	5,111	4,685
New York State.	6,490	6,710	5,930	4,954	4,844	5,970	5,942	6,616	5,696	4,640	4,115	2,612	2,786	3,306
Massachusetts...	1,628	1,831	2,214	1,626	1,218	1,455	1,394	1,801	1,784	1,677	1,426	706	*1,047	†1,475

* Cases reported 1899, 7,134. † Cases reported 1900, 12,641.

Table Showing Average Annual Deaths from Diphtheria and Croup per 10,000 of Population.

London,	before antitoxin,	1887–'93,	4·8;	since antitoxin, 1896–1900,	4·7
Berlin,	"	"	10·2;	" "	3·7
Paris,	"	"	6·5;	" "	1·3
New York,	"	"	14·5;	" "	6·3
Chicago,	"	"	13·1;	" "	5·0
Denver,	"	"	12·9;	" "	1·7
Philadelphia,	"	1890–'94,	11·9;	" "	9·6

Some explanation of these figures is necessary that they may be fully appreciated. The great reduction in the death-rate is seen only in those cities and countries where the serum treatment has been widely employed. Nowhere in Europe is this true to the same degree as in Paris, Berlin, and Germany generally; and probably nowhere in Europe has it been so little used and so slow in gaining favour as in London. In our American cities the effect of the serum treatment upon municipal mortality figures has been directly proportionate to the extent to which the health departments have believed in its efficacy and encouraged its use by furnishing it free to the poor, and sending their own inspectors to administer it. This is true particularly of New York and Chicago; in Philadelphia, on the contrary, the authorities for a long time were openly opposed to the serum treatment.

Summary.—1. Behring's antitoxin is a specific remedy for experimental diphtheria in animals.

2. Experience is now sufficient to justify the statement that it is so in man, and just to the extent in which we can fulfil the conditions which are essential in experimental diphtheria.

3. These conditions are, that the serum must be administered early, that the dose be adequate, and the case be one of pure diphtheria.

4. Experience shows the serum to be much less efficacious in cases of so-called mixed infection or septic diphtheria, and that it is valueless in membranous inflammations which are due to streptococci—i. e., pseudodiphtheria.

5. The serum itself is essentially harmless both when injected in healthy persons for immunization, or in those suffering from diphtheria.

6. In a young child the serum should be injected upon a clinical diagnosis of diphtheria without waiting for bacteriological confirmation; in older children one may wait in a mild case, but never in a severe one, particularly a laryngeal case.

7. For all patients, but especially for young children, the most concentrated preparations of antitoxin that can be obtained should be employed.

8. The actual mortality from diphtheria (including membranous croup) has been reduced in those cities and countries where it has been generally adopted by nearly 50 per cent; the mortality of intubated cases has fallen from 70 to less than 30 per cent; of tracheotomized cases from 60 to 33 per cent; the proportion of cases in which operation is required has been reduced fully 50 per cent.

9. The evidence is conclusive that in laryngeal diphtheria the serum in sufficient doses largely prevents the extension of the membrane into the trachea and bronchi, thus preventing broncho-pneumonia.

10. It is not yet possible to state to what extent the heart, the kidneys, and the nervous system are protected by the serum. It is, however, certain that such results can not be depended upon unless injections are made early and full doses given.

11. For a period of from three to four weeks the protection conferred by immunization is practically complete. The serum should therefore be given to every child in an infected household or institution.

12. Gratifying as were the earlier results with the serum treatment, they have been constantly improving, and there is every reason to believe that, with larger experience both in its preparation and its use, still better results will yet be reached. Certainly there is no remedy for any disease that has more testimony in its favour than has antitoxin for diphtheria.

Convalescence.—After a severe attack of diphtheria convalescence is always slow on account of the anæmia and the depressing effects of the disease. Patients should invariably be kept in bed for at least a week after the throat has cleared, and longer if any tendency to cardiac weakness is seen. The pulse should be carefully watched, and irregularity, intermission, dicrotism, or a weak first sound of the heart, should make one apprehensive. An abnormally slow pulse is generally more serious than one which is rapid. Under such circumstances the patient should be kept recumbent and absolutely quiet, since sudden and even fatal syncope may be the result of a violation of these rules.

The extreme degree of anæmia requires that iron be given for a considerable time during convalescence, to be followed by cod-liver oil, wine, and other tonics.

Great difficulty is occasionally experienced in getting rid of the bacilli in the throat. The tonsillar crypts, the adenoid tissue of the rhinopharynx, and the nasal sinuses are the places where the bacilli are most likely to remain. Inasmuch as it is now generally made a condition of release from quarantine that the throat shall have been shown by cultures to be free from bacilli, this becomes a matter of much importance. The most efficient means appears to be to syringe the nose gently three or four times daily with a solution of bichloride, 1 to 10,000, to which one eighth glycerin has been added, and to use the same solution as a gargle. For children under four years old a simple salt solution, or a dilute Dobell's solution, should be substituted and the gargle omitted.

PSEUDO-DIPHTHERIA.

Synonyms: False diphtheria, streptococcus diphtheria, scarlatinal diphtheria, diphtheroid inflammation, croupous tonsillitis.

At the present time there are included under the term pseudo-diphtheria all inflammations of the throat and upper air passages characterized by the production of a false membrane, in which the Klebs-Loeffler bacillus is not found. When these inflammations are primary they are rarely serious; but when they complicate scarlet fever or measles they may be very severe, and frequently prove fatal.

Frequency.—Numerical statements regarding the relative frequency of this disease and true diphtheria signify very little, because of the variable conditions under which observations have been made. From the investigations of Park, Baginsky, Martin, Morse, and others, it would appear that in from twenty-five to thirty-five per cent of the cases formerly sent to hospitals with a clinical diagnosis of diphtheria, the disease was pseudo-diphtheria. Most of these were mild, and were regarded by many physicians as simply cases of tonsillitis, the exceptions being those which were secondary to scarlet fever or measles.

Of the membranous inflammations occurring in the diseases just mentioned, the great majority are examples of pseudo-diphtheria. Of seven cases of membranous angina in measles and three in scarlet fever, studied by Prudden, all were proved to be pseudo-diphtheria; of nineteen occurring with scarlatina, studied by Park, only two were found to be true diphtheria; and of sixteen occurring with scarlet fever and three with measles, studied by Booker, none was true diphtheria. In 1,000 cases of scarlatina observed by McCollom, only twelve per cent of those showing distinct membrane in the throat were true diphtheria. It has been the general experience of all writers that when it complicates other diseases, pseudo-diphtheria nearly always occurs at the height of the primary disease, while true diphtheria may occur at any time, even during convalescence.

Etiology.—As was first shown by Prudden in 1888, and abundantly confirmed by others since that time, this inflammation is usually due to the streptococcus; it may be found alone, or associated with the staphylococcus aureus or albus, and occasionally the staphylococcus may be found alone.

The streptococcus is very frequently found in the throats of healthy children, particularly in winter and in cities, and more often in those who live in tenements or who are inmates of hospitals or other institutions. The local conditions in the mucous membranes during an attack of measles, scarlet fever, and other infectious diseases, are especially favourable for the development of these germs, which at such times are very often present in great numbers even when no membrane is seen.

This form of sore throat is more apt to occur in houses with bad drainage and other unsanitary conditions. From the fact that the streptococcus is so widely distributed, attacks of pseudo-diphtheria may occur in any place and at any time, irrespective of epidemic influences or even the occurrence of other cases.

To what degree these cases are to be regarded as communicable, and what precautions regarding isolation and disinfection are required, are questions of much importance. The most extensive investigations upon these points are those made by the New York Health Department. As a result of observations upon 450 cases which were followed, the conclusion was reached that the disease was so slightly contagious, if at all, and usually so mild, that strict isolation and subsequent disinfection were unnecessary. Of 113 cases occurring in 100 families, in only 14 was there a history of exposure to a similar case; and in only 9 was there another case in the same family. In many of the latter, a common origin appeared more probable than that one case was derived from another.

They are probably more contagious in the presence of the poison of scarlet fever or measles.

Lesions.—In the primary cases the membrane is generally confined to the tonsils or is chiefly there, there being only small deposits elsewhere. In the secondary cases, the entire pharynx may be covered and the disease may extend to the nose, the mouth, the middle ear, and occasionally to the larynx, trachea, and bronchi.

The structure of the membrane resembles that of true diphtheria, and it is impossible by a microscopical examination alone always to separate the two diseases. In many cases the membrane is softer, more friable, and contains a relatively larger number of cells than does that of true diphtheria, but the structure of the latter varies so much that it is not safe to draw any positive conclusions.

In the mild cases the inflammation of the mucous membrane is a superficial one and the pseudo-membrane is not very adherent. In the severe cases, chiefly the secondary ones, the process extends much deeper. Besides the pseudo-membrane upon the surface there is intense congestion, œdema, and cell-infiltration of all the lymphoid and cellular tissue of the pharynx. It affects the tonsils, soft palate, uvula, epiglottis, adenoid tissue of the vault and the entire pharyngeal ring, and also extends to the external lymph nodes and surrounding cellular tissue. The process both in the throat and externally in the neck may terminate in resolution, suppuration, or in necrosis.

The streptococci are found in the false membrane, in the underlying mucous membrane, in the lymph spaces and in the lymph nodes. In the most severe cases there are present the lesions of a general streptococcus infection. The blood swarms with these germs, and they may set up inflammations in any of the organs, but especially in the lungs and the kidneys, less frequently in the serous membranes. Small foci of suppuration may be found in any of the viscera.

Symptoms.—1. *The primary cases.*—The onset is usually abrupt, with well-marked symptoms: there are frequently chilly sensations, headache, vomiting, general pains, and in most cases the child complains of soreness of the throat and pain on swallowing. There are first seen a general redness and swelling of the tonsils, sometimes of the entire pharynx; shortly afterward membranous patches appear upon the tonsils. These vary greatly in appearance. In colour they are yellow or gray, often changing later to a dirty-olive tint. **(Plate XVIII, c.)** The membrane seems loosely attached and can frequently be wiped off with a swab. It is soft and friable, very rarely thick, firm, or tenacious. It is often irregular in its outline, which is not sharply defined. The membrane usually remains but three or four days and disappears rapidly. As a rule, it is limited to the tonsils, and does not spread after it first forms. Occasionally, however, small patches are also seen upon the fauces or the pharynx. The œdema and other evidences of inflammation in the throat are usually more marked than in true diphtheria, and the swelling of the lymph nodes behind the jaw is slight. The constitutional symptoms are generally more severe during the first two days, and the temperature may be 103° or 104° F., but by the third day it falls, and most of the symptoms subside. It is rare for the disease to extend either to the nose or the larynx. Generally there are no complications and no sequelæ.

2. *The secondary cases.*—Some of these are mild, and do not differ from those just described, but most of the severe cases are included in this group. The clinical picture of the latter is that of *scarlatina angi-*

nosa, as given by the older writers, and it does not differ in any essential particulars from the septic form of true diphtheria (page 1035). The local symptoms are those of severe pharyngeal diphtheria, and the constitutional symptoms those of septicæmia.

When the disease complicates scarlet fever, the symptoms may precede the eruption, but they usually begin at the height of the primary fever—i. e., from the second to the fourth day—and gradually increase in severity, reaching their maximum from the fifth to the eighth day of the disease. In measles the throat symptoms are somewhat later; they begin at the height of the primary fever, and often increase while the eruption fades. In most of the severe scarlatinal cases the disease involves the nose and the middle ear. In measles both these complications are less frequent, but there is a much greater tendency to involve the larynx, and if the larynx in a young child, the process is almost invariably complicated by broncho-pneumonia. In some cases the larynx is invaded when there is no membrane in the pharynx; but this is very infrequent unless the disease is true diphtheria. Catarrhal laryngitis in a young child may produce symptoms which are practically identical with those of the membranous form, and there is little doubt that many cases complicating measles in which the latter diagnosis is made are really examples of catarrhal laryngitis, particularly if no membrane is visible in the throat.

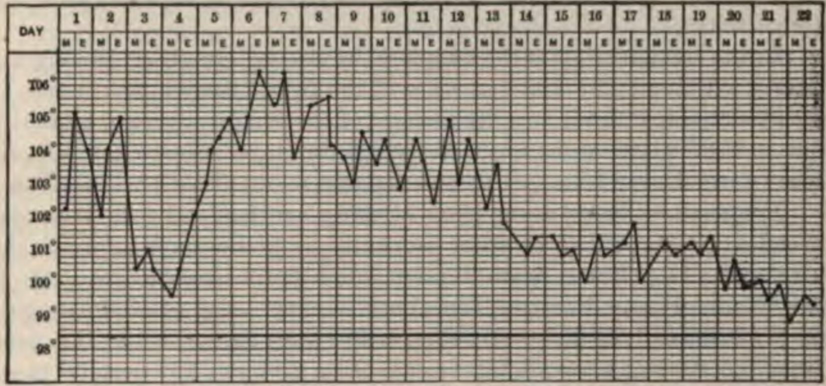

FIG. 203.—Pseudo-diphtheria following measles. The chart begins at the time of the full eruption in a severe case of measles. On third day temperature fell, with fading eruption, and child seemed convalescent. With secondary rise in temperature, the tonsils, which before had been only red, showed membranous patches, the exudation rapidly spreading until the entire pharynx was covered; throat symptoms very severe, with great swelling of cervical glands, but the membrane did not extend beyond the pharynx. From sixth to twelfth day a most profound septicæmia, so that life was despaired of. The patient was a vigorous child, and, escaping both nephritis and pneumonia, made a good recovery. Convalescence quite rapid; no sequelæ. Repeated cultures were made from the throat, but all showed only streptococci. Patient a girl four years old. Case observed in private practice.

Secondary cases as a class are characterized by high temperature (Fig. 203), rapid, feeble pulse, great prostration, and delirium, apathy or stupor, and often albuminuria. In fatal cases death usually occurs at the height of the disease, from asthenia, broncho-pneumonia, or nephritis, sometimes from laryngitis. If none of these complications develop, patients may withstand the toxic symptoms even when they are very severe. If the attack terminates in recovery, the local disease follows very much the same course as in diphtheria. The subsequent anæmia is, however, less severe, and none of the dangers of convalescence connected with cardiac or respiratory paralysis are present.

There may be in connection with the local process in the throat, deep sloughing of the tonsils or adjacent structures, suppuration of the lymphatic glands or in the cellular tissue of the neck, occasionally followed by serious hæmorrhage. However, these complications are rare, and if the patient survives the danger of the acute stage of the disease, he usually recovers.

Diagnosis.—The clinical features which distinguish pseudo-diphtheria from true diphtheria have already been considered (page 1040). It is impossible in any case to be certain of the diagnosis except by cultures; for, although by clinical symptoms alone one may in the great majority of cases be certain that a given case is one of true diphtheria, to say that any membranous inflammation of the throat is not diphtheria is impossible. The bacteriologists have taught us to be cautious in pronouncing too positively upon even mild cases, as it has been shown that some of them may be caused by most virulent diphtheria bacilli (page 1031).

In the secondary cases the diagnosis by clinical symptoms is more accurate. A membrane which appears in the throat early in the course of measles or scarlet fever, or at the height of the primary disease, is usually due to the streptococcus; while one which develops late or after the primary fever has subsided, is frequently due to the diphtheria bacillus.

Prognosis.—There is no more striking contrast between true and pseudo-diphtheria than in their mortality when they are seen side by side. Of 117 primary cases of pseudo-diphtheria observed by Park in the Willard Parker Hospital, New York, the mortality was 3·5 per cent; of 127 cases of true diphtheria seen in the same institution at the same time, the mortality was 34·5 per cent. In a group of 154 hospital cases reported by Baginsky, there were 118 of true diphtheria, with a mortality of 38·2 per cent, and 34 cases of primary pseudo-diphtheria, with a mortality of 5·5 per cent. From the same hospital, Philip has published a report upon 376 cases: 332 of these were true diphtheria, with a mortality of 37 per cent; 31 were cases of primary pseudo-diphtheria, with no mortality. The Bulletin of the New York Health Department contains a report upon 324 cases of pseudo-diphtheria in children, with a mortality of 9, or 2·8 per cent; 4 of the fatal cases complicated scarlet fever; of the primary cases, the mortality was but 1·5 per cent. These were not hospital cases. From the above data the deduction seems warranted that in a child previously healthy, primary pseudo-diphtheria is not a serious disease.

Turning now to the secondary cases, we find very different conditions. From the best available statistics it would appear that the usual mortality of pseudo-diphtheria, when it is secondary to scarlet fever and measles, is from 15 to 20 per cent. However, when these diseases prevail epidemically in institutions, the mortality is often higher than this.

Prophylaxis.—In primary cases strict quarantine is unnecessary after the question of diagnosis has been settled. Cases of pseudo-diphtheria occurring in measles or scarlet fever should certainly be separated from uncomplicated cases. By way of prevention, something can be done in these diseases by keeping both nose and throat as clean as possible during severe attacks by the use of an antiseptic mouth-wash or gargle, and a nasal spray. For young children only weak solutions should be employed, such as a diluted Dobell's or Seiler's solution, 1: 10,000 bichloride, or a one-per-cent solution of boric acid.

Treatment.—Every child with a membranous patch on its throat requires close watching; if under three years old diphtheria antitoxin should be administered, pending the result of a bacteriological examination. In all cases with doubtful diagnosis this should invariably be done. The primary cases require only the treatment of an attack of tonsillitis.

In the secondary cases local treatment should be begun with the appearance of the first patch upon the tonsils. In mild cases the use of gargles and antiseptic throat sprays is sufficient. In the severe cases local treatment should be thorough and energetic, but not repeated too frequently. It is seldom necessary to disturb a very sick child for local treatment oftener than every two hours by day and every four hours by night. The nose should be syringed with warm, bland solutions but not too forcibly. For the pharynx stronger solutions may be employed as hot as can be borne. In order to clear the secretions from behind the swollen tonsils a short piece of a soft catheter may be attached to the tip of the syringe, which should be inserted well back behind the molar teeth. Where the swelling and œdema are great, benefit may result from frequent spraying with solutions containing supra-renal extract, also from inhaling hot vapour impregnated with eucalyptol, benzoin, etc. For a local germicidal effect swabbing is most reliable; strong solutions should be used but not frequently repeated—e. g., 1: 500 bichloride of mercury or a 10-per-cent solution of nitrate of silver, from one to three times a day. As an external application nothing is so beneficial as the ice-bag, which, whenever there is great adenitis and cellulitis, should be constantly used covered with thin flannel, and kept well up against the throat by a four-tailed bandage.

The general management of these cases as to feeding, stimulants, etc., is the same as in diphtheria. Aside from stimulants no internal medication should be attempted with young children. Those who are older may take with advantage tr. ferri chlor., gtt. v to x, with glycerin, every three or four hours. The use of streptococcus antitoxin in these cases has thus far been attended with very little success, and can not yet be recommended.

CHAPTER IX.

TYPHOID FEVER.

TYPHOID FEVER is an acute infectious disease due to a specific germ —Eberth's bacillus. It may affect the fœtus *in utero*, or the newly born child, and it is seen in infancy and throughout childhood.

Fœtal typhoid.—Morse [*] (Boston) has collected the evidence bearing upon fœtal infection, from which the following conclusions seem warranted: Infection of the child from the mother is a frequent but not an invariable occurrence. The bacilli may pass directly from the maternal into the fœtal circulation. The fœtal form of the disease is a general blood-infection, since the intestines are not functionally active. The most common result is death of the fœtus and consequent abortion; but the child may be born alive still suffering from infection, and die in a short time because of its feeble resistance. Whether a fœtus may recover completely and be born alive and well, is not yet established.

Infantile typhoid.—Much difference of opinion exists regarding the frequency with which typhoid fever occurs in infancy. Some clinicians hold the opinion that the disease is of very common occurrence, but is often unrecognised because of the absence of many of the symptoms which are characteristic at a later age. They regard every protracted fever not malarial and not dependent upon a local inflammation as presumably typhoid. The symptoms from which we may regard the question of typhoid as established will be considered under Diagnosis. I have seen but two undoubted cases of typhoid under two years of age, and I believe it to be rare, at least in New York. No case recognised as typhoid occurred in a child under two years of age during my eight years' service in the New York Infant Asylum, where about ten thousand cases of acute illness were treated and over seven hundred autopsies made; and but two in my sixteen years' service at the Babies' Hospital where about the same number of autopsies have been made. No case has been recognised as typhoid, either in the wards or the post-mortem room of the

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[*] Archives of Pædiatrics, December, 1900.

New York Foundling Hospital during the past twenty-five years. Typhoid has been seen by Murchison at six months and by Ogle at four and a half months, the diagnosis being in both instances confirmed by autopsy; also by Griffith at seven months and by Taylor at eight months, with fairly typical symptoms. It is during epidemics that most of the infantile cases are seen; sporadic instances of infantile typhoid should always be regarded with suspicion, and I believe that most cases so diagnosticated are questionable. Even in epidemics it is surprising that so few infants are attacked. In that of Montclair, N. J., in 1894, of 115 cases, only 3 were under two years; in that of Stamford, Conn., in 1895, of 406 cases only 4 were under two years.

Typhoid in childhood is by no means rare, but it is not until after the fifth year that it can be said to occur frequently. The following figures, embracing groups of cases reported by eight writers, represent the relative frequency with which the disease is seen at the different ages: Of 970 cases, 8 per cent occurred under five years, 42 per cent between five and ten years, and 50 per cent between ten and fifteen years.

Typhoid fever is almost invariably contracted by drinking water or milk (see page 139) which contains the germs of the disease. The infrequency of typhoid in infants is explained, in part at least, by the fact that most of the water and a large part of the milk taken is previously boiled, or heated in some manner.

Lesions.—Typhoid in young children is so seldom fatal that opportunities for a study of the lesions have been limited. In a general way they resemble those of adults except in severity. In a considerable number of the cases the pathological process in the intestines does not go on to ulceration; and when ulcers form they are seldom large or deep, and perforation is very rare. Montmollin gives the following facts concerning 23 autopsies, most of them, however, being in children over eight years old: ulcers were present in 17 cases; they were situated in the lower ileum in 16, and in 10 they were only there; in the ascending colon in 9, and only there in one case; perforation occurred in 3 cases, in every instance in the lower ileum. Autopsies made upon infants may show even less severe intestinal lesions than those mentioned. In fact, some cases in which the clinical diagnosis was beyond question, have shown only moderate redness and swelling of Peyer's patches, the solitary follicles and the mesenteric lymph nodes—lesions which are exceedingly frequent in cases of simple diarrhœa. In a doubtful case such post-mortem findings do not establish the diagnosis of typhoid. Indeed, they prove nothing unless cultures from the intestinal contents, the mesenteric glands, or other organs, show the typhoid bacillus. Enlargement of the spleen is practically constant. The degenerative changes in the heart, the kidneys, and the liver are much less frequent and generally less severe than in adults.

Symptoms.—The peculiar features of typhoid in early life are seen only in children under ten years old; for after this time the disease does not differ essentially from the adult type. In brief, the typhoid of early childhood may be described as a fever characterized more often by nervous symptoms than by intestinal symptoms.

Onset.—A sudden onset with well-marked symptoms—fever, prostration, vomiting, etc.—is not uncommon; in fact, it is quite as frequently seen as the insidious beginning with lassitude, headache, coated tongue, anorexia, and gradual rise in temperature. In cases developing abruptly it often appears as if an acute indigestion had been the means of precipitating the attack. The most frequent initial symptom is vomiting; a chill is rare. Epistaxis occurs as an early symptom rather less frequently than in adults.

Condition of the bowels.—There is no constant relation between the severity of the intestinal lesions and the condition of the bowels. Taking large groups of cases together, diarrhœa is present in about half the total number. It is rarely profuse, from two to four discharges a day being the average. The appearance of the stools is seldom characteristic; they are usually thin and fluid, often containing mucus. Constipation may be present at the beginning only, or throughout the attack. Tympanites is generally moderate, and is often entirely absent; it usually accompanies constipation. Marked iliac tenderness and gurgling are infrequent.

Spleen.—By the end of the first week this is almost invariably found to be enlarged to a sufficient degree to be recognised by palpation. Usually the spleen extends but an inch or an inch and a half below the ribs, but at times it may be three inches or more; persistent enlargement always indicates that the disease is not at an end even though the temperature has reached the normal, and a relapse should be expected.

Eruption.—It is the experience of nearly all who have seen much of typhoid in children that the eruption is less constant, less abundant, and less characteristic than in adults. Of 670 cases in Morse's * collection, it was noted in but 60 per cent. The typical eruption consists of small, scattered, rose-coloured spots, which appear chiefly or solely upon the abdomen at the beginning of the second week. They come in successive crops, each one of which generally lasts three days, the whole duration of the eruption being about ten days.

Prostration, emaciation, etc.—As a rule the prostration is quite sufficient to keep a child in bed after the first few days. The general weakness after this time is in direct proportion to the height of the temperature. Loss of flesh is steady and usually marked; and in a prolonged attack there may be extreme emaciation.

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* Typhoid Fever in Childhood, with an Analysis of 284 Cases; Boston Medical and Surgical Journal, February 27, 1896.

Temperature.—In the cases with a gradual onset, the typical temperature curve is one which rises steadily for from two to seven days, fluctuates within the limits of one to three degrees during the second week, and steadily declines during the third week, reaching the normal on the average at the end of the third week. In cases with an abrupt onset, the temperature rises at once to from 102.5° to 105° F., but subsequently may run the same course as in the first group.

The following are the most important variations from the temperature curve of adults: The initial rise is much more frequently rapid; during the second week the remittent character is less marked, this probably depending upon the fact that ulceration is less frequent and less extensive; the average duration is shorter. In young children the proportion of cases in which the fever lasts only from eight to fourteen days is quite large (Fig. 204). After the age of ten years the type of the fever is much like that seen in adults. The maximum temperature in the mild cases is 103° or 104° F.; in the severe ones it often reaches 105° or 106° F., but rarely goes above this point. The range is usually higher than in adult cases of the same severity. At the beginning of convalescence a subnormal temperature is very frequent, and by many writers is considered to be the rule. A secondary rise is most frequently due to errors in diet, but may occur from the development of complications. A sudden fall indicates either perforation or intestinal hæmorrhage.

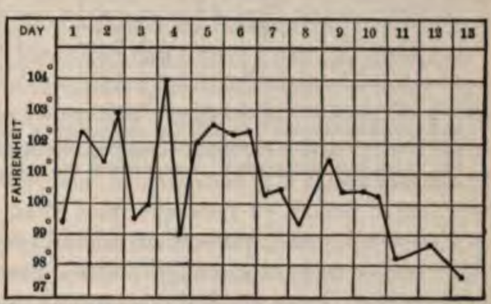

Fig. 204.—Typhoid fever of short duration in a child thirteen months old. Spleen enlarged; eruption typical; no diarrhœa and only moderate abdominal distention. There were two other cases in the family, all being due to the same cause—infected milk. (After Northrup.)

Relapses were present in 8·4 per cent of 533 cases collected by Morse. They follow about the same course as in adults (Fig. 205).

Nervous symptoms.—As a rule, these are more prominent in severe cases than the intestinal symptoms, and are directly proportionate to the height of the temperature. The extreme nervous symptoms belonging to the typhoid state in adults are rare in childhood, except in patients over ten years old. Headache and mild delirium at night are very frequent, the former being seen in the majority of cases. Young children are usually dull, apathetic, and often in a state of semi-stupor. Occasionally the disease may closely simulate meningitis. The nervous symptoms are usually most severe in the second, or early in the third week, and subside as the temperature declines.

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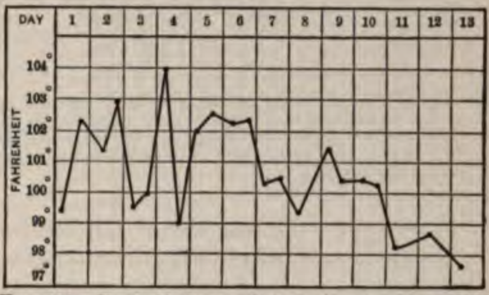

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Pulse.—This is increased in frequency, but not to the degree that is seen in most diseases of childhood with a similar elevation of temperature. The force and rhythm of the pulse are usually good, irregularity, very low tension, and dicrotism being rare as compared with adults.

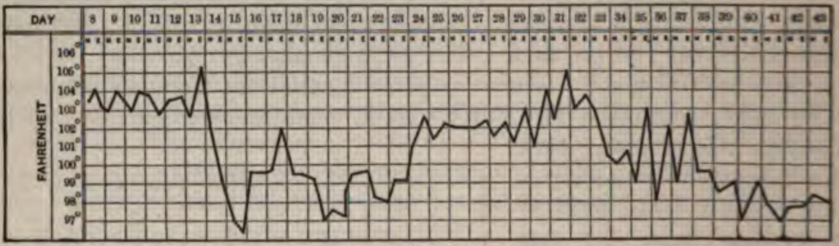

Fig. 205.—Typhoid fever with relapse. Child two and a half years old; early temperature high and symptoms typical; natural fall on fourteenth day; rise on seventeenth day apparently due to otitis; relapse on twenty-fourth day, with fresh eruption and return of splenic swelling which had disappeared. Temperature was subnormal at the end both of primary and secondary fever.

Urine.—A small amount of albumin is found in the urine of most of the severe cases at the height of the disease, and is due to acute renal degeneration; but a marked degree of nephritis is infrequent. In from one-fourth to one-third of the cases typhoid bacilli are found in the urine, generally in pure culture. They usually appear in the latter part of the disease, the second or third week, and may continue for months or even years. They are sometimes accompanied by evidence of cystitis or nephritis. Their number is in some cases so large as to render the urine turbid; in others they give no indication of their presence. Ehrlich's diazo reaction is usually present at the height of the fever.

Intestinal hæmorrhage.—Of 946 collected cases, mainly from hospital reports, intestinal hæmorrhage occurred in 30, or about three per cent; the majority of these were in children over ten years old. Of 24 collected cases of hæmorrhage in children, 10 terminated fatally. The youngest case of this nature which has come under my own notice was in a child of four and a half years.

Intestinal perforation.—This is even more rare than hæmorrhage. In 1,028 collected cases, this accident occurred but twelve times, or in 1·1 per cent. Eight of these proved fatal. Perforation is indicated by a sudden fall in the temperature, with collapse; usually there is vomiting and the rapid development of tympanites.

Complications and Sequelæ.—The complications of typhoid in early life are infrequent and usually mild. Bronchitis is present in most of the severe cases. Pneumonia has been noted in 9 per cent of the cases reported by various authors. Both serous and purulent effusions into the chest are occasionally seen, and sometimes abscess of the lung.

Complications referable to the nervous system are not very frequent, but are of much interest. Meningitis is extremely rare. Morse has collected twenty-one cases of aphasia, in two of which it was clearly due to embolism; in the remainder, however, it apparently was not dependent upon any organic lesion. In two thirds of the cases it came on during convalescence, and in nearly all complete recovery occurred after an average duration of three weeks. Aphasia usually followed a severe type of the disease, and in most of the cases was not accompanied by any other paralysis or by mental disturbance. Insanity is a rare sequel of typhoid in children, the usual type being acute mania. Adams (Washington) has reported two examples of this, both terminating in recovery. Chorea is seen rather oftener than after the other infectious diseases.

Otitis is not an infrequent complication, occurring much oftener than in adults. It is principally seen in young children and during the cold season. Among the less frequent complications may be mentioned: parotitis, which is usually suppurative and is seen in septic cases; abscess of the liver, examples of which have been reported by Bokai, Asch, and others; gangrenous inflammation of the mouth or genitals; pericarditis, endocarditis, and peritonitis, suppurative inflammations of joints, multiple abscesses and furunculosis. Tuberculosis of the lungs or bones not infrequently follows typhoid.

Diagnosis.—The diagnostic symptoms of typhoid are the Widal blood reaction, the discovery of the bacilli in the urine or fæces, the eruption, the course of the temperature, the enlargement of the spleen and the abdominal symptoms—diarrhœa, tympanites, intestinal hæmorrhage, and perforation.

The Widal reaction is present at some period in from 95 to 98 per cent of the cases, and thus becomes the most valuable single symptom for diagnosis. It is seldom obtained before the seventh day and frequently not until the tenth; it may not be present until convalescence or a relapse. Repeated tests should always be made if the first reaction is negative or doubtful; and the tests should be made by an experienced pathologist. The reaction is therefore of much less value for an early than for an exact diagnosis. A positive reaction may be present if the patient has previously had typhoid, something much less likely to be the case with children than with adults; in rare instances it has been obtained in other diseases or in health where no history of previous typhoid existed. Both these conditions, however, are very exceptional, and a positive reaction may as a rule be taken to establish the diagnosis.

Typhoid bacilli, according to the observations of Park (New York), may be demonstrated in the stools by culture in about 40 per cent of the cases. They are found in the urine, usually in the latter part of the disease, in about one-third the cases. Their discovery in either of these discharges is conclusive evidence of previous or existing typhoid.

An examination of both urine and fæces should, if possible, be made all doubtful cases.

The course of the temperature is an important aid to diagnosis, alone is not to be depended upon. The characteristic feature is a fe which continues for two, three, or four weeks, and subsides spon neously. The variations from the adult type have already been m tioned, also the frequency of the eruption, the enlargement of the sple and the abdominal symptoms. We are not warranted, I think, in mak the diagnosis of typhoid, if repeated tests fail to show the Widal re tion or if the eruption and splenic enlargement are absent, and no bac can be demonstrated in the discharges, no matter what the course of temperature may be.

One should be very slow to make the diagnosis of typhoid in a ch under two years old, unless the disease is epidemic. The great majo of sporadic cases reported as occurring in infancy are probably typhoid. After the fifth year the disease is more frequent, and symptoms in general resemble those of adults, except in severity.

A differential diagnosis is to be made from malarial fever, ileo-coli meningitis, tuberculosis, and from other ill-defined continuous fev of unknown origin. From malarial fever the diagnosis is to be made the temperature curve, the organisms in the blood, and the effect quinine. In most of the cases of malaria the temperature will be fou to touch the normal at some time in the twenty-four hours. The adm istration of full doses of quinine is a diagnostic test of much practi importance; an irregular or remittent fever which yields promptly quinine is most certainly not typhoid.

Ileo-colitis and typhoid fever are not often confounded. The form is chiefly seen in the first three years of life, a time when typhoid rare. The intestinal symptoms of ileo-colitis are marked even thou the temperature is not high, and they are altogether more severe th is usual in typhoid; while enlargement of the spleen, tympanites, a the eruption are not present.

The cerebral symptoms of typhoid may be difficult to distinguish fro meningitis, unless one has watched their development. Irregular respir tion, a slow, irregular pulse, localized paralysis and complete coma a seldom, if ever, seen in typhoid, and a retracted abdomen very rarel while the enlarged spleen and the peculiar eruption are not seen meningitis. In typhoid with pronounced nervous symptoms the te perature is usually higher than in meningitis.

General tuberculosis very often resembles typhoid so closely that differential diagnosis is almost impossible until local signs of tuberculos have appeared, usually in the lungs. (See page 1090.)

Prognosis.—Of 2,623 cases collected from the reports of twelve diffe ent writers, the mortality was 5·4 per cent. These are, however, almo all taken from hospital reports, where as a rule the mildest cases are not brought for treatment. The mortality of the disease in children over three years old probably does not exceed 3 or 4 per cent. Death seldom occurs from the disease itself, but usually from some accident or complication, the most frequent being pneumonia and intestinal hæmorrhage or perforation. Griffith's collection of cases occurring in infancy indicates a much higher mortality for this period. The death-rate for the first year reached nearly 50 per cent.

Treatment.—The usually low mortality of this disease shows how successful all methods of treatment are likely to be considered. In the great majority of cases very litte active treatment is required. Every patient with typhoid should be put to bed and kept there during the febrile period, and a few days beyond it, no matter how mild the attack may be. A fluid diet should be prescribed in every case, sterilised milk or animal broths, which should be given regularly every three hours, but not pushed beyond the desire of the patient. Milk may be diluted or partially peptonised, and kumyss or matzoon may be substituted for it if the stomach is irritable. Plenty of water should be given. Solid food should not be allowed until the temperature is normal.

Both the urine and fæces should be immediately and thoroughly disinfected by a solution of carbolic 1 : 20. If the movements are in a chamber or a bed-pan they should be covered with this solution for at least six hours before they are thrown into the water-closet. If napkins or diapers are used, they should be soaked in some effective antiseptic solution for twelve hours and then thoroughly boiled. Sheets stained by discharges should be treated in the same way, and all bed-linen should be boiled for two hours apart from the washing of the family. The efficiency of urotropin in removing typhoid bacilli from the urine seems now to be well established. It should be given at the close of the attack in doses of three to five grains, three times a day, and continued for a week or ten days.

Diarrhœa calls for treatment only when the movements exceed four or five in twenty-four hours. If no more than this number are present, they should not be interfered with. Opium and bismuth are undoubtedly the best means for controlling excessive diarrhœa, but care should be taken that they are not pushed to the degree of inducing constipation.

Constipation early in the disease may be relieved by calomel, followed by the salines, or castor oil, but all active purgation should be avoided. Later in the disease daily irrigation of the colon with tepid water is better than anything else. On the whole, constipation is more troublesome to overcome than diarrhœa.

Tympanites is rarely severe enough to require treatment; it may be relieved by turpentine stupes, by a glycerin suppository, or a small glycerin injection (one teaspoonful of glycerin to two ounces of water), or, better still, by the use of the rectal tube.

Whenever the temperature remains above 103° F., antipyretic measures are indicated. In mild cases cold or tepid sponging is generally sufficient. In those which do not yield to such measures, baths may be employed. Not all children bear baths well, and considerable discretion should be used in employing them. One should be guided quite as much by the effect upon the pulse and the nervous system as by the temperature. The best method is usually the graduated bath; the child is placed in the tub with the water at a temperature of 95° or 100° F.; this is gradually lowered to 95°, 90°, or even 85° F., but seldom lower. The body should be actively rubbed while the child is in the bath, to prevent shock and cardiac depression. The cold pack (pages 49 and 50) may be substituted for the bath where circumstances make the latter impracticable. The bath or pack should be repeated in an average case in from three to six hours. The method of applying cold which causes the least disturbance to the patient is the one which should always be selected.

The milder nervous symptoms—headache, restlessness, sleeplessness, etc.—may be relieved by an occasional dose of phenacetine, either alone or in combination with the bromides, or by cold or tepid sponging; the more severe ones usually occur with high temperature, and are best controlled by the cold bath.

Stimulants in most of the cases are not called for. They are to be given according to the indications afforded by the pulse, the first sound of the heart, and the child's general condition. They are seldom needed earlier than the middle of the second week; they should be well diluted. Brandy or whisky is to be preferred to wines, and, unlike the milk, they may be given at frequent intervals whenever the patient will take them best. Intestinal hæmorrhage calls for absolute quiet, morphine hypodermically, and an ice-coil to the abdomen, nothing being given by mouth except stimulants, turpentine, and possibly opium. Intestinal perforation is successfully treated only by early laparotomy.

CHAPTER X.

TUBERCULOSIS.

TUBERCULOSIS is an infectious communicable disease, due to the bacillus tuberculosis of Koch. It may be local or general, and may involve any organ and almost any structure in the body.

Etiology.—*Frequency.*—Müller, in 500 autopsies upon children in Munich, found tuberculosis in 40 per cent of the cases; in 30 per cent death was due to tuberculosis, and in the remaining 10 per cent tuberculosis was found at autopsy in patients dying from other diseases. I do not think it is so frequent in this country, for, of 726 consecutive autopsies in the New York Infant Asylum, tuberculosis was found in only 58, or 8 per cent of the cases; 6 per cent of the deaths were due to tuberculosis, and in 2 per cent the children died from other diseases. Of 319 consecutive autopsies in the Babies' Hospital, tuberculosis was found in 44, or 14 per cent.

Predisposing causes.—The predisposition to tuberculosis is general or local. General predisposition may be inherited directly from parents who have themselves suffered from tuberculosis, or from those who, in consequence of syphilis, alcoholism, or any other constitutional vice, have transmitted a feeble constitution to their children. Inherited predisposition is exceedingly common, and really signifies a diminished resistance of the cells of the body to tuberculous infection. It should be distinguished from the very exceptional condition of congenital tuberculosis, where infection takes place before birth. General predisposition includes the child's surroundings, in so far as they affect the constitution and lower the general vitality. Children reared in the city, either in institutions or in crowded tenements, are more frequently affected than those who have had the advantage of the best surroundings, not only because of their increased chances of exposure, but also from their feebler resistance. Marasmus, intestinal diseases, and, in fact, any debilitating general or local disease, may predispose to tuberculosis.

A local predisposition is created by any pathological condition of the mucous membranes or organs most exposed to infection. The most important are repeated attacks of bronchitis, broncho-pneumonia, or pleurisy, and chronic catarrhal inflammation of the mucous membrane of the nose or pharynx, so frequently associated with enlarged tonsils or adenoid growths of the pharynx. Much less frequently the local predisposition is the result of some previous disease of the intestines.

The rôle played by other diseases in the development of tuberculosis is an important one, and until recently but little understood. In a very large number of cases tuberculosis develops as a sequel of one of the acute infectious diseases, particularly measles, pertussis, or epidemic influenza. In such cases there has probably existed previously a latent tuberculosis, usually in the bronchial lymph nodes. This process, sometimes long quiescent, under the stimulus of a new infection may be awakened to activity. It is to be noted that it is the infectious diseases that are intimately associated with pulmonary complications, which are liable to be followed by tuberculosis.

Age.—No age is exempt from tuberculosis. It was formerly believed that the disease was rare in infancy, but recent observations have shown that, although its form is somewhat different, it is more frequent in infancy than at any period of later childhood. Statistics, taken chiefly from two institutions where children up to four years of age are received, give the following results, the diagnosis being confirmed by autopsy in nearly every case under two years old:

Under three months	5	cases
From three to six months	21	"
" six to twelve months	31	"
" twelve to eighteen months	29	"
" eighteen to twenty-four months	10	"
" two years to five years	32	"
Over five years	15	"
Total	143	"

It will be seen that the first year furnished 57 cases, the second year 39, and the succeeding three years but 32 cases.

Intra-uterine infection, or the direct transmission of tuberculosis, although rare, has been established by the report of at least seven complete and well authenticated cases. Tuberculosis of the placenta is still more frequent, there being according to Wollstein * now twenty cases on record. In most of the cases of congenital tuberculosis the mother has been suffering from the disease in an advanced form, and the child is either still-born or dies soon after birth. Besides tuberculosis of the placenta, there are found tubercle bacilli in the organs of the child, and when life is prolonged, there are generalised lesions showing infection through the blood. In some cases cheesy nodules in the umbilical cord have been observed.

Intra-uterine infection is highly probable in many of the cases of children born of tuberculous mothers, who develop the disease during the first few months of life, although they may show no evidence of it at birth. Among my own cases there were five which died of tuberculosis during the first three months. One of these children was but twenty days old. It was born prematurely of a mother who at the time was suffering from advanced tuberculosis, and died from that disease shortly after the child. Besides other lesions, the autopsy showed, in the case of the mother, tuberculosis of the endometrium. In this instance the infection of the child certainly took place before birth.

In another case, a child died of general tuberculosis, with wide-spread lesions, at the age of seven weeks. The mother of this infant died from tuberculosis eleven days after the birth of the child. Intra-uterine infection must, however, be considered rare in comparison with the frequency with which infection takes place after birth, instead of being, as was formerly supposed, very common.

Tuberculosis may be communicated by direct inoculation, as in the case of a bite from a person suffering from the disease, several instances of which are on record. The rite of circumcision performed by a rabbi

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* See Archives of Pædiatrics, May, 1905, for literature on congenital tuberculosis.

suffering from tuberculosis is also known to have caused the disease. One of the most striking instances of direct infection is that reported by Reich.* In a town of about 1,300 inhabitants, the obstetric practice was divided between two midwives. Within fourteen months no less than ten infants, who had been delivered by one of these women, died of tuberculous meningitis. In none of these families was there a history of tuberculosis. This midwife was found to be suffering from pulmonary tuberculosis, and died from that disease. It was her custom to remove the mucus from the mouth of the newly-born infants by direct mouth-to-mouth aspiration, and then to establish respiration by blowing into the nose. In the practice of the other midwife, who was healthy, no cases of tuberculosis occurred, although she treated the newly-born infants in the same fashion.

The following instance of infection has recently come to my notice: Two little girls were much in the room and about the bed of a young woman who was suffering, it was afterward discovered, from pulmonary tuberculosis. Within three months of that time, and within six weeks of each other, both died of tuberculous meningitis.

Examples might be multiplied indefinitely of cases where children have contracted the disease from a close exposure to nurses or other persons in the household. More frequently, however, the mode of infection can not be traced, the exposure doubtless being in most of these cases long antecedent to the development of symptoms.

Aside from accidental inoculation already mentioned, the tubercle bacilli may gain an entrance to the body either through the respiratory or the alimentary tract or the skin—the last, however, being so very rare that it need only be mentioned. In infancy and early childhood, infection is most frequently through the respiratory tract. This is indicated by the situation of the primary lesions (pages 411 and 1076). The source of the bacilli in the inspired air is mainly the sputum of patients suffering from pulmonary tuberculosis, which dries and becomes part of the dust of the street, of the railroad car, the home, or the hospital. Bacilli may be taken into the alimentary tract with milk from tuberculous cows or tuberculous women. Infection in this way I believe to be rare.† Unless the udder is the seat of disease, the number of bacilli in cow's milk is so small that the chances of infecting a child after these bacilli have passed the stomach are exceedingly small. Its possibility even is questioned by many good authorities. The same may be said regarding the transmission of tuberculosis through the milk of a nurse. Infection from the meat of tuberculous animals is doubtless a possibility, but hardly more. Bollinger's experiments in feeding animals with the expressed juice of such meat gave negative results.

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* Berliner klinische Wochenschrift, No. 37, 1878.

† In this connection the following incident is interesting as bearing upon the other side of the question: Near a large American city was a fancy stock farm of registered Jersey cows, which supplied milk for table use and infant feeding to a large number of families in the wealthiest part of the city, for a period of over ten years. At the end of that time the tuberculin test was used for the first time, and 45 per cent of these cows were found to be tuberculous, and were killed by order of the State Board of Health. The diagnosis was confirmed by autopsies upon the animals in every instance. An investigation was instituted among the children who had been fed upon this milk, but in only one case of many hundreds could it be learned that tuberculosis had developed, and in this instance it was by no means established that the

The Various Paths of Infection adopted by the Tubercle Bacillus.—The tubercle bacilli which enter the body with the inspired air are arrested upon the mucous membrane of the upper or the lower respiratory tract; upon which one of these, is largely determined by local conditions in the various mucous membranes. Both clinical experience and animal experiments indicate that the bacilli may pass through a mucous membrane without inducing in it a tuberculous disease, but that penetration is much easier if the mucous membrane is the seat of a catarrhal inflammation, or if the epithelium has been injured. The bacilli are taken up by the lymphatics from the surface of the mucous membrane upon which they have lodged, and are carried to the nearest lymph nodes, where, for a considerable time at least, they are arrested. It has long been a familiar clinical fact that the great majority of children who suffer from tuberculosis of the cervical lymph nodes escape general tuberculous infection, so eminent an authority upon this subject as Treves considering this to be a very exceptional result.

It is not infrequent, in autopsies both upon children and adults dying from various non-tuberculous diseases, to find tuberculosis limited to the bronchial lymph nodes. In a series of 125 autopsies at the New York Foundling Asylum upon children with tuberculosis, Northrup[*] found 13 such cases, these being children who had died from acute non-tuberculous diseases. Many confirmatory reports have been published by Bollinger (Munich) and others. I have myself seen it in a number of instances.

H. P. Loomis[†] (New York) made inoculation experiments with the bronchial lymph nodes taken from the bodies of thirty persons dying by violence or from acute disease, in whom no evidence of tuberculosis in any other part of the body could be found at autopsy. From eight of the cases he produced tuberculosis in animals by inoculation. Arnold has shown

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milk had been the source of infection. It should be stated that this was before the days of sterilizing milk for infant feeding. Besides the families who took the milk in the manner mentioned, the employees at the farm were accustomed to drink the skimmed milk in large quantities daily as a beverage in the place of water. Many of them continued to do this for years, and yet not one of them developed tuberculosis.

[*] New York Medical Journal, February 21, 1891.

[†] The Medical Record, December 20, 1890.

by experiments with dust inhalation in animals, that in a short time the bronchial lymph nodes were filled with dust, though the bronchi and alveoli were free; but, however prolonged the inhalation, dust was never found in the lymphatic vessels beyond the nodes.

Arriving at the lymph node, the bacilli light up a tuberculous inflammation of varying degrees of intensity, depending upon their number and upon local conditions. This inflammation may pass through the usual changes of tuberculous glands—congestion, swelling, cell proliferation and caseation; or the process may be arrested at any point, and the products of inflammation become encapsulated by a proliferation of fibrous tissue, in which condition they may remain latent in the body for an indefinite number of years—possibly for a lifetime. This is what occurs in older and more vigorous children, and it is consistent with every outward sign of health; but it is a smouldering ember which at any time may be fanned into flame under the stimulus of an inflammation excited by some other cause.

In infants and young children, the tendency is always for the bacilli to lodge first in the bronchial lymph nodes, probably on account of the favourable conditions for entrance existing in the bronchi and lungs. In those who are delicate and have but little resistance, the process in the lymph nodes is likely to go on to caseation and softening, and secondarily to this process in the glands, the lung may become infected. Of 91 cases observed by Northrup, in which the mode of infection could be pretty accurately traced, in 88 it was primarily in the bronchial lymph nodes. The manner of the extension of the disease to the lung is not always easy to trace; but in many instances it has been shown to be the result of the softening of one of these small tuberculous lymph nodes, which then ulcerates through the wall of one of the small bronchi or a blood-vessel, in this way distributing its bacilli through the lung.

Although this is the course usually taken by bacilli when they are inhaled, it is not always the case. Lesions in the lungs are occasionally found where the lymph nodes are not involved; and there are other cases in which advanced changes exist in the lung, while only the earlier ones are seen in the lymph nodes. In these cases, which perhaps are to be considered as exceptional, the tuberculous process probably begins in the walls of the small bronchi, the alveoli, or in the connective-tissue septa.

Tubercle bacilli entering the alimentary tract rarely cause lesions of the gastric mucous membrane, or through it reach the lymphatic circulation. In the intestines, however, more favourable conditions exist. It is possible for the bacilli to reach the mesenteric lymph nodes without causing disease of the intestinal mucous membrane, but I believe it to be exceedingly rare; for by careful search I have seldom failed to find intestinal ulceration where the lymph nodes were manifestly tuberculous.

Lesions.—In the following table are given the different lesions of tuberculosis as they were found in 119 autopsies, of which I have notes. These represent the lesions of infancy and early childhood, 66 per cent of these children being two years old or under. There are introduced for comparison, the statistics of 131 autopsies from the Pendlebury Hospital Reports (Manchester, England). Very few of the cases in this series were under three years, the hospital admitting only older children :

Frequency of the Different Visceral Lesions of Tuberculosis.

ORGANS.	Personal cases; 119 autopsies (chiefly under three years).		Pendlebury Hospital Reports; 131 autopsies (chiefly over three years).	
Lungs	117	99·0 per cent.	122	93·0 per cent.
Pleura	69	58·0 "	100	76·0 "
Bronchial lymph nodes	108	96·0 "	91	70·0 "
Brain	40	37·0 "	60	46·0 "
Liver	77	65·0 "	86	65·0 "
Spleen	88	75·0 "	76	58·0 "
Kidneys	46	39·0 "	54	41·0 "
Stomach	5	4·0 "	1	0·8 "
Intestines	40	37·0 "	65	50·0 "
Mesenteric lymph nodes	38	35·0 "	77	59·0 "
Peritonæum	10	9·0 "	37	28·0 "
Pericardium	7	6·0 "	4	3·0 "
Endocardium	1	0·8 "	..	....
Thymus	3	2·5 "	..	....
Suprarenal capsules	2	1·7 "	2	1·6 "
Pancreas	3	2·5 "	..	....

The varieties of tuberculosis seen at different ages.—During the first two years of life, tuberculosis, with great uniformity, involves first the bronchial lymph nodes and the lungs. It is most frequently the pulmonary process which is the cause of death, and next to the lungs, death is due to tuberculosis of the brain. It is rare for any other local tuberculous process to be fatal at this time of life. Of 72 cases of tuberculosis in the first two years of life, in which the exact nature of the lesions was determined by autopsy, the lungs were extensively involved in all; but death was due to meningitis in 13, in only one to tuberculous peritonitis, and in one to hæmorrhage from a tuberculous ulcer of the intestine. During infancy, meningitis is rare except when associated with pulmonary tuberculosis; but after the second year, meningitis is relatively more frequent. Of the deaths from tuberculosis during the third year, meningitis was present in over one half the number. After this time it frequently exists with few and sometimes with no lesions in the lungs, it being often secondary to tuberculosis of the bones or lymph nodes.

Beginning with the third year, tuberculosis of the bones, cervical and mesenteric lymph nodes, peritonæum, and intestines, becomes more frequent, and in any of these organs it may occur as the principal lesion, although at autopsy the lungs, even at this age, are rarely found free from infection.

Pulmonary Lesions.—As compared with adults, the pulmonary tuberculosis of children is more widely diffused, and the predominance of cases in which the lesion is at the upper lobes, though less marked, still exists. The peculiarities are principally seen in children under two years. In those who have passed the sixth or seventh year, the pathological processes resemble those of adult life. In my own autopsies the oldest lesions were found 69 times in one of the upper lobes (left 35, right 34); 23 times in the right middle lobe, and 35 times in one or other of the lower lobes (left 24, right 11). Although localized tuberculous processes are frequently met with in patients dying from other diseases, those who die from tuberculosis usually show wide-spread lesions of the lungs, and the younger the child the more diffuse they are.

1. Miliary tuberculosis of the lungs.—In nearly every case of pulmonary tuberculosis, miliary tubercles are found in some part of the lung; usually they are seen upon the surface and in scattered areas in the vicinity of some older process. Occasionally in older children, but very rarely in infants, they are distributed through nearly the whole of both lungs.

In some places the lung, with the exception of these gray granulations, appears quite normal; in others it is congested, and shows between the tubercles the lesions of simple broncho-pneumonia in its various stages. There is also an acute bronchitis of the middle-sized and smaller bronchi. The microscope shows that the tubercles usually develop in the walls of the small bronchi or the blood-vessels, or very close to these structures. In their gross appearance, the lungs in these cases resemble those in ordinary acute broncho-pneumonia, with the exception that everywhere upon the surface and throughout the substance of the lung are seen the small gray granulations, and in most cases some small yellow tuberculous nodules. The pleura is usually normal except for the presence of the tubercles. This form of the disease represents the rapid dissemination of tubercle bacilli throughout the lungs, the miliary tubercles being the result of the inflammation excited by their presence.

2. Tuberculous broncho-pneumonia.—This is the most frequent and the most characteristic form of tuberculosis in infants and young children, and it is the one which at this age usually causes death. In this form of disease there are produced in the lung, caseous nodules, or larger caseous areas, some of which have usually undergone softening by the time the case comes to autopsy. The process generally runs a somewhat subacute course. With the lesions mentioned there are always associated those of simple broncho-pneumonia.

The pleura is involved in almost every case. There may be simply dense connective-tissue adhesions which bind the lung firmly to the chest wall, or the pleura may be greatly thickened and contain caseous deposits. Occasionally empyema is seen, but it is almost always sacculated and small.

Both lungs are usually involved, but one to a much greater degree than the other. There are found large areas of consolidation which sometimes involve an entire lobe, but more often areas are seen in several lobes. These portions of the lung appear much firmer and harder than in ordinary pneumonia. The upper lobes are more often affected than the lower, and especially that part of the lobe which is near the root of the lung, on account of its frequent association with tuberculosis of the bronchial glands; the disease very often extends forward from this point to the middle lobe of the right, or the corresponding part of the left lung. On section the affected part of the lung usually shows many caseous nodules varying in size from a pin's head to a walnut, which appear of a pale yellow colour, and resemble caseous lymph nodes. They contain giant cells and are usually filled with bacilli, those which have softened containing yellow pus. There is nearly always seen in some part of the lung a large caseous area; and not infrequently there may be diffuse caseation of almost an entire lobe (Fig. 206). Sometimes no spot of softening is seen even in these large areas, but in the great majority of them there are found cavities of variable size with ragged but not dense walls.

Softening and excavation represent the final stages of the process in tuberculous pneumonia. It has been shown by Prudden that these changes are chiefly or entirely due to other pathogenic organisms—usually the streptococcus or staphylococcus—and not to the tubercle bacillus. Softening usually begins in the centre of a caseous part, often at several points at the same time. Areas of excavation large enough to deserve the name of cavities were present in thirty-five of seventy two autopsies upon tuberculous patients, two years old and under. They are found in the great majority of the cases in which continuous pulmonary symptoms have been present till death. They vary in size from a cherry to a hen's egg, and sometimes a much larger one is seen (Fig. 207). They are usually rather deeply seated, and partially or entirely filled with caseous masses or pus, but very seldom perforate the pleura, causing pneumothorax or pyo-pneumothorax. It is rare in a young child to find cavities surrounded by dense fibrous walls such as are seen in older children or in adults; for in infancy the process of softening once begun usually advances steadily until the death of the patient.

It is very frequent to find at autopsy small cavities surrounded by larger areas of caseous pneumonia, and these in turn surrounded by a zone of simple pneumonia through which are scattered many miliary tubercles. Often the lesions mentioned will be present in one lobe, while the other lobe or the opposite lung will show only the changes of a simple pneumonia.

The bronchial lymph nodes are in these cases invariably found to be tuberculous, and not only those at the root of the lung, but if a dissection is made, a chain of these tuberculous glands will be found to follow the larger bronchi for some distance into the lung (Fig. 210). Sometimes one may discover one of these which has softened and ulcerated through into a small bronchus, and in this way has spread the infection throughout that part of the lung.

Microscopical examination of these cheesy nodules shows that they most frequently begin as tuberculous deposits in the walls of the small bronchi, either in the mucous membrane, the fibrous coat, or the lymphatics; sometimes, however, they begin in the walls of a small vein or artery. Cell proliferation takes place, separating the coats of the bronchus or blood-vessel, and partly or entirely obstructing its lumen. Softening may

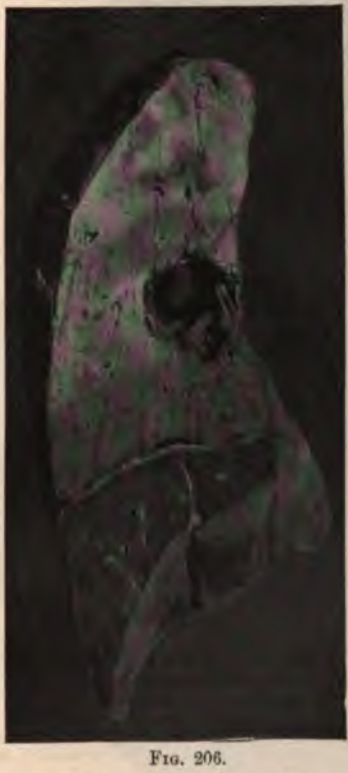


FIG. 206.

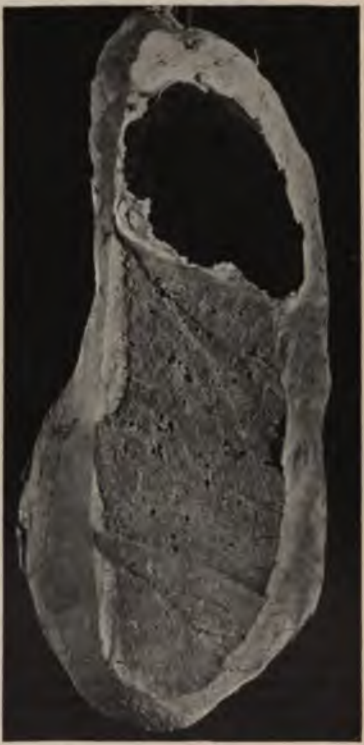


FIG. 207.

FIG. 206.—Tuberculous pneumonia. A vertical section through the middle of the right lung of a child thirteen months old. The greater part of the upper lobe is uniformly caseous—a diffuse tuberculous pneumonia; near the centre the commencement of a cavity is seen; below it has the appearance of a consolidation from simple pneumonia. The part of the lower lobe shown is normal.

FIG. 207.—Cavity from breaking down of tuberculous pneumonia; another view of the same lung, the section being made very near the posterior border of the lung. The cavity occupies at this point nearly the whole of the upper lobe. At autopsy this cavity contained numerous loose caseous masses, the largest being the size of a marble. The lower lobe is normal. (For history, see Fig. 213.)

take place and the contents be discharged into the bronchus or blood-vessel. About this focus other changes of an inflammatory character occur, as a result of which each cheesy nodule is surrounded by a zone of simple broncho-pneumonia (Fig. 208) which tends, in a measure at least, to limit the tuberculous process. The larger caseous areas are formed by an extension of this process to the zone of pneumonia which surrounds it; but in its further growth it is still preceded by a simple pneumonia (Fig. 209). The rapidity with which the lesions advance differs much in the different cases, and is greatly modified by the patient's age; in infants the progress is apt to be continuous until the death of the patient; in older children it is usually slower, and is often interrupted by longer or shorter intervals of arrest and even of partial retrogression. Such periods are marked by the absorption of the simple inflammatory products in the zone of pneumonia surrounding the tuberculous nodule, accompanied by improvement in the symptoms and

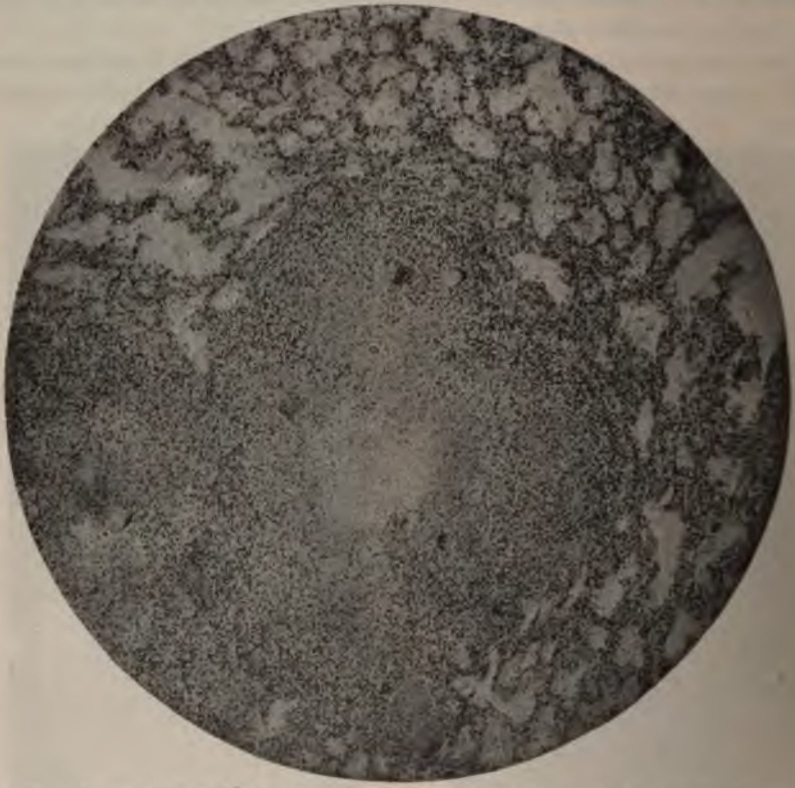


FIG. 208.—A small tuberculous nodule surrounded by lung tissue which shows only slight inflammatory changes. The centre of the nodule is necrotic; at its periphery is shown infiltration with round cells and several giant cells. (From Karg and Schmorl.)

often by a disappearance of some of the physical signs. During these times of quiescence there is an opportunity for the organization of the cells infiltrating the alveolar walls and septa into a more or less resistant fibrous wall which acts as a barrier against the advance of the pathological process.

Not infrequently one sees in the post-mortem room one or two caseous, or less frequently calcareous, nodules encapsulated by firm, organized connective tissue where a most careful search fails to show any other tuberculous lesion in the lung. If, however, the nodules are widely scattered through the lung, such an arrest of the process is not to be expected.

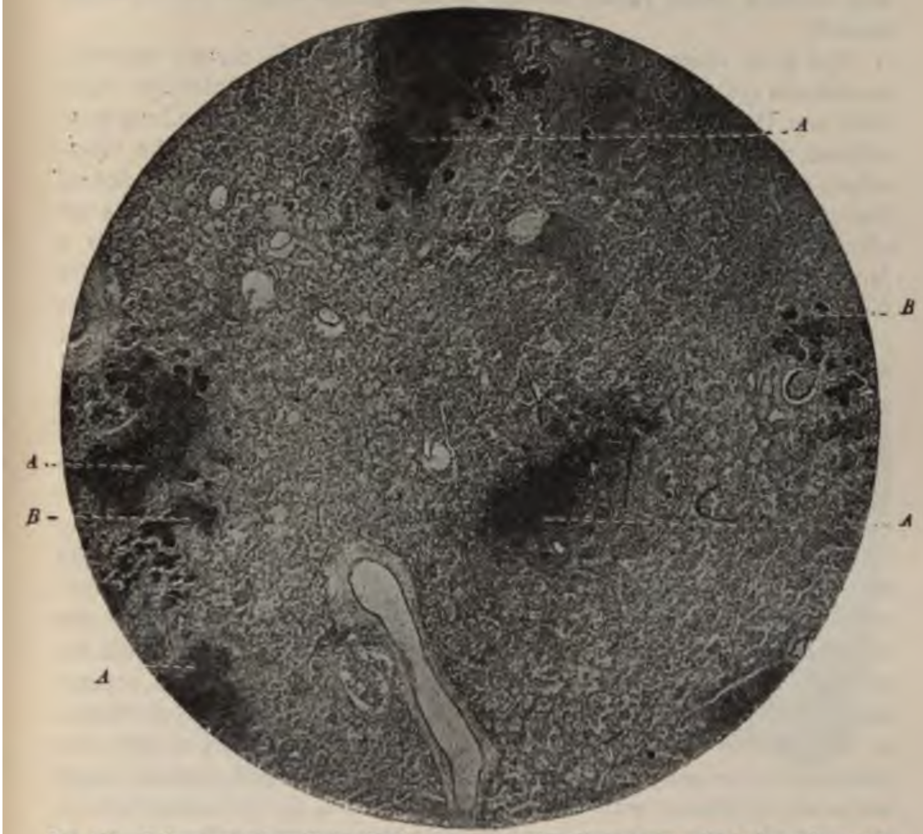


FIG. 209.—Pulmonary tuberculosis, showing areas of tuberculous pneumonia and conglomerate tubercles. In the greater part of the specimen the air vesicles are filled with the products of simple pneumonia. The larger dark areas, *A A A*, are spots of tuberculous pneumonia, while at *B B* only single air vesicles or groups of two or three are affected by the tuberculous process. The specimen shows a comparatively early stage of the process, of which the late stage is represented by Fig. 208. Patient, a child three months old; the symptoms, those of simple acute pneumonia. There were conglomerate tubercles scattered through both lungs, and large areas of cheesy pneumonia in the left lower lobe.

3. Chronic pulmonary tuberculosis, chronic phthisis.—With the patho-

logical process as it is seen in adults, we have nothing to do in infants and very young children. In those who have reached the age of eight or ten years the disease is essentially the same as in adult life, and need not be described here.

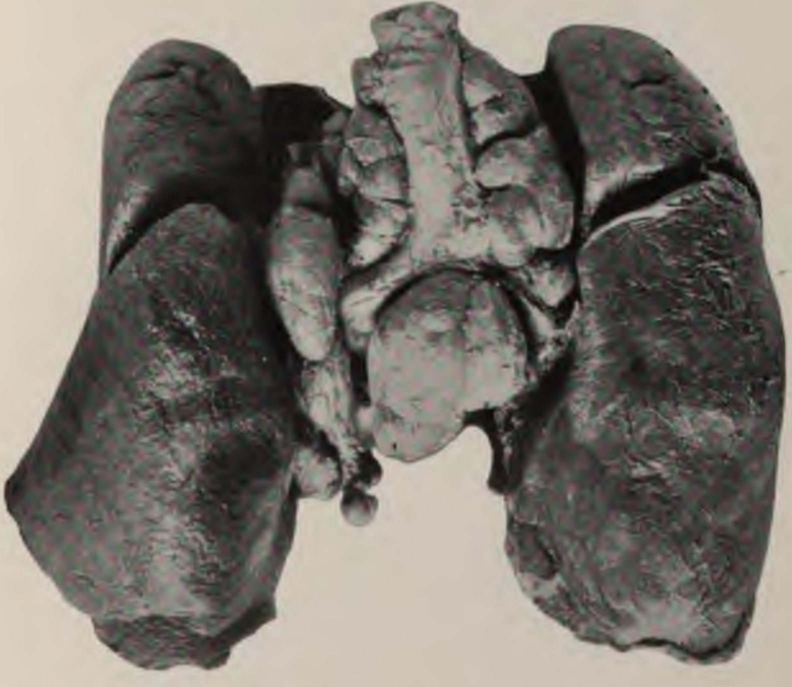
In little children the nearest approach to this condition is seen in the cases of tuberculous broncho-pneumonia, which run a slow, irregular, and somewhat chronic course. The essential features of the process in these patients is a chronic interstitial broncho-pneumonia with tuberculous nodules which rarely undergo softening, but usually become encapsulated.

The gross lesions closely resemble those of simple chronic broncho-pneumonia (page 583). There are the same generalized pleuritic adhesions and the shrunken cicatricial condition of the part of the lung most affected, with bronchiectasis, compensatory emphysema, etc. The tuberculous nodules are old and for the most part converted into dense fibrous tissue in the centre of which, however, some softened, caseous areas are often seen. Lesions like those described, which may be regarded as a form of recovery, are usually found in patients who have died of other diseases; sometimes in those who have died of other forms of tuberculosis —of the brain, bones, or peritonæum; at other times they are associated with a recent process in some other part of the lung. The bronchial glands may be somewhat enlarged and contain encapsulated caseous masses, or they may be calcareous.

Bronchial lymph nodes (*bronchial glands*).—The prominence of the lesions of the lymph nodes is one of the most striking features of tuberculosis in infancy and early childhood. Those which are most frequently affected are connected with the bronchi. The lymph nodes, to which the term "bronchial glands" is generally applied, consist of three groups: the first of which surround the trachea; the second are situated at the bifurcation of the trachea and surround the primary bronchi; while the third follow the course of the bronchi into the lung, being found, according to anatomists, as far as the fourth division. The anatomical relation of the different groups should be borne in mind, since upon them the symptoms principally depend. The first group, or the peri-tracheal lymph nodes, are in relation with the superior vena cava, the pulmonary artery, the pneumogastric and recurrent laryngeal nerves; the second group, at the bifurcation of the trachea, with the œsophagus, pneumogastric nerve, and aorta; the third group, with the bronchi and the branches of the bronchial and pulmonary arteries and veins.

All the groups are usually involved at the same time, but in varying degrees, and in most cases those belonging to one lung to a greater extent than the other; in my own cases those of the right side have more often been involved than those of the left. There may be simply two or three tumours as large as a hazelnut, or there may be a mass two or three inches

PLATE XX.



Tuberculosis of the Tracheo-Bronchial Lymph Nodes.

From a fairly nourished child, four months old, who was under observation for three weeks, with slight fever and a most severe, teasing, dry cough, which was almost constant, and upon which no treatment seemed to have the slightest effect. At first there were no signs of disease in the lungs; later there were a few coarse scattered râles.

There were small tuberculous deposits throughout both lungs, with quite a large area of cheesy pneumonia in the right middle lobe, and scattered miliary tubercles in other organs.

in diameter, which is made up of ten to twenty of these nodes fused together by inflammatory products, completely surrounding the trachea and both the large bronchi. It is rare that the individual glands are more than an inch in diameter, and most of them are smaller than this.

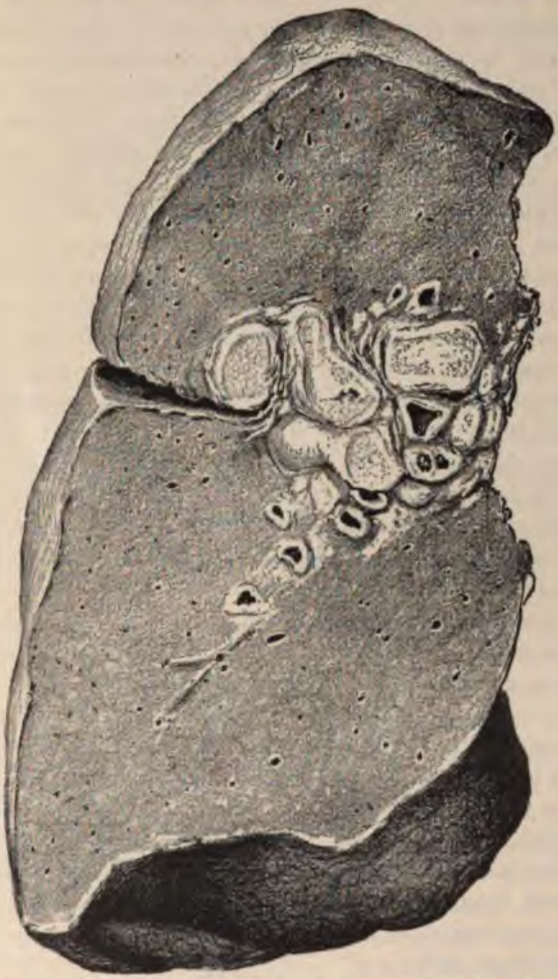


FIG. 210.—Tuberculous bronchial lymph nodes. Section of the lung of an infant through cheesy bronchial lymph nodes at the root of the lung, and adjacent cheesy masses, several of which have softened at the centre; the lung otherwise normal; life-size. (After Northrup.)

A well-marked but not unusual example of this condition is shown in Plate XX. There is usually found a chain of these tuberculous glands following the course of the large bronchi for some distance into the lung; sometimes these are almost as large as the external group (Fig. 210); at other times they are not noticed unless a somewhat careful dissection is made. The process is not infrequently more advanced in these deeply-seated glands than in those situated at the root of the lung; and lesions here are also more important, as it is very frequently through them that the lung becomes infected.

The pathological changes through which these glands pass as a result of tuberculous infection, are very similar to those already described with reference to the cervical glands. Suppuration is less frequent than in the region of the neck, while calcific degeneration is much more so. This applies especially to children over three years old. In infancy suppuration is not infrequent in the bronchial glands, while at this age calcification is extremely rare. Infection of these lymph glands is not always followed by general tuberculosis or even by infection of the lung. Although the process has gone on to caseation, these inflammatory products with bacilli may become encapsulated, and may remain innocuous for an indefinite period. The bacilli may die or may exist here, living, for years. At any time the old process may be lighted up, and a more or less rapid dissemination of tubercle bacilli take place through the lungs or through the whole body. Latent tuberculosis more frequently exists in the bronchial lymph nodes than in any other structure in the body.

Secondary lesions may be produced by these lymph nodes. The pneumogastric and recurrent nerves may be surrounded by one of these cheesy masses which causes pressure and irritation. The œsophagus, the trachea, or the bronchi, may be compressed or opened by ulceration. The superior vena cava usually suffers only compression, but this or any of the other large vessels may be opened. Ulceration may also take place into one of the large or small bronchi or the trachea. If the gland has softened and broken down, and if the bronchus is a small one, the only result of this may be a rapid spreading of tuberculous infection throughout the lung. If sudden rupture occurs, a large caseous mass may escape into the trachea, or a large bronchus, with a result similar to that produced by any other foreign body. If suppuration occurs, the abscess may rupture into the surrounding cellular tissue, causing mediastinal or retro-œsophageal abscess. This may open externally at the suprasternal notch, or in the first or second intercostal space, or may ulcerate into any of the large vessels, the œsophagus, or the pericardium, or may burrow downward into the peritoneal cavity.

Pleura.—This is rarely normal in any case of tuberculosis. In acute general tuberculosis the only lesion may be a deposit of miliary tubercles upon the visceral pleura. In most of the other cases there are found fibrous adhesions over the part of the lung involved, binding it to the pericardium, the diaphragm, or the chest wall. The amount of thickening of the pleura varies a good deal, but is rarely great. In about one fifth of my own autopsies tuberculous nodules were found in the pleura; with these lesions there is usually considerable thickening. Pleurisy with a hæmorrhagic exudation is very rare in the tuberculosis of early childhood. Empyema is also rare, being seen in but five per cent of my cases, and then it was small and sacculated. Pneumothorax and pyopneumothorax are very rare in children under three years of age; they were not seen in any of my cases.

Heart.—It is exceptional for the pericardium to be affected even in the most generalized forms of miliary tuberculosis. In such cases the usual lesion is a deposit of a few gray tubercles upon the visceral surface. In chronic cases other lesions analogous to those of the pleura may be seen, but all are rare in childhood. In a single instance I have seen miliary tubercles upon the endocardium. They are extremely rare, and the development of cheesy nodules in the heart is almost unknown in early life.

Brain.—Tuberculosis of the brain is not uncommon during infancy, being then associated in nearly all cases with general tuberculosis, and especially with tuberculous pneumonia; but it is relatively twice as frequent after the second year. There may be found miliary tubercles alone, or these may be accompanied by inflammatory products—tuberculous meningitis—or there may be caseous nodules. Miliary tubercles are frequently found in small numbers in cases which have presented no symptoms. The lesions of tuberculous meningitis have already been described. Cheesy nodules are rare in infancy, being noted in but 2.5 per cent of my own autopsies, which were mainly on children under three years old; while in the Pendlebury Hospital cases, including those between four and twelve years old, they were noted in 24.4 per cent. These nodules vary in size from a pea to a child's fist; they are usually associated with tuberculous meningitis, but they may exist alone. When they are large they rank as cerebral tumours, being most frequently seen in the cerebellum. They rarely soften, but may be the seat of calcareous deposits.

Liver.—This is frequently involved in general tuberculosis, although it is doubtful if it is ever the seat of primary infection except in the congenital cases. Usually the only lesion is the presence of miliary tubercles on its surface and in its substance, and in most cases these are not numerous. They are found in about two thirds of the cases. In a smaller number there are tuberculous nodules of various sizes. In nearly every protracted case the liver is markedly fatty. In very late cases of tuberculosis of the bones, it is frequently the seat of amyloid degeneration.

Spleen.—This is more frequently affected than the liver, but in very much the same way. In most of the cases of general tuberculosis, miliary tubercles are present in the spleen, these being usually numerous, both upon the surface and throughout the organ. Not infrequently small tuberculous nodules are also seen, but there are rarely any which are larger than a pea. The size of the spleen is not altered if only miliary tubercles are present; but with the tuberculous nodules it may be much enlarged.

Amyloid degeneration is found under the same conditions as in the liver.

Stomach.—Tuberculosis of the stomach is one of the rare lesions; both its contents and its acid reaction seem to protect it against direct infection from the mouth. Tuberculous ulcers were seen in five of my autopsies, which is a larger proportion than is usually noted.

Intestines.—These are less seriously affected in infancy than in older children, which is rather surprising when we consider how susceptible are the intestines of infants to other forms of infection. The explanation of this difference seems to me to be this: Intestinal infection is nearly always secondary to disease of the lungs; primary lesions being extremely rare. Infants usually die from the more rapid tuberculous processes in the lungs or brain before there has been time or opportunity for intestinal infection to occur. The opportunities for such infection depend upon the number of bacilli which are coughed into the pharynx and swallowed. In infancy this number is small, because of the many who die of tuberculous pneumonia or meningitis before extensive softening in the lungs has taken place. In older children the slower course of the pulmonary disease gives ample time for intestinal infection, while the more extensive softening and excavation are accompanied by the discharge of a much larger number of bacilli. The intestinal lesions and those of the mesenteric lymph nodes with which they are almost invariably associated, are described on page 411.

Peritonæum.—In infancy the peritonæum is not often involved even in general tuberculosis, and at this age it is very rare for it to be the seat of the principal tuberculous process. This occurred but once in my own 119 autopsies. In older children it is more frequent; of the 131 Pendlebury Hospital cases, the peritonæum was involved in 37, or twenty-eight per cent. In most cases of general tuberculosis there are only deposits of miliary tubercles; less frequently there are tuberculous nodules with other inflammatory products. The lesions in these cases are described with Diseases of the Peritonæum.

Thymus gland.—In three of my cases tuberculous nodules were found in the thymus body, the size varying from a small pea to a hazelnut. Some of the largest nodules had undergone softening at the centre. All these were cases showing widely disseminated tuberculous lesions.

Pancreas.—In three of my cases this organ also was the seat of small tuberculous nodules, all of them being cases of general tuberculosis.

Uro-genital organs.—Serious tuberculosis of any part of the urinary tract is very rare in children. Miliary tubercles were found in the kidneys in about one third of my autopsies on tuberculous patients. They are generally few in number. Tuberculous nodules of the kidney I have seen but once in a young child. They are very rare before the fourteenth year (page 670). In two of my autopsies tuberculous nodules were found in the suprarenal capsules. Tuberculosis of the testicle has been observed in rare instances among children, although not in one of my own series. Koplik (New York) has reported several cases.

Tuberculosis of the bones and of the external lymph nodes have already been described.

THE CLINICAL FORMS OF TUBERCULOSIS.

I. GENERAL TUBERCULOSIS.—Cases of tuberculosis present a wide variety in their symptomatology. Almost every case possesses some peculiar features which depend upon the constitution of the patient, the source of infection, the rapidity with which the bacilli are disseminated through the body, or the numbers in which they enter. The general symptoms usually precede the local ones, but in probably the majority of cases they are masked and unrecognised. It is not often possible to recognise tuberculosis until the process is quite well advanced in some one organ. The early symptoms in most cases are very indefinite and susceptible of many explanations.

1. **Cases Resembling Infantile Marasmus.**—In early infancy, tuberculosis often gives at first and for a long time only the symptoms of marasmus. Infants are pale and thin, they do not gain in weight, and finally become emaciated. There is nothing characteristic about these symptoms, and it should be remembered that they depend much more frequently upon simple marasmus than upon tuberculosis. There may be no cough and no fever sufficient to attract attention, and the case may even go on to a fatal termination without any symptoms except those of infantile marasmus. This I have seen at least a dozen times in cases that came to autopsy.

More frequently, however, there are developed toward the end of the disease both the symptoms and signs of pulmonary disease and fever. These are generally found together, as the process in the lungs is the cause of the rise of temperature. The febrile symptoms are often not seen until the last two or three weeks of life. The course of the temperature is irregular. It is never of the hectic type and rarely high. The usual range is between 100° and 102° F. The pulmonary symptoms are generally few and not very well marked. There is usually some cough, but it is rarely severe. The breathing is more rapid than would be explained by the temperature alone. Severe dyspnœa and cyanosis are rare, and are seen only at the close of the disease. The physical signs are those of either localized bronchitis or of broncho-pneumonia.

The other symptoms usually relate to the digestive tract. There may be indigestion, with occasional vomiting and green undigested stools, or there may be diarrhœa. The intestinal symptoms depend on the general condition of the child and the constitutional disease, rarely upon a tuberculous process in the stomach or bowels.

If the case has gone on to the development of constant fever and recognisable physical signs which slowly spread, the infant's fate is sealed. The progress of the case from this time is steadily downward, and the child can live at most but a few weeks. Death generally occurs from progressive asthenia without the development of any new symptoms. Occasionally toward the close, cerebral symptoms rapidly develop, and the child is carried off in a few days by tuberculous meningitis; sometimes there is a rapid spreading of the disease in the lungs, and death occurs with symptoms of simple acute pneumonia.

Diagnosis.—The difficulty in diagnosis is chiefly during the first year of life. Every circumstance in the patient's surroundings and family history which bears upon the development of tuberculosis must be weighed to establish the fact of inheritance or of exposure to contagion. In simple wasting, the usual history is that the infant was plump and well nourished at birth. A sufficient cause for its condition can in most cases be found in improper or insufficient nourishment or the want of proper care. (See causes of marasmus, page 238.) Often the wasting follows some acute disease of infancy, most frequently some form of gastro-intestinal disease.

In tuberculosis, the infant may show all the signs of malnutrition at birth, but in most cases they are of later development. They either come without adequate cause, or are associated with pulmonary disease or they follow measles or pertussis. No explanation of the wasting can be discovered in the food, the surroundings, or in the condition of the digestive organs. Diarrhœa and vomiting more frequently follow than precede it. The above facts are sufficient to warrant a suspicion only that tuberculosis is present until some local manifestation occurs, usually in the lungs. The early wasting without adequate cause, followed by the gradual development of low fever, and finally the appearance of signs of subacute broncho-pneumonia, form the most characteristic features of general tuberculosis in early infancy. Yet all these symptoms are occasionally met with in cases in which the autopsy shows none of the lesions of tuberculosis, for simple broncho-pneumonia frequently occurs in patients suffering from marasmus; but in such cases fever is usually slight and it may be absent.

The wasting and cachexia of hereditary syphilis sometimes resemble tuberculosis, but the early history in syphilis is usually so characteristic, and other symptoms of the disease are so rarely wanting, that the mistake is not likely to be made if a patient is submitted to a careful examination. In the absence of definite syphilitic symptoms the chances are greatly in favour of tuberculosis.

2. **Cases in Older Children with Symptoms Resembling a Continued Fever.**—Before the development of fever in these cases, there is usually quite a protracted period of very indefinite symptoms, each one of which alone is unimportant, but all of which taken together should excite sus-

picion. Such children are usually delicate; they are persistently anæmic without sufficient reason; they often show a loss in weight; there is a marked cachexia, sometimes a capricious appetite, and a digestion easily disturbed. In some of them a change in disposition is observed, and they become peevish or fretful and are disinclined to muscular exertion. All these symptoms indicate a gradual decline in the general health.

This clinical picture may be due to many causes, but it should always arouse in the mind of the physician a suspicion of incipient tuberculosis, particularly in a child who by surroundings or inheritance is predisposed to that disease. After these indefinite symptoms have lasted a few weeks fever is added. Sometimes the prodromal symptoms are absent or unnoticed and fever is the first evident symptom. This fever is peculiar in that it comes without evident cause and without any local manifestations of disease. The temperature is not often high, but it is continuous. The tympanites and the rose-coloured spots are not present, but the general aspect of the patient is strikingly like that belonging to typhoid fever.

After the fever has lasted from one to three weeks there develop some signs of localized tuberculosis, generally in the lungs, or the fever may decline gradually, and although the patient improves he does not get well. He is still weak and does not gain in weight, and the thermometer shows the existence of a very slight amount of fever. Before long he may grow rapidly worse and the course of the temperature becomes irregular, with alternate exacerbations and remissions. Such an irregular and inexplicable fever sometimes puzzles the physician for three or four weeks before the characteristic features which stamp the process as tuberculous are present. One general symptom is almost invariably associated with the fever, viz., wasting. This may not be rapid, but is progressive. The tuberculous cachexia is frequently unmistakable; but in most of the cases one must wait for the process to advance far enough in some one of the organs to give local signs or symptoms before he can be sure of tuberculosis. In four cases out of five this is in the lungs. Less frequently it is in the peritonæum, the brain, or a general infection of the lymph glands throughout the body. If in the lungs, the process manifests itself as a broncho-pneumonia whose tuberculous character may be suspected from its localization—the apex or the middle of the lung in front—but chiefly from the fact that the general symptoms, fever and wasting, have for so long a time preceded the local signs of disease. From this time, the course of the disease may be that of a typical tuberculous broncho-pneumonia.

If the tuberculous process is localized in the brain, we have dulness, vomiting, headache, apathy, irregular pulse, irregular respiration, and finally convulsions and coma—in short, the symptoms of tuberculous meningitis; if in the peritonæum, we have abdominal distention from gas or fluid, tenderness, pain, diarrhœa, or constipation; if in the lymph glands, there is a general enlargement of those situated in the neck, and sometimes those of the axillary and inguinal regions, with symptoms indicating similar changes in those at the root of the lung.

Diagnosis.—In distinguishing general tuberculosis from typhoid fever, very great stress is to be laid on the family and previous history of the patient and the surroundings, as favouring tuberculosis. On the other hand, the prevalence of typhoid fever in the family, the neighbourhood, or the institution in which the case occurs, is important. The extreme infrequency of typhoid in children under two years old should always lead the physician to scrutinize very carefully every case in which he is disposed to make such a diagnosis at that time of life. In typhoid, the course of the fever is more regular than in tuberculosis, but less so than in the typhoid of adults, and the spleen in nearly every case is sufficiently enlarged to be easily felt below the ribs. The rose spots are usually present. But the most conclusive evidence is that afforded by the blood reaction in Widal's serum-test; without this, by the gradual cessation of the fever in the third or fourth week and complete recovery of the patient.

In tuberculosis, on the contrary, the fever is less regular. It commonly shows wider fluctuations, the spleen is not usually enlarged, and there are no rose spots. Tympanites and abdominal tenderness are sometimes seen, but the fever shows no disposition to stop after the third week, and the wasting is continuous. The signs in the lungs, at first few, increase from day to day. In most cases one must wait for ten days at least, and in many three weeks, before a positive diagnosis can be made.

II. TUBERCULOUS BRONCHO-PNEUMONIA.—This occurs clinically under the following conditions: (1) It may begin in the lungs or extend to the lungs from the bronchial glands, the symptoms in either case being essentially pulmonary from the outset. (2) It may follow either form of general tuberculosis described—that resembling marasmus in infants, or that resembling a continued fever in older children. In both of these the pulmonary symptoms develop gradually in the course of the general symptoms of the disease. (3) It may occur in the course of any of the forms of local tuberculosis,—of the bones, peritonæum, intestines, external lymph glands, or skin. In such cases the invasion of the lungs frequently marks the last stage of the process. (4) It may follow any of the infectious diseases, especially measles or pertussis, even though they are not complicated by broncho-pneumonia, but more frequently when they are. (5) It may follow single or repeated attacks of simple bronchitis or pneumonia.

Clinically the cases may be divided into three groups: First, the most rapid ones, lasting from one to three weeks; secondly, those running a more protracted course, with a duration of from three weeks to three months; thirdly, those which are more or less chronic. In the first two groups the progress is nearly always steadily downward, and a fatal termination the almost inevitable result; in the third form the course is more irregular, and marked by a series of exacerbations and remissions.

1. **The Most Rapid Cases.**—In this form of the disease there are found scattered through certain portions or nearly the whole of both lungs, miliary tubercles and minute tuberculous nodules, the intervening parts of the lung being involved more or less seriously in a simple inflammation. In most of the cases the clinical picture is that of simple acute broncho-pneumonia, for it is to the accompanying broncho-pneumonia, and not to the scattered tuberculous deposits themselves, that the symptoms and the physical signs are due. The development of the disease, although acute, is not usually abrupt. There are present, fever, cough, dyspnœa, accelerated respiration, prostration, and sometimes cyanosis. The temperature in these cases is never hectic, but its course is a somewhat irregular one the usual range being between 100° and 104° F. In most of the cases it differs in no respect from the temperature of simple broncho-pneumonia. Sometimes it is seen that the general symptoms are severe and the physical signs wide-spread, and yet the range of temperature is not high. To be sure, this is occasionally seen in a simple broncho-pneumonia, but it is more frequent in tuberculosis. The cough early in the disease is slight, but later becomes severe and often distressing. In infants and young children it may be of a paroxysmal character, resembling pertussis. Expectoration is wanting in infancy, and is not often seen in those under seven years, so that bacilli in the sputum is a symptom of only a small number of cases. Bloody expectoration, likewise, is rare in children.

The conditions in the lungs which give physical signs are bronchitis of the smaller tubes, with areas of complete or partial consolidation. In character, these signs are identical with those of simple broncho-pneumonia (page 547). They may be scattered throughout the whole of both lungs; but when localized they are more frequently in the upper than in the lower lobes, and rather more frequently in front than behind. Although both lungs are involved, they are usually not affected to the same degree. The patient may die before signs of complete consolidation are present; more often there are during the last few days small areas of partial consolidation, as shown by broncho-vesicular breathing, exaggerated voice, and slight dulness. These signs may be due to the simple broncho-pneumonia, and are often found in the lower lobes behind. Large areas of complete consolidation, with pure bronchial breathing, bronchial voice, and marked dulness are infrequent.

From the beginning of acute symptoms the progress of the disease is steadily downward, death resulting from the same causes as in simple broncho-pneumonia. The end is marked by cyanosis, great dyspnœa, weak pulse, and extreme prostration. In a few cases there develop shortly before death cerebral symptoms, indicating tuberculous disease of the brain. Such symptoms may be the first to lead the physician to suspect the process to be a tuberculous one. In these cases death may occur in convulsions in two or three days from the first cerebral symptoms. In other cases the course is slower, with the typical symptoms of meningitis.

2. **The More Protracted Cases.**—In this form of the disease there are found in the lungs caseous nodules, with larger areas of caseous pneumonia, and usually some spots of softening. The process is not usually so generalized as in the cases just described, but as in them there is always associated a certain amount of simple pneumonia. This is the most frequent and most characteristic form of pulmonary tuberculosis in infancy and early childhood. Its usual duration is from one to three months; its course is then steady and uninterrupted. In its slower or subacute form it lasts from three to six months, and its course is then more irregular.

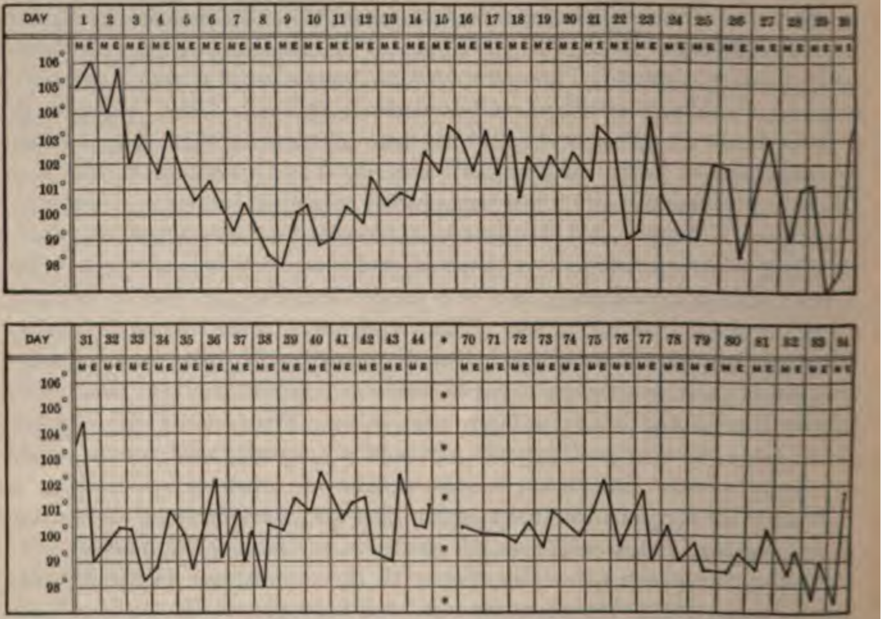


Fig. 211.—Tuberculosis following measles. Child sixteen months old, inmate of an institution. Chart begins on fifth day of a severe but uncomplicated attack of measles, and shows a natural decline to normal. Fever then returned and continued till death, twelve weeks later. Record for the period which is omitted was much like that which immediately precedes and follows. Early symptoms not acute, only slow wasting, slight cough and fever, with scattered râles throughout chest. Signs of consolidation not distinct till eighth week, then present in right upper lobe. Toward the end, rapid emaciation, marked pulmonary symptoms, and signs of cavity at right apex. Autopsy showed a large cavity, extensive tuberculous deposits throughout both lungs and in nearly all abdominal organs.

The mode of onset will depend upon the conditions under which the disease develops. When the general symptoms of tuberculosis—fever and wasting,—have preceded those in the lungs, the evolution of the latter is gradual, with cough, rapid breathing, dyspnœa, increased prostration, etc. When the pulmonary symptoms are present from the beginning, they are the same as in simple broncho-pneumonia, with the exception that they usually come on less acutely. The latter is true of cases which are secondary to some other form of tuberculosis in the bones, peritonæum, etc.

When pulmonary tuberculosis follows measles (Fig. 211) or whooping-cough which has been complicated by simple pneumonia, the early symptoms may present no unusual features. After two or three weeks the temperature gradually falls, and the physical signs improve, but neither quite disappears. The cough continues, though its severity somewhat abates. In the course of a few weeks the child, who has meanwhile improved somewhat in his general condition, becomes distinctly worse, often without any assignable cause. The temperature rises to 102° or 103° F.; the cough increases, and an extension of the disease in the lungs is evident by the physical signs. In other cases the progress of the disease after the pneumonia which complicated measles is without an intervening period of apparent improvement. It sometimes happens that the attack of measles or whooping-cough is not accompanied by any serious pulmonary symptoms, and the case goes on to apparent recovery, except that there remain anæmia, a slight cough, and fever. The temperature, although not high, persists; but it may be two or three weeks before there are present definite symptoms and signs of disease in the lungs.

Fever is a constant accompaniment of all active tuberculous processes in the lungs in the child as in the adult, it being absent only during the periods of remission which occur in the cases of slow and irregular progress. It is a very important guide to the progress of the disease. The early fever depends chiefly upon the coexisting broncho-pneumonia, and its course resembles that of simple pneumonia of the protracted variety. There is no typical curve. The fever is not often steadily high, and in many cases it is never high (Fig. 212). It frequently runs for

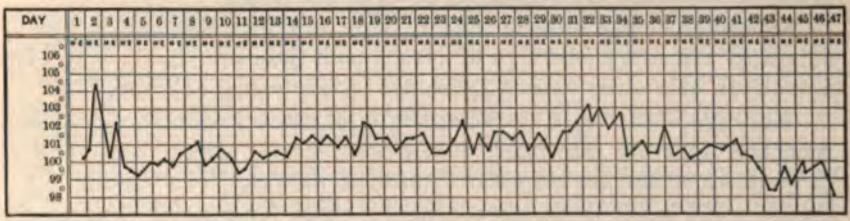


FIG. 212.—Tuberculous pneumonia, general tuberculosis. Patient eleven months old, and under observation at the time he was taken sick. Chart of entire illness is given. Disease began as an acute pneumonia in lower part of left axilla and spread to entire lower lobe. Early signs of consolidation; at end of two weeks, flatness so marked that a needle was inserted, fluid being suspected. Vomited frequently, and had loose discharges from bowels throughout the illness; abdomen much swollen for last two weeks. Autopsy showed cheesy pneumonia of part of the upper and the entire left lower lobe, where were two small cavities. Recent tubercles found throughout right lung, and extensive deposits in abdominal organs with peritonitis, intestinal ulcers, etc.

several days between 99° and 102° F., and then, without evident cause, rises to 104° F. or over; again, it may be scarcely over 100° F. for days together. In infants the morning temperature is frequently subnormal, although the evening temperature may be 102° or 103° F. Even toward the close of the disease, when softening and breaking down are actively going on, the regular hectic temperature of adults is rarely seen in a young child (Fig. 213). While the presence of fever is of great significance, its course has almost no diagnostic importance in early life. Especially should one beware of drawing the conclusion that, because the fever is not hectic, there is no breaking down of the lung.

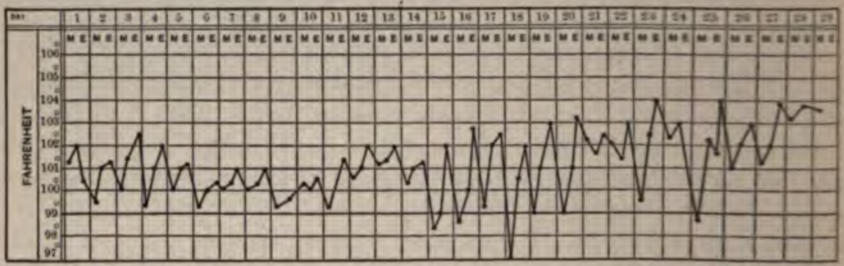


Fig. 213.—Tuberculous pneumonia, with extensive softening and excavation. A delicate child, thirteen months old; weight, 10 pounds; came under observation four weeks before death, with consolidation at apex of right lung. Signs increased in intensity, and extended in area until there were heard, from clavicle to below the nipple, exaggerated bronchial voice and breathing and many moist râles; percussion note was flat; behind, the same signs at extreme apex. No distinct signs of a cavity; no hectic fever; no sweating. Autopsy showed large cavity (Fig. 207) at right apex partly filled with caseous masses; diffuse caseous pneumonia (Fig. 206) of the rest of right upper lobe, with scattered deposits in the other lobes, the opposite lung, and a few in the abdominal organs.

Sweating belongs only to the late stage of the disease, and is usually associated with the hectic type of fever; both these are regular symptoms in children over seven years old, but not in very young children.

Wasting, like fever, is characteristic of all active tuberculous processes. Whenever they are associated, tuberculosis should always be suspected, no matter how obscure the other symptoms may be. The wasting is not always rapid, but it is usually continuous while fever lasts. During the periods of temporary improvement, children may not only cease to lose, but may actually gain in weight. In the early stage of the disease, wasting is especially suggestive when it continues without apparent cause after measles or pertussis, or when it persists under other circumstances in spite of a good appetite and apparently good digestion. It may at first be so slight as not to be noticed unless the scales are employed. In obscure cases this steady loss of weight is a point of much diagnostic value, and is frequently overlooked. Toward the close of the disease there is rapid and frequently extreme emaciation.

Cough, although almost invariably present, shows no peculiarities. It may be hard, dry, or suppressed; it sometimes occurs in paroxysms resembling pertussis, which may or may not depend upon the presence of enlarged bronchial glands.

Expectoration is absent in infants, the matters coughed up being swallowed. In children over seven years old, we often get a profuse muco-purulent expectoration, but it is very exceptional below this age.

Hæmoptysis is a rare symptom, but not unknown even in young children. Henoch has reported a case of fatal hæmoptysis in a child ten months old, where the hæmorrhage was due to the rupture of an aneurism in the wall of a cavity. Herz, in 247 clinical cases of tuberculosis in children, records 8 of hæmoptysis—4 of them under five years, and the youngest only eighteen months old. The records of 131 autopsies on tuberculous children in the Pendlebury Hospital, show that hæmoptysis was four times a cause of death; two of these patients were under five years, and one was only twelve months old. I have never met with a case of hæmoptysis under five years old. As in adults, fatal hæmoptysis is usually due to the opening of a large vessel by ulceration in the wall of a cavity, which is sometimes in the lung and sometimes in one of the bronchial glands.

The respiration in all cases of tuberculous pneumonia is accelerated, and usually out of proportion to the rise in temperature. As the lung becomes more and more extensively invaded there is constant dyspnœa. The pulse is rapid in the early stage, and continues so throughout the disease; toward the end it becomes weak and irregular. Irregular respiration and a slow, irregular pulse, may occur at any time from the development of cerebral complications.

Pleuritic pains in the chest are not frequent in children. Gastro-intestinal symptoms, such as indigestion, vomiting, diarrhœa, etc., are generally present, but are not peculiar in this disease. They usually depend upon the patient's general condition, only exceptionally upon tuberculous disease of the stomach or intestines. The characteristic symptoms of intestinal tuberculosis—abdominal pain, tenderness, uncontrollable diarrhœa, and intestinal hæmorrhage—are not often met with in children under five years. With such symptoms, and sometimes when they are doubtful or absent, careful palpation of the abdomen may disclose the presence of enlarged mesenteric glands. When these are not readily felt through the abdominal walls, they may sometimes be discovered by a rectal examination after the method of Carpenter (London).

The spleen is often enlarged, sometimes very much so, but this does not occur with sufficient frequency to be of much diagnostic value. It may be due to tuberculous deposits, to causes connected with the lungs or heart, or to fever. The liver is never enlarged from tuberculous deposits, but may be so from amyloid or fatty degeneration, or from obstructed circulation, as in the case of the spleen.

Dropsy is rare and seen only toward the close of the disease. It may depend upon anæmia, upon complicating nephritis, especially amyloid degeneration, upon cardiac or pulmonary conditions leading to interference with the return circulation, or upon pressure of tuberculous retro-peritoneal or mesenteric glands upon the inferior vena cava. Clubbing of the fingers is occasionally seen in cases running a very protracted course, and is due to obstructed circulation.

Anæmia is commonly associated with wasting, and it is of special importance where the latter is slight or absent. It is a frequent sequel of acute disease in infancy when not dependent on tuberculosis; when, however, it is associated with low fever, cough, and persistence of râles in the chest, it should always excite apprehension.

3. **Chronic Tuberculous Pneumonia.**—In young children this is a chronic interstitial pneumonia associated with tuberculous deposits. These cases have usually had their beginning in one of the more acute forms just described. The primary attack runs a tedious, protracted course; there is a slow convalescence and apparent recovery, although this is not complete. Often a slight cough remains, or returns from the slightest exposure or other exciting cause. The child does not regain his former weight or vigour, and careful examination of the lungs shows that some abnormal signs remain. There are frequently present feeble breathing and slight dulness over the affected part of the lung, and occasionally friction-sounds may be heard.

After a few months, possibly, the child has another attack resembling the first and running the same tedious course. It is accompanied by fever, cough, and perhaps there is a fresh consolidation of some part of the lung, generally in the neighbourhood of the old disease. All active symptoms finally subside, and most of the signs of recent disease disappear; but it is usually found then that the lung is not quite in so good condition as it was before this second illness. The acute attacks may be repeated several times and pass under the name of bronchitis, broncho-pneumonia, or pleurisy. They may extend over a period of two or three years or even longer. The general health in the interval is not good, there being present in most cases anæmia, with the usual symptoms of malnutrition; the children are regarded as being very delicate.

The course of this disease thus differs in no essential particulars from that of simple chronic broncho-pneumonia (page 583); the physical signs likewise are identical in character, although they may differ in their location. They are generally found in the same situations as are the signs in the more rapid forms of pulmonary tuberculosis in early childhood. A fatal result in these cases is usually brought about in one of three ways: (1) by the development of acute tuberculous pneumonia or miliary tuberculosis of the lungs, occurring with the symptoms of one of the previous exacerbations which has come on without apparent cause or perhaps has followed an attack of measles or whooping-cough; (2) by tuberculous meningitis; (3) by a simple acute broncho-pneumonia.

Physical Signs of Tuberculous Pneumonia.—Speaking generally, there is no difference in a young child between the signs of a tuberculous and those of simple broncho-pneumonia except in their position; for cavities, although they are present at autopsy in most of the cases, are very rarely of such size and so situated as to be recognised during life. In children over seven or eight years old, and sometimes in those of five or six, the signs are essentially like those in adults.

By reference to the description of the lesions (page 1077) it will be noted that the upper lobes are the seat of the most advanced disease twice as frequently as the lower lobes, and the right lung rather more frequently than the left. When the disease is in the upper lobes it is rarely at the extreme apex, and when it is in the lower lobes it is very exceptional to find it at the base, posteriorly. The region most often involved is the middle zone of the lung. If the signs appear first behind they are, in the great majority of cases, in the interscapular space; if in the lateral part of the chest, they are in the middle or upper part of the axilla; if in front, they are in the mammary region, more frequently above than below the nipple, but rarely extending quite to the clavicle. In other words, it is near the root of the lung that the disease most frequently begins, spreading thence forward more often than backward. The explanation of this is found in the fact that the disease in infants and young children so often extends from the lymph nodes at the root of the lung to the lung itself. The physical signs themselves may be grouped under four heads, corresponding to the pathological conditions existing in the various stages of the disease—viz., (1) localized bronchitis; (2) partial consolidation; (3) complete consolidation; (4) excavation. The early signs in the first two stages are identical with those described in broncho-pneumonia, those of the third stage being the signs of the persistent form. As a rule, however, the transition of the signs from one stage to another is much slower in tuberculous than in simple broncho-pneumonia.

As stated in the description of the lesions, cavities are found in the lungs in the majority of cases of infants dying from tuberculosis of the lungs. It is, however, rare that they can be recognised in children under three years old. From three to eight years they give more positive signs, and after eight years practically the same signs as in adults. The reason why in infancy cavities are so seldom recognised during life is because they are generally small, often centrally located, nearly always filled with thick pus or cheesy matter, and rarely communicate freely with the bronchi. On the other hand, it is very common to find signs in young children which, if heard in adults, would be regarded as almost positive evidence of a cavity, although none is present. These signs are cracked-pot resonance and cavernous breathing. They are not usually due to bronchiectasis, since this condition belongs to chronic cases, and especially to older children; but most frequently to consolidation about a large bronchus superficially situated—viz., below the clavicle, high in the axilla and in the interscapular region. The wide area over which this broncho-cavernous breathing is heard, is one of the most striking points of difference from the signs of a cavity.

Course, Duration, and Termination.—Whatever may be the evolution of the symptoms, and the variations are almost endless, the cases fall readily into two groups,—those in which the progress is rapid and steady and those in which it is slow and intermittent. The duration of the first group is from four to eight weeks. Fever is constant, wasting progressive, and the physical signs show a steady advance of the disease in the lungs. Dyspnœa becomes severe and constant; the pulse grows more and more rapid and feeble; and death occurs from exhaustion, pulmonary œdema, or syncope, less frequently from meningitis.

In the second group the duration is from two to twelve months. The course can not better be described than as a succession of attacks of broncho-pneumonia, sometimes separated by an interval of several weeks, at other times one coming on before the first is fairly over. During exacerbations the symptoms resemble those of the first form, there being marked fever, wasting, cough, and dyspnœa. The child may seem hopelessly ill when, without any special reason, a change for the better occurs, the acute symptoms abating and the signs of consolidation in great measure disappearing. Toward the end of the disease the pulmonary and constitutional symptoms become constant, and frequently there are added symptoms due to extension of the tuberculous process to other parts of the body—the brain, peritonæum, intestines, mesenteric glands, etc. These cases die, as do the more acute ones, from the local disease in the lungs or from general infection.

Diagnosis.—The evidence upon which a diagnosis of tuberculosis is made, is of two kinds—that which relates to the patient and that which relates to the local disease. In any case, a diagnosis is reached by weighing the evidence as a whole rather than by relying upon the presence of particular symptoms or physical signs. One should investigate the family history, surroundings, and previous condition of the patient; also the mode of onset, and course of the disease, and consider the evidence afforded by the examination of the patient.

A careful examination of the family history should be made to determine, first, the existence of phthisis in the parents or in other members of the family, near or remote. Children more often inherit a predisposition from the mother than from the father, and are more likely to contract it from her, owing to the closer contact. It is not enough simply to investigate the question of phthisis. Inquiry should be made regarding meningitis, disease of the cervical glands, spine, hip, knee, or ankle, especially in the other children of the family. These points are important not only to establish the fact of heredity but also the probable chances of exposure. Other conditions favourable for acquiring the disease should be considered, such as, in a private family, exposure to nurses or other members of the household; also whether the surroundings have been such as would give opportunities for infection, as in cases where a child has been reared in a tenement house, or has been long an inmate of a hospital or other institution. In the child's previous history, it is important to know whether there have been other manifestations of tuberculosis in the cervical glands, spine, hip, knee, or ankle, or the skin; also whether he has been liable to attacks of severe or protracted bronchitis or broncho-pneumonia. If he has had measles or pertussis, it is important to know whether they were severe, accompanied by pulmonary complications, or followed by a protracted cough or obscure fever. The child's general constitution should be considered, whether he is delicate, narrow-chested, poorly nourished, or anæmic.

In its symptoms and course it is with simple broncho-pneumonia that tuberculous disease is likely to be confounded. The onset of simple pneumonia is usually rapid and often abrupt; tuberculous pneumonia more frequently develops gradually with constitutional symptoms preceding the local ones by several days or even weeks. When tuberculosis develops rapidly, the pulmonary symptoms and the physical signs may be identical in the two conditions. One is often struck during the acute stage with the disproportion between the general symptoms—loss of flesh, prostration, and temperature—and the local evidences of pulmonary disease. When the patient dies in the early acute stage the disease is rarely recognised, nor, indeed, can it be diagnosticated with certainty. Usually it is not until the time for resolution to occur that the course of the disease suggests something different from broncho-pneumonia. The question then arises whether we have to deal with a case of persistent broncho-pneumonia or with tuberculosis. It should be remembered that it is not infrequent for simple broncho-pneumonia to resolve slowly or to go on to the development of chronic interstitial pneumonia; and that local conditions as determined by physical signs, which in adults would be regarded as certainly tuberculous, very often in children are simple processes.

Often the course of the disease, after the first acute period has passed, furnishes further evidence to clear up the diagnosis; but not necessarily, for in tuberculosis it may be steadily downward, or it may be marked by periods of remission and exacerbation, and the same is true of simple pneumonia. Fever is a more constant symptom in tuberculosis, and it is usually higher than in persistent broncho-pneumonia; but the exceptions are so many and the variations so wide that it is not safe in young children to lay very much stress upon the temperature curve. Anæmia and wasting are more marked in tuberculosis, and in most cases progressive. A copious muco-purulent expectoration is seen almost as frequently in pneumonia as in tuberculosis; but in neither disease is it common under five years. The presence of the bacillus tuberculosis in the sputum is, of course, positive evidence of tuberculosis.

With infants and young children the only satisfactory method of obtaining the sputum for examination is to pass the stomach-tube well into the œsophagus, and stain the mucous which adheres to it when withdrawn. This procedure, first employed I think in the Babies' Hospital, has been in constant use by us in that institution for several years with the most satisfactory results.

Simple broncho-pneumonia may affect any part of the lungs, but by preference the lower lobes posteriorly. The signs of tuberculosis may likewise be found anywhere, but most frequently in the anterior part of the lung, the mammary region, the axillary margin, or the apex; if posterior, the signs are usually at the apex or in the interscapular region. From the character of the physical signs, no inference can be drawn unless a cavity can be positively made out; but when the process has advanced to that stage, the diagnosis is generally plain from the general symptoms.

Tuberculin with older children is quite as useful for diagnosis as with adults. With infants and very young children, on account of the well-marked fever which is usually present, it is less frequently applicable.

Meningitis developing during a pulmonary disease of doubtful character, is generally tuberculous, and its occurrence is usually to be interpreted as establishing the tuberculous nature of the process in the lungs. The development of cheesy lymph glands in the neck, the groin, or axilla, or the presence of symptoms pointing to enlargement of the bronchial glands, or those of chronic peritonitis with or without ascites, or intestinal hæmcrrhage—all point strongly to tuberculosis.

If the acute symptoms begin during measles and persist, they may be due either to broncho-pneumonia or to tuberculosis. If, however, they begin insidiously during convalescence from measles, they are very probably due to tuberculosis. If the symptoms begin acutely during pertussis, they may be due to simple broncho-pneumonia or a tuberculous process; but if they develop gradually and insidiously after pertussis, the disease is probably tuberculosis. It should not be forgotten, however, that it is not uncommon for simple broncho-pneumonia occurring with pertussis, to persist two or three months, or until the attack of pertussis has subsided.

If the child was previously healthy and living in good surroundings, and if the disease began with acute symptoms, the process is simple pneumonia in nine cases out of ten, no matter how irregular its course, how prolonged its duration, or what the physical signs. Still, after all has been said, the diagnosis is in all cases difficult, and in some, particularly the more chronic ones, a positive diagnosis is impossible, as no one knows so well as he who has an opportunity to follow his cases to autopsy.

III. CHRONIC PHTHISIS.—This form of tuberculosis, with its chronic hectic fever, slow cavity formation, progressive emaciation, night sweats, etc., is very rarely seen before the fifth year, and it is not at all frequent until the tenth or twelfth year. In its symptoms, course, termination, and physical signs, it resembles the same disease in adults, and need not be described at length here.

IV. TUBERCULOSIS OF THE BRONCHIAL LYMPH NODES (BRONCHIAL GLANDS).—This condition is usually associated with some form of pulmonary tuberculosis, but it may exist as the most important and sometimes as the only tuberculous lesion.

Its symptoms are usually associated with those of pulmonary or general tuberculosis; but they may occur when the pulmonary changes are too few to be recognised either by symptoms or physical signs. From the great frequency with which this lesion is found in infants and young children, it might be expected that local symptoms would be common in such patients. They are, however, in my experience, quite exceptional. Most of the cases in which well-marked symptoms occur are in children over two years old, and it is between the third and tenth years that they are usually seen. In infancy, although these glands are almost invariably affected, death in the great majority of cases occurs from the pulmonary disease, before the later changes in the glands have had time to develop.

General symptoms indicating a tuberculous cachexia may or may not precede the local ones. The latter are chiefly mechanical, and depend upon the size of the glands and upon their anatomical relations, and very little or not at all upon the nature of the changes in them. The most important relations, so far as the production of symptoms is concerned, are those which they bear to the pneumogastric and recurrent laryngeal nerves, the superior vena cava, the trachea, and bronchi; those less important are to the aorta, pulmonary artery, and œsophagus.

Pressure upon or irritation of the pneumogastric or recurrent nerves produces cough, dyspnœa, and sometimes a change in the voice. The cough is hoarse, persistent, and teasing, and frequently occurs in paroxysms which in many respects resemble those of pertussis, but it lacks the characteristic whoop, and is not accompanied by the expectoration of the mass of tenacious mucus. These paroxysms are severe and often prolonged, but careful observation shows distinct differences from those of pertussis, though by an unfamiliar ear the two are easily confounded. The dyspnœa, like the cough, is paroxysmal, and sometimes strongly resembles ordinary spasmodic croup; at other times it is like a severe attack of asthma. Such symptoms may come and go, but they are frequently prolonged, and usually in the interval between the severe seizures the patient is not wholly free from dyspnœa. Although the chief cause of dyspnœa is no doubt nerve irritation, it may be due in part to pressure upon the trachea or one of the large bronchi. In dyspnœa from pressure on the trachea the head is usually thrown back, and the obstruction is more frequently on expiration than on inspiration.

After such symptoms as those mentioned have existed for a few days or weeks, and in some cases without any warning, there may occur a sudden attack of asphyxia which may prove fatal. This is generally due to ulceration of a caseous gland into the trachea or a large bronchus and the escape of a large mass into the air passages, where it produces the same effects as any other foreign body.

Loeb has collected fifteen cases of this description, a summary of which gives a good idea of the circumstances under which this accident usually occurs: In four cases death took place in the first attack of suffocation, the only previous symptom having been cough; in three there had been a number of milder attacks extending, in two of the cases, over a considerable period before the occurrence of the fatal one; in three, death occurred in the first attack, in children who had no previous cough and who were apparently healthy; in one, the fatal attack came on during pertussis. In the majority of the cases, death followed in from five to ten minutes from the first symptom; in a few the patients lived for an hour. In rare cases after ulceration into the trachea, the patient has coughed up a large quantity of foul pus, and recovered.

Pressure upon the superior vena cava is usually associated with spasmodic dyspnœa and cough, and causes cyanosis of the face and blueness of the lips. There is frequently a puffiness of the face, and there may be marked œdema. The coexistence of cyanosis with such œdema, when the urine is free from signs of renal disease, should always lead one to suspect pressure at the root of the lung. In some rare cases the interference with the return circulation has been so marked that meningeal hæmorrhage has resulted. By a process of ulceration set up by these glands they may open, not only into the air passages, but into the pericardium, the œsophagus, or any of the large vessels. The last mentioned is usually followed by instant death. Aldibert reports two cases in which the pulmonary artery was opened, death occurring from hæmoptysis, as there was also a communication with one of the large bronchi. In Vogel's case the subclavian vein was perforated, and death resulted from the entrance of air. If ulceration takes place into the surrounding connective tissue, a mediastinal abscess may result, producing any of the pressure symptoms noted above, and, in addition, dysphagia from pressure on the œsophagus. Such an abscess may point in the supra-sternal notch; it may open through the chest anteriorly between the ribs or at the xiphoid cartilage; or it may burrow along the œsophagus to the peritoneal cavity. As a rule, however, patients die of general tuberculosis before the local conditions have advanced so far.

Physical Signs.—In order to produce physical signs, the mass of tuberculous lymph nodes must be large enough to form a mediastinal tumour, or so situated as to produce pressure on the trachea or bronchi. As a rule, the signs are more characteristic behind than in front. Percussion may give dulness anteriorly over the first piece of the sternum or posteriorly along one or both sides of the spine from the second to the fifth dorsal vertebra; the dulness is rarely complete. Auscultation posteriorly may give in the most marked cases amphoric or cavernous breathing, or exaggerated bronchial breathing with prolonged expiration, in those which are less pronounced. Large, moist râles are sometimes heard. The auscultatory signs are so like those of a cavity that it is often difficult to believe that a cavity does not exist. The sounds heard appear to be those produced in the trachea and bronchi transmitted to the ear with great exaggeration by the mass of lymph nodes which surrounds them and fills the space between them and the chest wall. When the head is thrown back a venous hum may sometimes be heard. If one of the primary bronchi or one of its lobar divisions is compressed, there may be very feeble respiration over one lung or one lobe; if the pressure is sufficient to prevent the entrance of air, or if one of these large tubes has been plugged by a caseous mass, there is an absence of respiratory murmur over a single lobe or an entire lung. This sign is of great diagnostic value, but it is not often present.

Diagnosis.—Enlargement of the bronchial glands to a sufficient degree to produce symptoms, may occur in syphilis, in Hodgkin's disease, and in various forms of malignant disease of the mediastinum. A certain amount of swelling is seen in nearly all cases of simple bronchitis or pneumonia, especially in those running a subacute or chronic course. Whether this simple hyperplasia is ever sufficient to cause such symptoms as those mentioned is exceedingly doubtful. I have myself never known it to produce anything more marked than a spasmodic cough. The great infrequency of other forms of enlargement to a sufficient degree to be of clinical importance, usually warrants us, from the symptoms mentioned, in making the diagnosis of tuberculosis. The development in a child of a chronic abscess in the anterior mediastinum, is almost always due to tuberculous glands; and so is one in the posterior mediastinum, provided Pott's disease can be excluded.

The most important points for diagnosis are the association of a spasmodic cough with paroxysms of dyspnœa resembling asthma or croup, and œdema or congestion of the face. More stress is to be laid upon the symptoms than upon the physical signs; the latter are at most only confirmatory. The chief difficulty in diagnosis is found in those cases which present few or no other signs of tuberculosis, and which come first under observation with attacks of dyspnœa or asphyxia resembling laryngeal stenosis. In many such cases tracheotomy has been done without finding any cause for the dyspnœa, the autopsy showing it to be due to ulceration and impaction of a caseous gland.

General Prognosis of Tuberculosis.—The outlook for a young child with general or pulmonary tuberculosis is always bad. So long as the disease remains confined to the lymph nodes, the child is not usually in danger, except from accidents connected with their softening and ulceration, which after all are rare. Spontaneous cure may occur in these glands in the same way as in others in the body—viz., by encapsulation, calcification, etc. Such a result is no doubt a very frequent one; exactly how often it occurs it is impossible to say. But when once the disease has gained any headway in the lung itself, its steady advance is almost certain in a young child. In those who are older and have more resistance the chances of an arrest of the process are much greater.

If the bacilli have gained entrance into the body in any considerable numbers, even though they are shut up in an encapsulated, caseous, bronchial gland, the patient is never free from the danger of general infection.

Prophylaxis.—The prevention of tuberculosis must have constant reference to its cause. The first essential is the destruction of the tubercle bacilli wherever they exist. Since most of the germs existing in the air are derived from the sputum of patients affected with pulmonary tuberculosis, it should be insisted upon, everywhere and at all times, that the sputum from such cases should be collected in special cups or cloths and destroyed either by germicides or by fire. The next point is to avoid needless exposure. A tuberculous mother should on no account nurse her child nor kiss it upon the mouth. A wet-nurse likewise should be free from any tuberculous taint. No nurse or other care-taker should ever be employed about children who has, or ever has had, pulmonary tuberculosis. It is wise to exclude also those who suffered when children from tuberculosis of the bones or the cervical glands, although the danger from such persons is extremely slight. If active tuberculosis exists in any member of the family, a young child should be kept away from the room, and if possible should not reside in the house. On no account should infected persons be allowed to kiss children or sleep in the same bed with them. The danger from drinking-cups and other dishes should not be forgotten. A tuberculous person should either have his special dishes, or the utmost care should be taken to boil all those which he has used. Cows whose milk is used for children should be under regular veterinary inspection and should have passed the tuberculin test. In any case where the slightest doubt regarding the health of the cows exists, or where the source of the milk is unknown, the milk should be heated to a temperature of 155° F. for thirty minutes. The danger of infection through the alimentary canal is very much less than through the respiratory tract, and consequently the precautions first mentioned are much more important than those relating to the food, although the latter should on no account be neglected.

In the case of delicate children and those of tuberculous parents or with other tuberculous relatives, everything possible should be done to fortify them against the disease. They should be kept under more or less constant medical supervision as regards their clothing, manner of life, etc., and should take cod-liver oil every winter. Every attack of bronchitis or broncho-pneumonia should be watched with the greatest solicitude. Exposure to measles or pertussis should especially be avoided. The country rather than the city should be chosen for residence, and the child should spend the winter and spring in some warm, dry climate, such as that of southern California, the interior of South Carolina or Georgia, or Lakewood, N. J. Parents should be distinctly taught that watchfulness and care do not mean coddling or the keeping of children in the house the greater part of the time. Such children should live as much as possible in the open air, and every form of sport encouraged which tends to keep them there. Overheated houses are one of the most prolific agencies in perpetuating a delicate condition of health. Plenty of fresh air in sleeping apartments should always be insisted upon. All catarrhal troubles of the nose and pharynx should receive early and prompt attention, especially should hypertrophied tonsils and adenoid growths of the pharynx be removed, since these are conditions which form a most favourable nidus for the growth of tubercle bacilli.

Treatment of General and Pulmonary Tuberculosis.—If fresh air and a proper climate are necessary for the cure of this disease in adults, they are tenfold more necessary in the case of children. Without them there is little hope for a child with active pulmonary tuberculosis. Nowhere do these cases do so badly as in a hospital located in a city, and no class of hospital cases do worse than these. The same regions that are beneficial for adult cases usually agree with children, with the exception that the latter, as a rule, do better in a warm than in a cold climate. Plenty of fresh air and sunshine are essential. A child must be where he can be kept in the open air for at least several hours each day, in spite of fever, cough, or other acute symptoms.

For the most acute cases where the children are confined to the bed, the largest, best-ventilated, and sunniest room available should be secured, and a window should be open the greater part of the time. The general management of such cases is the same as for those with acute pneumonia.

No specific remedy for tuberculosis has as yet stood the test of experience. The diet is a matter of the utmost importance. Tuberculous patients must be fed like most other sick children, care being taken not to disturb the digestion by the unnecessary use of drugs. For a staple article of diet, milk is the best, and where this is not well borne some of its substitutes—kumyss, matzoon, etc.—may be tried. Cream is almost as useful as cod-liver oil, and should be given in one form or another whenever the child can take it.

The two drugs which are most useful are creosote and cod-liver oil. Creosote may be given both by the stomach and by inhalation, as in cases of pneumonia. By the stomach there may be used for older children, the shellac-coated pills containing one or two drops of creosote; for those who are younger, it may be given in combination with the liquid peptonoids or in an emulsion with cod-liver oil. Cod-liver oil is usually best given in a fresh emulsion, although some children bear the pure oil better than any other preparation. Inunctions of this or other oils are of some value when it is not well tolerated by the stomach. Arsenic, iron, and the compound syrup of the hypophosphites are all useful as general tonics, but as specifics their action is very questionable.

When symptoms pointing to tuberculosis of the bronchial glands are present, the syrup of the iodide of iron should be used in the same way as in disease of the cervical glands. When they ulcerate into the trachea or larger bronchi, they generally cause death, no matter what is done.

CHAPTER XI.

SYPHILIS.

SYPHILIS is a communicable disease due to a specific poison. Of the various micro-organisms which have been associated with this disease the only one which deserves mention is the *spirochæta pallida* of Schaudinn. Although but recently published, many confirmations of his work have already appeared. The organism is an elongated spirillum. It has been found in the adult in the primary lesion and in inguinal lymph glands; in cases of congenital syphilis, in the cutaneous lesions and in the liver and spleen. It has not yet been cultivated.

In infancy and childhood both the acquired and the hereditary forms of syphilis are seen.

ACQUIRED SYPHILIS.

While acquired syphilis is very much less frequent than the hereditary variety, it is by no means a rare disease in early life. It is not improbable that some of the manifestations of syphilis in later childhood which are usually denominated "late hereditary syphilis," are really due to the acquired form.

Etiology.—An infant may be infected by its mother during parturition; but this is extremely rare and can take place only when there are lesions upon the mother's genitals. Infection is more likely to be from a mother who contracts syphilis subsequently to the birth of the child, and may occur through nursing or accidental contact by kissing, etc. In either of these ways children may be infected by wet-nurses, or from a venereal sore upon the nipple. Whether syphilis can be communicated through the milk when the nipple is perfectly healthy and free from fissures, is somewhat doubtful.

Syphilis may be communicated directly from a syphilitic child to one who is healthy by kissing, sexual contact, or indirectly by means of bottles, spoons, cups, clothing, etc. The latter mode of infection is most likely to occur in institutions. Vaccination was formerly a not infrequent mode of communicating syphilis, but since the general introduction of bovine virus this is very rarely seen. Cases have been recorded by Taylor, Hutchinson, and others where the disease has been conveyed by the rite of circumcision, either from the mouth or the instruments of the operator.

The relative frequency of the different sources of infection is shown by Fournier's statistics of forty cases: The source of infection was the parents in nineteen; nurses, in eight; servants, in four; sexual contact, in four; vaccination, in two; other children, in two; a physician, in one. The ages at which the disease was acquired in this series of cases were as follows: during the first year, nineteen; during the second year, ten; during the third and fourth years, seven; from the fifth to the fourteenth years, six.

Symptoms.—The symptoms of acquired syphilis in children are in all respects similar to the same disease in the adult. A primary sore is present at the site of infection, which is most frequently the lips, the mouth or some part of the face; very rarely is it seen on the genitals. There are very few individual symptoms belonging to hereditary syphilis which may not also be present when the disease is acquired. Its course, however, is very much milder in the latter and a fatal termination is rare. Fournier states that of his forty-two cases only one died of marasmus. This marked contrast to hereditary syphilis is due chiefly to the fact that in the acquired variety the infant is rarely affected during the early months of life, a time when hereditary syphilis is so very fatal.

Tertiary symptoms may appear at any time from three to twenty years after the original infection.

The treatment is the same as in hereditary syphilis.

HEREDITARY SYPHILIS.

Etiology.—A child may inherit syphilis from both parents or from either separately. If both parents are syphilitic, the child is usually but not invariably so. The symptoms, however, are not more severe than when the inheritance is from one parent only. The likelihood of transmission depends upon the stage of the disease in the parents. If both are suffering from secondary symptoms, transmission is almost certain. If active treatment has been employed for several months, if the child is born at a period when no active symptoms are present, or if the symptoms are of a tertiary character, the offspring will probably escape. First-born children are more likely to suffer severely from syphilis than the later ones, provided infection of the parents has taken place prior to the birth of all the children.

Infection from the father.—Syphilis may be inherited from the father alone. In this case the disease is probably communicated directly from the semen to the ovum. It is more likely to be transmitted from the father than from the mother, as the child is frequently syphilitic when the mother has few or no active symptoms. Of twenty cases observed by Meyer in which the father alone was syphilitic, the fœtus was discharged macerated in eleven cases, and nine children were born with congenital syphilis, all but one dying soon after birth. It is possible, though rare, for the father to convey syphilis when he is free from symptoms, or when he is suffering from tertiary symptoms only.

Infection from the mother.—It is certain that syphilis may be transmitted when the mother alone is diseased, as is shown by cases where women who have acquired syphilis while wet-nursing infected children, have subsequently borne syphilitic children, the father remaining healthy. If the mother only is syphilitic the probabilities of transmission to the child appear to be considerably less than if the father alone is affected. If the mother's symptoms are tertiary the child will probably escape.

Both parents healthy at the time of conception and the mother infected during pregnancy.—Under these conditions the child may or may not be syphilitic. Transmission to the child is much less likely to occur if the mother is infected during the last two months of her pregnancy than earlier, although, as Hutchinson's cases conclusively show, there is no certainty that the child will escape. Diday states that if the mother is infected before the fourth week and proper treatment is instituted, the child will usually escape on account of the relation of the embryo to the maternal circulation during this early period.

Can a healthy mother bear a syphilitic child?—In 1837 Colles enunciated the following proposition, the truth of which has been abundantly verified since his time: "A new-born child affected with inherited syphilis, even although it may have symptoms in the mouth, never causes ulceration of the breasts which it sucks if it be the mother who suckles it, although continuing capable of infecting a strange nurse."

Caspary inoculated with syphilis a woman, apparently healthy, who had aborted with a syphilitic child; the result was negative. A similar experiment was made by Neumann, with a like result. Widal reports a case of an apparently healthy woman who had a syphilitic child by an infected husband; later, by a second husband who was free from syphilis, she had a syphilitic child. The conclusion seems irresistible that the carrying of a syphilitic child gives immunity to the mother against the disease and that this immunity is due to the fact that she herself suffers from syphilis, or a modification of that disease. According to Hutchinson, the modified syphilis acquired by a woman under the circumstances mentioned, bears to syphilis acquired from a chancre a somewhat similar relation to that which vaccinia bears to smallpox. The mother under these circumstances can not be inoculated, either by her syphilitic nursing-infant or artificially.

The communicability of hereditary syphilis.—That hereditary syphilis is contagious is conclusively shown by a number of recorded instances in which a healthy wet-nurse has been infected by a syphilitic infant. However, such examples of contagion are very rare, and many writers of large experience state that they have never seen it. It is certainly true that the danger of spreading infection from a case of hereditary syphilis has been exaggerated, and that it differs so much in this respect from the acquired form of the disease that this peculiarity is of some value in differential diagnosis.

Lesions.—Death may be due to syphilis, and yet the autopsy may reveal no characteristic anatomical changes, and in fact there may be no demonstrable changes in any of the organs.

Bones.—In the case of a syphilitic fœtus, a stillborn child, or one dying soon after birth, the changes in the bones are more uniformly present than are any other lesions. They are in fact rarely wanting, and it is by them usually that syphilis is recognised post mortem. The long bones are principally affected, the most important changes being found at the junction of the shaft with the epiphyseal cartilage. The lesion is termed an epiphyseal osteo-chondritis or acute epiphysitis. There is in the early stage congestion, swelling, and cell proliferation, which may be followed by separation of the epiphysis, suppuration in the neighbouring joint, osteomyelitis, and necrosis. These changes are more fully considered under Diseases of the Bones.

Liver.—This is probably more frequently involved in the fœtus and newly-born infant than any other organ. The syphilitic lesions of the liver have been studied very fully by Hudelo. He describes as present in the youngest infants an interstitial hepatitis, a gummatous hepatitis, and a combination of the two varieties.

In the interstitial form, which is most frequent in infancy, there is first a congestion and swelling of the organ, with the exudation of leucocytes in groups. The liver is enlarged, frequently very much so, but presents few other gross changes. Later there is increased exudation between the liver cells, new connective tissue forms, and atrophy of the liver cells takes place, with obliteration of some of the portal and hepatic vessels. This process may be diffuse, but it is usually in patches. Groups of miliary syphilomata may also be found. If the process is diffuse, the liver is large, firm, and of a grayish-yellow colour. If it is localized, the affected areas are yellow or gray and the other parts are normal.

The gummatous form is not frequent in early infancy, but belongs to a little later period. In this there may be miliary syphilomata with interstitial changes, and in addition the formation of small or large gummatous tumours, which may be softened at the centre. They are surrounded by zones of new connective tissue and the liver cells are atrophied. Amyloid changes may be present.

In the late form of hereditary syphilis, usually seen in children over four or five years old, the liver is rarely affected. Hudelo was able to collect but forty-seven such cases. The lesions resemble those of the congenital variety. There are found cirrhotic changes, which may be diffuse or circumscribed, and gummatous deposits, which vary from a minute size to that of a cherry; there may be amyloid degeneration.

Spleen.—This is almost invariably enlarged in newly-born children with syphilis and in syphilitic fœtuses, but nothing characteristic is found under the microscope (Birch-Hirschfeld). In older children the enlargement of the spleen is apt to be greater than at birth; the organ may be the seat of interstitial changes, and sometimes there may be gummatous deposits. These changes are rare in children under two years of age.

Respiratory system.—In syphilitic infants which are stillborn and in those which die soon after birth, there is frequently found in the lungs what is known as "white pneumonia." This process consists, according to Hillier, in fatty changes in the epithelium of the air vesicles; with this there is associated a certain amount of interstitial pneumonia, which is chiefly peri-bronchial. In older cases the interstitial pneumonia is extensive, and the lungs may be the seat of gummatous deposits, which soften and form small cavities. Accompanying these changes there may be bronchiectasis, emphysema, and the usual secondary lesions which follow chronic interstitial pneumonia. In syphilitic infants there is a strong tendency for all inflammations of the lungs to become chronic.

The trachea and bronchi are in rare cases the seat of stenosis, which results from cicatrization following the softening of gummatous deposits in their walls. Lesions of the larynx (page 507) are also infrequent. There is usually perichondritis, which more often involves the epiglottis than any other part, and sometimes there is the formation of papillomatous masses; but ulceration and stenosis are both rare.

The nasal mucous membrane in the early stage of the disease is very constantly the seat of a chronic catarrhal inflammation, which may be accompanied by superficial ulceration. In the late cases there is deeper ulceration, from the breaking down of gummata, with extension to the periosteum, cartilages, and bones, causing perforation of the septum, necrosis of the bones, etc.

Nervous system.—Syphilitic lesions of the brain and cord are rare in children as compared with adults, and they are especially so in infancy. The most characteristic cerebral lesion of the newly-born child is hydrocephalus, which may depend upon ependymitis, as in two cases reported by D'Astros, the disease proving fatal in the second month. Syphilitic meningitis is exceedingly rare under two years. There is occasionally seen in young infants a chronic basilar meningitis of syphilitic origin. Chronic pachymeningitis associated with gummata has been observed as early as the fourth year. Money (London) has reported a case with symptoms beginning at eleven months, in which there was chronic meningitis with great thickening of the dura mater and cerebral sclerosis. A few other cases of a similar nature have been recorded.

Nearly all the syphilitic lesions of the nervous system which are seen in adult life have been observed in childhood, but infrequently, and in young children they are extremely rare, although Barlow's patient with multiple gummata at the base was only fifteen months old.

Heart and arteries.—These may be affected even in young infants. Adler (New York), of four cases examined, found two in which well-marked lesions were present in infants under four months. There was endarteritis of the coronary arteries accompanied by the early changes belonging to interstitial myocarditis. Chiari has reported syphilitic endarteritis of the brain at fifteen months, followed by thrombosis and softening.

Digestive system.—Chronic catarrhal pharyngitis is almost a constant symptom of the early cases. Later there is seen superficial or deep ulceration of the pharynx, tonsils, or fauces, which may lead to perforation of the soft palate or to the formation of condylomata.

There are no important lesions of the stomach or intestines either with early or late syphilis. The rectum is occasionally the seat of ulceration, and condylomata may form even in young children.

Organs of special sense.—Otitis is a frequent accompaniment of the early syphilitic pharyngitis. It is very likely to become chronic, and in many cases results in a permanent impairment of hearing. Iritis is relatively rare in children, but it may occur even in intra-uterine life, as shown by the presence of adhesions in newly-born children. It is usually seen in infants four or five months old, and is always serious. Interstitial keratitis occurs frequently as a late manifestation of syphilis. Choroiditis and optic neuritis are both occasionally seen, but they are rare.

Genito-urinary organs.—Nearly all these may be affected, but generally in the late period of the disease. There may be chronic interstitial nephritis and more rarely gummatous deposits in the kidney, interstitial changes in the suprarenal bodies, and orchitis, which usually affects the body of the organ, rarely the epididymis; it is generally an interstitial inflammation, with or without gummatous deposits.

Among the less frequent visceral lesions may be mentioned, abscesses of the thymus, which are usually small and multiple; enlargement of the pancreas, with an increase of connective tissue and glandular atrophy; and chronic peritonitis. The lesions of the mucous membranes will be considered under Symptoms.

Symptoms.—As the result of syphilis, abortion may take place at any period of pregnancy, with the discharge of a dead or macerated fœtus, or the child may be stillborn at term, or it may be born alive prematurely, but with so feeble a vitality that it survives but a few hours. Under these circumstances it is often difficult and sometimes impossible to decide positively with reference to the existence of syphilis. Maceration of the fœtus or peeling of the skin is no proof, and even the examination of the internal organs may not be conclusive. Lomer examined 43 fœtuses, all dying before the thirtieth week of pregnancy; he found the spleen and liver enlarged in all, and marked bone changes in 21. Birch-Hirschfeld examined 108 newly-born syphilitic infants; he found the spleen invariably enlarged; typical bone changes were present in 35, but in many cases the bones were normal. Mervis, from an examination of 92 syphilitic fœtuses, states that no eruption upon the skin was found earlier than the eighth month.

Symptoms are present at birth in only a small number of cases. In such there is usually a very severe degree of infection, and the infants do not often live more than a few days. Upon the skin there may be seen an eruption of pustules, papules, or bullæ. The bullæ are usually upon the soles and palms, but may be found upon other parts of the body. The name "syphilitic pemphigus" is often given to this condition. Pemphigus in the newly born, however, is not invariably due to syphilis, but may be present in other conditions of low vitality. The bullæ are at first small, and then coalesce and form larger ones two inches or more in diameter. They contain a turbid serum which is sometimes tinged with blood, and sometimes yellow from pus. Pustules, when present, are usually seen upon the face or scalp. The general appearance of these infants is wretched in the extreme. The body is wasted, the skin wrinkled, and temperature subnormal. The spleen is usually enlarged and often the liver also. They suck feebly or not at all, and usually die from inanition within two weeks.

In the great majority of cases the infant appears healthy at birth, and continues so for a variable time before the manifestation of the characteristic symptoms of syphilis. As a rule, the more intense the infection, the earlier the symptoms make their appearance. The earliest symptoms are generally seen between the second and the sixth weeks. If three months pass without evidence of syphilis, the child may be considered safe, the exceptions to this rule being very few. Miller (Moscow) gives the following statistics of the time of beginning of symptoms in 1,000 cases:

Symptoms appeared during the first week	85	cases.
" " " " second week	138	"
" " " " third week	240	"
" " " " fourth week	177	"
" " " " fifth week	86	"
" " " " sixth week	54	"
" " " " seventh week	50	"
" " " " eighth week	30	"
After the eighth week	140	"

Sometimes the constitutional symptoms—wasting, cachexia, etc.—are noticed before the local ones, but usually this is not the case. Generally the first symptom is the coryza or " snuffles," which resembles an ordinary cold in the head, except that it persists. It is accompanied by a hoarse cry, indicating that the larynx participates in the catarrhal inflammation. Soon the eruption makes its appearance, being generally first seen upon the hands, feet, and face. Fissures and mucous patches may be seen upon the lips, about the anus, etc. There is often slight fever, from 99° to 101° F. There may also be observed excessive tenderness and swelling about the shoulders, elbows, wrists, or ankles, due to acute epiphysitis, which may cause the child to cry from the slightest amount of handling, and the limbs may be moved so little that paralysis is suspected.

In a severe case, as these local symptoms develop, the infant's general nutrition suffers. It loses steadily in weight; it becomes extremely anæmic; it whines and frets almost continually, but especially at night. The features have a pitiful, drawn expression; and the face is wrinkled, giving the infant a very old appearance. The skin has a peculiar sallow colour, which has been well described as *café au lait*. The symptoms may continue until a condition of extreme marasmus is reached, or death occurs from some intercurrent affection of the lungs or digestive organs.

In the milder forms of infection the severe constitutional symptoms described are not seen, although the local evidences of disease are well marked. The severity of the symptoms is also much modified by treatment, especially when this is begun early.

The most important local symptoms are the coryza, eruption, fissures about the mouth and anus, mucous patches, painful swellings at the extremities of the long bones, pseudo-paralysis, and onychia.

Coryza.—In most of the cases this is the first symptom. Beginning like an ordinary catarrh, it is distinguished by its severity and its persistence. There is a copious discharge of mucus and serum, often tinged with blood. Thick crusts form, which produce the usual symptoms of nasal obstruction; there is great difficulty in nursing; the infant breathes through the mouth, and the mucous membrane of the mouth is dry, causing great discomfort. If untreated, the process, which at first involves the mucous membrane only, may extend to the submucous tissue, causing ulceration; but the cartilages and the bones of the nasal fossæ are not involved till a later period in the disease.

The nasal catarrh is associated with more or less laryngitis, causing hoarseness or aphonia, and rarely there may be laryngeal stenosis. Dillon Brown has reported one case in an infant six weeks old, which recovered after intubation.

Eruption.—This usually occurs after the coryza has lasted about a week; but the two may come at the same time; or the coryza may be absent or so slight that the rash appears to be the first symptom.

Occasionally there is seen a diffuse blush or roseola, but more frequently the eruption is macular, occurring in small, dark-red spots about the size of the infant's finger nails, usually circular and often slightly elevated; there is no surrounding inflammation, and rarely any itching. It is usually most abundant upon the face, the neck, and the extensor surface of the upper and lower extremities, especially the hands and feet, sometimes extending over the entire body, although it is generally scanty over the chest and abdomen. At first the colour is bright, but gradually becomes of a dusky-red or coppery hue. After a little time very fine scales may be seen upon the surface of the red macules. The rash comes out slowly, usually requiring from one to three weeks for its full development. It fades gradually, leaving a coppery discoloration of the skin, which continues for a long time. The duration of the eruption is from three to eight weeks; less if active treatment is employed.

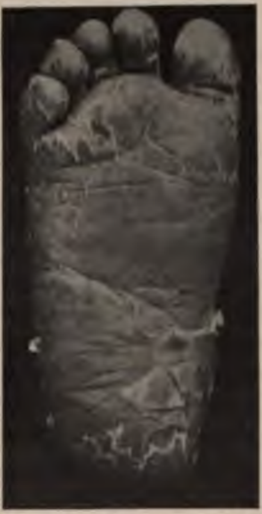


Fig. 214.—Syphilitic scaling of the foot. From an infant eight weeks old.

A papular eruption is rarely seen alone, but is usually associated with the macular variety. The papules are of a brownish colour and are hard. They are seen most frequently upon the palms and soles.

A squamous eruption is frequently seen upon the palms and soles, but very rarely elsewhere. In a few cases this scaliness forms the most distinctive feature of the cutaneous lesion (see Fig. 214).

Fissures and mucous patches.—These are among the most diagnostic features of early hereditary syphilis. Fissures are most frequently seen on the lips and about the anus, but they may occur about the nostrils and occasionally elsewhere. The fissures of the lips are really linear ulcers, and are distinguished by their persistence in spite of local treatment.

They are multiple, deep, painful, and bleed easily. Those at the angle of the mouth are especially troublesome.

Mucous patches may develop from fissures, but more frequently from papules which are situated in regions where they are exposed to constant moisture and friction. They are very common upon the muco-cutaneous surfaces and wherever the skin is especially thin. They are most apt to be seen about the lips, anus, scrotum, and vulva, but they may also be found behind the ears, between the toes, in the folds of the groin, axillæ, or buttocks. They vary from an eighth to half an inch in diameter, are whitish in colour, and are raised rather than excavated.

Ulcers may be present upon any of the mucous membranes, frequently in the mouth or on the genitals; they are seldom symmetrical, and while they may be broad they are never deep.

Hæmorrhages.—They are generally associated with the lesions of the mucous membranes, especially of the nose. In young infants with severe infection, bleeding may occur from the bullous eruption upon the skin, or from the fissures at any of the orifices, particularly the mouth and anus. Fischl has reported seven cases of multiple hæmorrhages in the newly born, associated with other symptoms of congenital syphilis. Mracek noted hæmorrhages in thirty-three per cent of 160 autopsies on syphilitic stillborn infants or those dying soon after birth. Examination of the blood-vessels in some of these cases showed infiltration of their walls and narrowing of their lumen. The vascular changes were thought to be the cause of the bleeding.

Nails.—The nails present several peculiarities in syphilitic infants. There may be a disease of the matrix resulting in suppuration and exfoliation of the nail; frequently the dorsum is much arched, and the nail appears as if it had been pinched by a pair of forceps—i. e., claw-shaped; this is an early symptom of some diagnostic importance. The hair and eyebrows frequently fall out completely. This symptom is not usually present in very early infancy.

Pseudo-paralysis.—This is due to acute epiphysitis, and it may be the first symptom of hereditary syphilis to attract attention. It is usually noticed when the infant is a few weeks old that one or sometimes both arms are not moved, and that the parts are tender when handled. The arm is very frequently held in marked inward rotation with the palm looking outward, resembling the position in Erb's palsy; but careful examination makes it evident that the loss of power is only apparent, and that it is due either to the pain which motion produces or to epiphyseal separation. A history will usually be obtained that loss of power did not exist at birth, but developed subsequently. The electrical reactions in these cases are normal, and the rapid improvement under mercurial treatment is diagnostic.

The only visceral symptoms of importance relate to the spleen, which is almost invariably much enlarged in the active stage of hereditary syphilis.

Late Hereditary Syphilis.—These symptoms may come on at any period during childhood or about the time of puberty, but very rarely at a later time than this. They are seen both in those who have had the usual symptoms of hereditary syphilis in early infancy, and in others where the most careful examination into the history fails to disclose any symptoms whatever of early syphilis. It is fair to assume in such cases either that early symptoms were absent or that they were of trivial importance. It is still a matter of dispute whether these late symptoms should be regarded as hereditary, tertiary syphilis, which has not previously given signs, or as the late stage of ordinary syphilis in which the early symptoms have been overlooked. It is certain that the symptoms are quite as apt to be severe when there is no history of early syphilis as when this has been typical. It is quite possible that some of these may be the late manifestations of the acquired syphilis not recognised in the early stage.

Late hereditary syphilis shows itself by symptoms which in acquired disease would be classed as tertiary. The most characteristic are the affections of the teeth, the bones, gummatous deposits in the solid viscera, the skin, or mucous membranes, the breaking down of which may lead to ulceration.

Teeth.—There are no peculiarities in the first teeth of syphilitic children except their proneness to early decay. They are rather more likely to appear early than late.

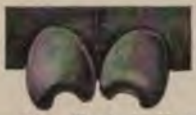


FIG. 215.—Typical "Hutchinson's teeth." (After Fournier.)

The characteristic teeth of syphilis are those of the second set. In estimating the diagnostic value of these changes, only the upper central incisors are to be relied upon; these are the test teeth. Although changes are frequently seen in other teeth, they are not always diagnostic. Typical syphilitic teeth, according to Hutchinson, have each a single notch in the centre of the edge (Fig. 215). The notch is usually shallow and more or less crescentic in shape. The enamel is generally deficient in the centre of the notch, and the tooth here is apt to be discoloured. The teeth in other cases are variously dwarfed and deformed. (See Fig. 216.) They often taper regularly from the base to the edge, giving rise to the term "screw-driver teeth." The teeth are not so flat as the normal incisors, but often rounded and peg-like. They are not properly placed, but incline either toward or away from each other. They are seldom large enough to touch the adjacent teeth on both sides.

Although Hutchinson's teeth may generally be taken as conclusive evidence of syphilis, they are not invariably so, as Keyes and others have shown. It is to be remembered in this connection that the absence of changes in the teeth is of no importance whatever as evidence that syphilis is not present. Hutchinson states that they are wanting in more than half the cases.

Bones.—The form of disease which is usually seen at this period is an osteo-periostitis, affecting principally the shaft of the long bones and the cranium. It has already been described elsewhere.

Lymph nodes.—They are much less frequently affected than in adults, and in early infancy they are seldom involved. In most cases after the first year there may be found a moderate degree of enlargement of the post-cervical and epitrochlear glands, swelling of the latter having considerable diagnostic value. They are situated just above the internal condyle of the humerus, and under normal conditions can scarcely be felt. In syphilitic children they may be as large as a pea or a small bean; sometimes two or three of them can be distinguished. They are so rarely enlarged from other constitutional conditions that, provided no local cause for the swelling exists, they should always create a suspicion of syphilis. The post-cervical glands are frequently affected, but are not so diagnostic. The degree of enlargement is rarely great. Occasionally there are seen in the neck large masses of swollen lymph glands which resemble tuberculous swellings. They are, however, very rare.

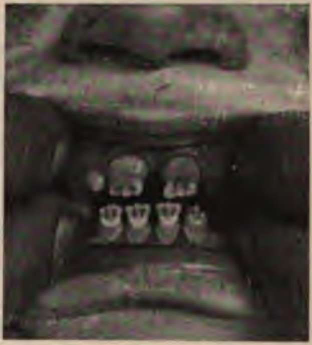


FIG. 216.—Syphilitic teeth; boy eight years old; under observation several years with various syphilitic manifestations.

Special senses.—The most frequent affection of the eye in late syphilis is interstitial keratitis, the close connection of which with hereditary syphilis was first pointed out by Hutchinson. It is usually found associated with the typical notched teeth. The diagnostic value of keratitis in syphilis is denied by Fournier, who states that, while often syphilitic, it is not infrequently due simply to malnutrition. Both eyes are usually affected, and in all degrees of severity, from a slight haziness of the cornea to complete opacity. However, with an early diagnosis and prompt treatment, recovery may be expected in most cases.

Chronic otitis may be a result of the acute process seen in early infancy. There is nothing peculiar about the inflammation in these cases. A form of deafness occurs in older children, which Hutchinson states is almost invariably due to syphilis. Its onset is quite sudden, without pain and frequently without discharge. The loss of hearing is apt to be permanent, and if it occurs early in childhood it is a cause of deaf-mutism.

Skin.—The most important of the later manifestations of syphilis consists in the formation of subcutaneous gummata. In the early stage they are indurated, elastic, of a grayish colour, with red borders. Under treatment they disappear quite rapidly by absorption; but when neglected they break down, leaving large deep ulcers. These ulcers are quite characteristic in appearance, but may be confounded with those due to tuberculosis. The syphilitic ulcer has rounded, thickened, indurated borders, and a base which is depressed and has the appearance of being scooped out. It is sometimes covered by hard crusts and is surrounded by a red areola. It leaves a smooth white scar. The most frequent situation is upon the face and upper part of the legs or thighs. Tuberculous ulcers have usually soft, flat edges, and do not extend so deeply; they are more irregular in outline; the cicatrix left is of a purplish colour, which becomes red and slowly fades. Tubercle bacilli may be found. Sometimes it is only by the effect of treatment that the diagnosis can be made between these two lesions.

Nose and palate.—Disease of these parts generally begins as the breaking down of gummatous deposits in the mucous membrane. The nose may in consequence be the seat of a protracted fetid discharge (ozæna). The disease may take on a destructive form of ulceration which is at times phagedenic, and may cause rapid destruction of the nasal cartilages and bones, perforation of the septum, and occasionally of the floor of the nasal fossæ. There may be necrosis of the turbinated bones, the vomer, or the ethmoid. In the most severe forms the nose may be almost destroyed in the course of a few weeks. There may be at the same time deep ulceration of the soft palate, leading to perforation. In a young person this is almost invariably due to syphilis. In many particulars these ulcerations of the nose and palate resemble lupus; they are distinguished by the rapidity of their progress, syphilis often doing as much damage in weeks as is done by lupus in years (Hutchinson).

Other symptoms.—Syphilitic disease of the larynx and bronchi is rare in childhood. The former (page 507) may give rise to hoarseness or aphonia and occasionally to stenosis; the latter to a chronic cough and asthmatic attacks. There are no characteristic symptoms belonging to syphilis of the lungs. The different lesions of the central nervous system which may be due to syphilis are all quite rare. The forms have already been mentioned, and their symptomatology is discussed in Diseases of the Nervous System.

The only visceral changes which aid much in diagnosis are those of the liver and spleen. The liver is often enlarged, sometimes to a marked degree, and occasionally there is ascites, but very seldom jaundice.

Enlargement of the spleen is a very frequent symptom—in fact, it is almost constant during active syphilitic disease. I have several times seen it so swollen as to form an abdominal tumour of considerable size. In one case, in a boy three years old, the spleen extended five inches below the free border of the ribs, quite to the crest of the ileum. It was associated with moderate enlargement of the liver, as is usually the case.

In addition to the local symptoms of late hereditary syphilis enumerated, there are others of a general character which are quite as important. The body is usually undersized; the constitution is delicate, and shows but little resistance to all forms of disease; puberty is frequently delayed, and the development of the breasts and the genital organs often imperfect; anæmia is usually present, and the skin has a sallow appearance. Mentally, many of these children are somewhat deficient, and in a few instances they become idiotic, epileptic, or the subjects of dementia.

Diagnosis.—The diagnosis of early syphilis in most cases is not difficult. The coryza, eruption, labial fissures, mucous patches about the anus and genitals, enlarged spleen, and general cachexia—all form a picture which it is difficult to mistake. In irregular cases the diagnosis is easy just in proportion to the number of the foregoing symptoms which are present. Special care should be taken not to confound the moist papules of simple intertrigo upon the buttocks or thighs with those of syphilis.

In late syphilis the following symptoms are the most reliable for diagnosis: notching of the teeth, falling in of the bridge of the nose, interstitial keratitis, deafness not traceable to ordinary otitis, enlargement of the spleen and epitrochlear glands, ulceration of the palate or nose, the sabre-like deformity of the tibia, and nodes upon the tibia or cranium.

It becomes at times important to distinguish hereditary from acquired syphilis. While this is not always possible, it is often so. Visceral lesions in acquired syphilis are not common and belong to the late period of the disease; in the hereditary form they are well-nigh constant and occur early, often being present at birth. The acute epiphysitis, sometimes accompanied by pseudo-paralysis, seldom if ever occurs in acquired syphilis, though frequent in the hereditary form. Symptoms due to defects in development, like the misshapen finger-nails, are seen only in hereditary syphilis. The early symptoms of the mucous membranes and muco-cutaneous surfaces—coryza, hoarseness, hæmorrhages, labial fissures, etc.—so characteristic of hereditary syphilis, have no place in the acquired form, while the single primary lesion sometimes found in the acquired form does not exist in the hereditary disease. Finally, hereditary syphilis is very slightly, whereas the acquired form is highly contagious.

Prognosis.—Generally speaking, the prognosis is much worse in infantile syphilis than in that of adults. In infancy it is much worse when hereditary than when acquired, for the reason that often the child who is the subject of hereditary syphilis has been affected by the poison from the very beginning of its existence, and this has modified its entire development.

The results of 206 syphilitic pregnancies observed by Jullien (Paris) were as follows: abortion occurred in 36, stillbirths in 8, and 69 children died soon after birth, making a total mortality of 55 per cent; 50 were living and syphilitic; only 43 living and in good health. Still worse were the results in cases observed by Le Pileur: of 154 pregnancies in syphilitic women, there were 120 abortions or stillbirths, 26 children died soon after birth, and only 8 survived. The statistics of the Foundling Asylum in Moscow for ten years showed that of 2,038 syphilitic infants the mortality was over 70 per cent.

Such a mortality as that indicated in the above statistics is seen only in institutions where little or no previous treatment has been employed. In private practice certainly nothing approaching it occurs.

In addition to those who die early as the result of syphilitic infection, there must be added many whose constitutions are so impaired by syphilis that they fall an easy prey in infancy to pneumonia, diarrhœa or other forms of acute disease. The remote effects of syphilis in infancy it is hard to estimate; it exerts a modifying influence upon the constitution in childhood and even throughout the life of the individual.

The prognosis in an individual case depends upon the age at which the symptoms develop, the time when treatment is begun, upon its thoroughness, and upon the surroundings and mode of nourishment of the child. The outlook is better the longer after birth the first symptoms appear; it is also better in infants who are nursed than in those who are artificially fed.

As compared with syphilis of the adult, relapses are rare, and when they occur early they are nearly always the result of insufficient treatment. If proper early treatment is carried out, the severe late symptoms are rare; patients are usually free from all symptoms until six or seven years old, or until near the time of puberty—two periods when they are likely to develop.

The prognosis is better in the later children of syphilitic parents than in the earlier ones, provided infection has preceded the birth of all the children. This fact illustrates the general tendency of the syphilitic poison to diminish in virulence as time passes, even without treatment. The following instance cited by Bertin well illustrates this point:

In the first pregnancy, the mother aborted with a dead child at the sixth month; in the second, at the seventh month; in the third, at seven and a half months; in the fourth the child was born at term, and lived eighteen days; in the fifth it lived six weeks; in the sixth the child lived four months, without treatment.

Prophylaxis.—No infected person should be allowed to marry until at least two years have passed after the initial sore, steady treatment being continued meanwhile; nor if there are any active symptoms, no matter how long a time has elapsed since infection. There is no certainty in any case that the child will escape.

The mother should be treated during her pregnancy: (1) if she is syphilitic, whether the disease was acquired at the time of conception or subsequently; (2) if the father is known to be suffering from syphilis, whether the mother has symptoms or not; (3) if the mother has previously shown signs of syphilis, but has had no active symptoms for a considerable period. In all these conditions if efficient treatment is carried on throughout pregnancy there is a strong probability, but in no case a certainty, that the child will escape. The third condition mentioned is the one in which treatment is most likely to be neglected, especially if the mother has previously borne a child who was not syphilitic. Syphilis, however, shows a strong tendency to reappear and become active during pregnancy, even though it has been long quiescent, as the following case cited by Diday shows:

A woman who had lost seven children from syphilis was put under treatment during the eighth pregnancy; result—child born healthy, and continued so. In the ninth pregnancy treatment was continued with a like result; in the tenth pregnancy, no treatment, child syphilitic, dying when six months old; in the eleventh pregnancy, treatment repeated, child healthy.

The danger of infection during labour is slight. If there are upon the genitals of the mother either a chancre or syphilitic ulcers, they should be thoroughly cauterized before labour.

As the greatest danger of infecting a child after birth is from its parents or a wet-nurse, syphilitic parents should be duly warned of the danger to their children, and especially should be cautioned against kissing them or sleeping in the same bed with them. The utmost care should be exercised to prevent a healthy child from being infected by a syphilitic nurse. A nurse should never be accepted without a thorough examination, no matter how clear a history may be given. As a syphilitic child in the household may be the means of infecting other children, the same precautions should be taken as in the case of other contagious diseases. The chief danger to other children comes from kissing or from using bottles, spoons, or cups which have been infected; as the syphilitic infant is chiefly dangerous on account of the lesions in the mouth. Trouble most frequently occurs because of ignorance regarding the nature of the disease. It is possible for a syphilitic child to nurse a healthy woman without communicating syphilis, if the child's mouth is treated and the nipple not allowed to become fissured; but it is an experiment which should never be tried.

Treatment.—This should always be begun as soon as the first positive symptoms of syphilis appear. Under certain circumstances it may be advisable not to wait for symptoms; as, for example, where both parents have recently suffered from active symptoms, where previous children have died soon after birth, or where, with marked symptoms in the parents, the child exhibits the cachexia of syphilis, but no definite symptoms. Such anticipatory treatment need not be continued l than six weeks unless symptoms appear.

The indirect treatment, designed to reach the child througl mother's milk, has fallen into deserved disuse, as it is very uncertai altogether unsatisfactory.

Mercury is as much a specific for hereditary as for acquired sy There are many ways of introducing it into the system : it may be by inunctions, by the mouth, by fumigations, by baths, or hypodermi In most cases inunction is the manner to be preferred in young in Gr.x of mercurial ointment, diluted with the same amount of vaseline be rubbed daily into the palms, soles, axillæ, or the inner surface o thighs. It is advisable to change the place of inunction from day to and if this is done, it is extremely rare that erythema is produced. any reason inunctions are objectionable, as they may be where the f are to be kept in ignorance of the treatment, either the gray powder o bichloride may be given by the mouth. The usual dose of the gray po should be gr.j four times a day; that of the bichloride gr. $\frac{1}{60}$ four tin day, always well diluted. It is rare that larger doses are advisable. W the symptoms are urgent, it is often best to substitute calomel for a weeks, as the system can usually be brought more rapidly under the i ence of mercury by this than by the other preparations mentioned; g four times a day is the usual dose required. Other methods of admini tion and other preparations offer no advantages, and have some very vious disadvantages.

The iodide of potassium is to be used, either alone or in combina with mercury, whenever such lesions exist as are classed among adul tertiary. This includes all the late manifestations, and the earlier whenever the bones or viscera are affected. The iodide is usually borne by children, and may be given in almost any desired dosage. infancy it is rare that more than twenty grains daily are required, in older children the necessary amount may be from one to two dracl daily. It should always be given largely diluted.

The duration of mercurial treatment should be at least one year. doses during the last six months may be reduced to one half or one tl those employed while active symptoms are present. Treatment shoulc longer than a year if symptoms exist. It is often better not to give mercury continuously, but with short periods of intermission.

The tonic treatment of syphilis is important and should not be r lected. After specific treatment has been carried on for a time, parti larly if rapidly pushed, the child often becomes anæmic, and suffers gre from general malnutrition. Under such circumstances also it is o wise to discontinue mercury altogether for a time, or at least to red the dose very much, and administer cod-liver oil, iron, wine, and ot tonics. Such a change is frequently found to act most beneficially, even when lesions are present, which perhaps have been very little or not at all affected by the specific remedies employed. A judicious combination of specific and tonic treatment is required in every case, whether the remedies are given simultaneously or alternately.

Local treatment.—Ulcerative lesions of the skin require cleanliness, dusting with calomel or iodoform, or bathing with the black wash. Mucous patches should be dusted with equal parts of calomel and bismuth. Fissures and ulcers of the mucous membranes should be treated by nitrate of silver. Phagedenic ulcers of the palate or nose should be cauterized with nitric acid or the acid nitrate of mercury. The late syphilitic ulcers of the skin, due to the breaking down of gummata, should be treated with iodoform.

CHAPTER XII.

INFLUENZA.

Synonym: La grippe.

INFLUENZA is an infectious, communicable disease, which is now generally admitted to be due to the bacillus described by Pfeiffer in 1892. It is serious in children chiefly from its tendency to complications of the respiratory tract, in which respect it closely resembles measles.

Etiology.—The influenza bacillus is found chiefly in the sputum and nasal discharge; it is also present in the lower air-passages, and has occasionally been found in the exudation of otitis, empyema, and meningitis accompanying the disease, but rarely in the blood. It is not easily detected in the sputum, repeated examinations often being necessary; but in typical attacks if carefully sought it is found with great uniformity. In acute cases it may disappear very early; in protracted cases its presence is sometimes detected for weeks or even months. Besides the bacillus of Pfeiffer, there are frequently found, either associated or separately, in the organs of patients dying from influenza, the streptococcus and the diplococcus pneumoniæ, for the development of which influenza creates conditions in the highest degree favourable.

Influenza is highly contagious; the poison may be carried by clothing or fomites and clings for some time to infected apartments. The disease prevails epidemically, and after epidemics it may be endemic for a number of years. In New York the disease has probably been present for many years, although it attracted little attention under the name of influenza until the great epidemic of 1891. Epidemics prevail chiefly in winter and spring. All ages are liable to the disease, infants under one year least so, and in some epidemics they may escape altogether. The disease has, however, been observed in infants only a few days old, where the mother was suffering from it at the time of delivery. The children most frequently affected are those from two to ten years of age.

The period of incubation is uncertain. It is usually short, being generally believed to be from one to seven days. Little if any immunity seems to be afforded by one attack; recurrences and second attacks are not uncommon in the same epidemic, and a patient who has once had influenza seems to be more susceptible to the disease in consequence.

Lesions.—There are no characteristic lesions of influenza; those which are most frequently found are due to catarrhal inflammation of the respiratory or the digestive tract. In some cases only the upper respiratory tract is involved, in which case the disease often spreads to the middle ear; in others, only the lower respiratory tract, this in infancy usually spreading rapidly to the lungs, and resulting in bronchopneumonia. Inflammation of the stomach and intestines is much less frequent and, as a rule, less severe. This will be considered more fully under Complications.

Symptoms.—The symptoms of influenza are due to the systemic effects of a general poison, and to certain local congestions and inflammations which are regarded as complications. The two classes of symptoms—the general and the local ones—are found in all possible combinations.

1. *The mild, uncomplicated variety.*—This lasts from two to five days, occasionally a week. The onset is usually abrupt, with chilliness, muscular pains, and sometimes vomiting. The temperature ranges from 101° to 103° F. Even though the fever is not high, the prostration is considerable, and children are often ill enough to remain in bed for several days. The usual general symptoms which accompany fever are present. After the fever has subsided, the child is left weak and anæmic; convalescence is frequently protracted, and it may be three or four weeks before the general health is regained. This is the most common variety seen, the essential symptoms being fever and prostration without evidences of local inflammation. Often there is in addition a mild coryza at the outset and a slight but persistent cough.

2. *Uncomplicated cases of the severe type.*—These are not very frequent in children. They are characterized by high temperature, severe toxic symptoms, and great prostration. They often resemble cases of pneumonia, except that the local symptoms and physical signs in the chest are wanting. The onset is usually abrupt with vomiting and headache, sometimes even with convulsions. The temperature ranges from 100° to 106·5° F. It seldom remains steadily high, but often fluctuates widely. I have repeatedly seen a temperature over 106° F. in uncomplicated influenza. Marked nervous symptoms are usually present; there may

be headache, photophobia, delirium, stupor, opisthtotonus, and convulsions—all strongly suggesting meningitis, but not so continuous as in that disease. In other cases the tongue has a brown coating, the lips are dry and parched, the pulse is weak and rapid, and other symptoms of the typhoid condition are present. The usual duration of these severe attacks is from two to five days; but even where no complication develops severe symptoms may last for two weeks and sometimes longer until a change of climate is made. (See Fig. 217.) Although the symptoms are very alarming, except in young infants, the attacks are seldom fatal unless pneumonia develops; but it may be a long time before the full effects of such an illness have entirely disappeared.

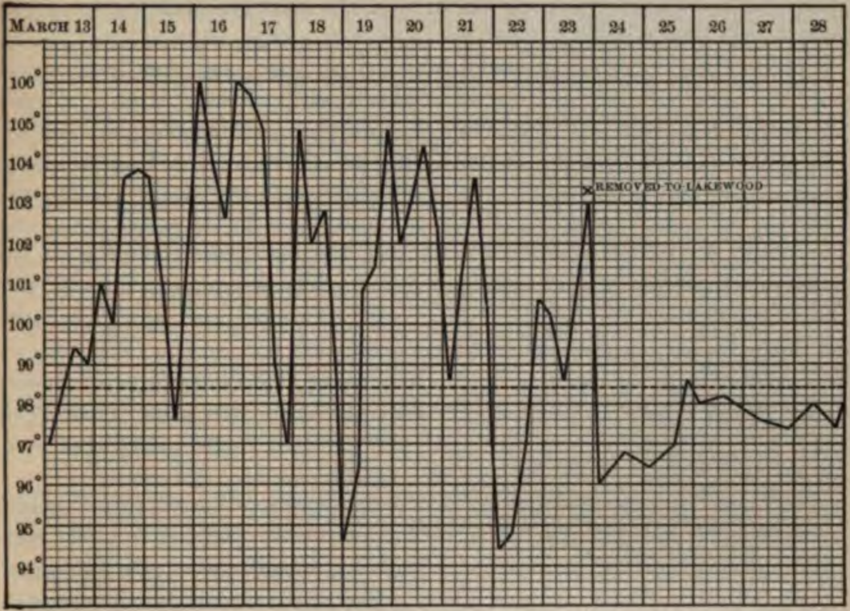


FIG. 217.—Temperature chart of uncomplicated influenza; infant fourteen months old. No local signs of disease; repeated blood examinations for malaria negative; the wide fluctuations of the temperature independent of therapeutic measures. Prompt cessation of fever on removal from the city. (Patient seen with Dr. L. E. La Fétra.)

3. *Cases complicated by catarrhal inflammation of the upper respiratory tract.*—In this group there are added to the general symptoms of the mild uncomplicated variety, a severe coryza, with pharyngitis and often stomatitis. The catarrhal symptoms differ from ordinary catarrh of these mucous membranes chiefly in severity. They are also likely to be more prolonged, and there is a greater tendency to involve the ears and the cervical lymph nodes. The usual symptoms of acute rhino-pharyngitis are present with its serous, sero-mucous, or muco-purulent discharge. The whole pharynx may be the seat of an acute, erythematous

blush, or the mucous membrane may present a granular or spongy appearance. The tonsils are red; occasionally there is follicular tonsillitis; rarely membranous patches. The nostrils and upper lip are often excoriated from the nasal discharge. The mouth may be the seat of a simple or a herpetic stomatitis with superficial ulceration. These catarrhal symptoms are usually severe for three or four days, and gradually subside. In infants the temperature may be 104° or 105° F. at the outset, but continues high only for a day or two. In older children the temperature ranges from 100° to 102° F.

There are two complications which in infancy are very frequent—otitis and cervical adenitis. Otitis may be either catarrhal or purulent. It runs the usual course of otitis following simple catarrhal processes of the pharynx, and usually terminates in complete recovery. Exceptionally these cases may go on to the development of chronic otitis, or the disease may extend to the mastoid cells. In addition to the severe cases, there are frequently seen attacks of catarrhal deafness from inflammation of the Eustachian tube. Pain in this form is less severe, and may be absent; there is no increased fever. Deafness is the chief symptom, and in most cases it disappears spontaneously.

The adenitis usually involves either the lymph nodes situated below the ear and behind the angle of the jaw, or those of the retro-pharyngeal region. The inflammation runs the usual course of such inflammations when associated with other diseases.

4. *Cases with broncho-pulmonary complications.*—A moderate amount of inflammation of the mucous membrane of the larynx, trachea, and large bronchi occurs in most of the cases of influenza. In the more severe forms, broncho-pneumonia or lobar pneumonia often develops. Sometimes the pulmonary symptoms do not appear for two or three days, or even a week; at other times they are coincident with the development of the fever and other constitutional symptoms, and, except for the prevalence of influenza, this would not be considered a factor in these cases. A striking feature in these attacks is that the temperature, prostration, and cerebral symptoms are out of all proportion to the pulmonary signs and symptoms.

The broncho-pneumonia complicating influenza may not differ essentially from the ordinary types, except that the proportion of cases which do not go on to the development of areas of consolidation is larger than is seen under most other conditions. If lobar pneumonia develops, it frequently runs its regular course. But besides these two varieties of pneumonia, quite a large number of cases of an irregular type are seen with influenza. These are often of short duration, but accompanied by extremely high temperature (Fig. 218). In many cases there is an excessive amount of pleurisy, so that the process is really a pleuro-pneumonia. In an epidemic occurring in the New York Infant Asylum in the winter of 1891 and 1892 nearly every pneumonia was of this type, and in a few weeks there were about twenty cases, all of a very severe form. This is often followed by empyema.

5. *Cases with gastro-enteric complications.*—Vomiting and diarrhœa are frequent at the beginning of influenza, and in some cases, especially in infants, they may be the predominant symptoms of the attack. The stools may be large and fluid, or they may contain mucus and even blood, and be passed with pain and tenesmus — the symptoms being those of an acute gastritis or of ileo-colitis of moderate severity. The duration of these attacks is usually three or four days, and except in very young or delicate children they are rarely fatal. In older children there may be initial vomiting, abdominal pain, tympanites, protracted diarrhœa, and other symptoms strongly suggestive of typhoid fever.

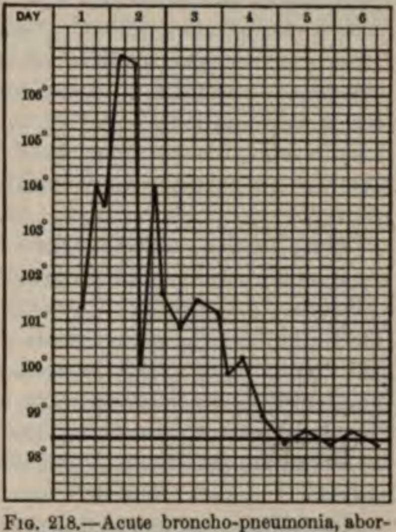


Fig. 218.—Acute broncho-pneumonia, abortive type, complicating influenza, in an infant six months old. The entire left lung posteriorly, was involved.

6. *Influenza in very young infants.*—The severe cases in infants under six months old often present peculiar features. The temperature may be very high, or it may be only two or three degrees above the normal, but the prostration is extreme. The eyes are sunken, the face is pale, there is marked apathy, and food is often refused altogether. In other cases there is cyanosis and very rapid respiration, indicating acute congestion of the lungs, although no abnormal signs are present, except very feeble breathing sounds. Nearly always there is a disturbance of digestion, with vomiting and undigested stools. Death may occur in two or three days; sometimes it is postponed for a week, the chief symptoms being gradually increasing prostration, and finally collapse, without the development of any marked local evidences of disease. The system seems in these cases to be overpowered by the intensity of the poison. In other cases pneumonia develops, and from this death occurs.

7. *Protracted cases.*—There has long seemed to be sufficient clinical ground for the opinion that influenza poisoning may sometimes assume a chronic or persistent form, and Pfeiffer and others have demonstrated the presence of the influenza bacillus for months in the secretions of such patients. The protracted cases in my experience have almost in-

[illegible] after which there is [illegible] irregular [illegible]. The temperature is not high, seldom [illegible] 102° F. [illegible] The [illegible] are not [illegible] that the general symptoms of a chronic [illegible]—poor appetite, coated tongue, anorexia, headache, lassitude, irritability, and occasional pains. The cases are often called malaria, or chronic malarial poisoning, and not infrequently tuberculosis is suspected; but the special features of all these diseases are wanting. In two cases I have seen the symptoms have been controlled by change of climate, but without this they have usually continued until the following warm season.

Complications and Sequelæ.—The most frequent ones—pneumonia, otitis, acute nephritis, and gastro-enteritis—have already been considered. Cutaneous eruptions are not infrequent, and are often very puzzling. There may be a general erythema resembling scarlatina, or an erythema which sometimes simulates measles, but more frequently scarlet fever. These eruptions are irregular in their course and often in their distribution, and are not followed by desquamation. In most of the cases with high temperature the urine contains albumin; although nephritis is rare, one should be on the watch for it even in young children. I have seen acute jaundice as a complication. The nervous sequelæ of adults—mental depression, multiple neuritis, etc.—are extremely rare in childhood, although they have been observed. One of the most frequent sequelæ is anæmia; this may be very severe, and in one case I have known it to continue to a fatal termination. Following the inflammation of the mucous membranes, there may be enlarged tonsils, adenoid growths of the pharynx, or chronic enlargement of the cervical lymph glands. Attacks of influenza bear the same relation to the development of tuberculosis as do those of measles.

Convalescence after influenza is usually very slow, and it is often many months before the full effects of a severe attack have disappeared. A recurrence of the symptoms before complete recovery is not uncommon, and often second attacks during the same season are seen. For a long time the mucous membranes are in an extremely sensitive condition. Relapses are often brought about by slight exposure before the symptoms have quite disappeared, and I have often seen them occur simply from airing an infant in the room.

Diagnosis. This is usually easy when the disease is epidemic. The sporadic cases often present great difficulties, particularly early in the disease. It is often impossible to tell for two or three days whether the case is one of pneumonia, malaria, or influenza. In most of the severe cases I have seen, pneumonia has been the diagnosis first made; it is only by the course of the disease and the absence of any physical signs, as shown by careful and repeated examinations, that influenza can be distinguished from pneumonia. From malaria, influenza is differentiated by the fact that the fever is not materially affected by quinine, there are no organisms in the blood, and the spleen is not usually enlarged.

The cerebral symptoms are less continuous than in meningitis and are usually in direct proportion to the fever. In the protracted cases, the temperature may bear some resemblance to typhoid, but the other characteristic symptoms of that disease are wanting. Measles is distinguished by Koplik's spots. In its mode of onset, and sometimes in its eruption, influenza often resembles scarlet fever, but the course of the symptoms usually clears up the doubt. In general, influenza is characterized by severe constitutional symptoms without evidence of local disease of sufficient importance to explain the temperature.

From ordinary catarrh, influenza differs only in its high communicability, its severity, and the frequency with which it is complicated by otitis, adenitis, and pneumonia. Mild cases when not epidemic can not be distinguished from simple catarrh of the respiratory tract.

Although in most cases the bacilli may be found by staining the sputum or nasal discharge, or may be cultivated from either of these, the difficulties in the way are such that this method of diagnosis has been as yet but little employed. In many cases the bacilli disappear early, and in others careful and repeated examinations are necessary to discover them. In general, therefore, the other symptoms of influenza must be relied upon for diagnosis. Since none of these is wholly characteristic, exact diagnosis is by no means easy, and in some cases it may be impossible. A probable diagnosis is made by excluding the other diseases mentioned; the probability is greatly increased if influenza is prevalent, especially if there are other cases in the same house. The tendency in practice is to call a great many other kinds of infection by the name of influenza, particularly when the disease is epidemic.

Prognosis.—As a rule, the type of influenza seen in children is milder than that which occurs in adults. In the case of children previously healthy, few die except from pulmonary complications, while the great majority of attacks are mild and recovery is prompt. In infants the tendency to pulmonary complications is much greater than in older children. Uncomplicated cases are seldom fatal, except in infants under six months old; and even though the temperature is very high and the symptoms severe, recovery may usually be predicted as long as there is no evidence of serious complications. The prognosis of the pneumonia of influenza is rather worse than that of simple broncho-pneumonia, and depends chiefly upon the age of the patients affected. In a word, influenza is particularly serious in the very young, or when there are pulmonary complications, but rarely otherwise. In infants the constitutional depression which results may be the beginning of a condition of malnutrition which goes on to the development of marasmus; or a child falls an easy victim to some other form of acute disease. The remote effects of influenza may therefore be serious, even though the attack itself is not especially severe.

Treatment.—The communicability of the disease makes it desirable that cases of influenza should be isolated whenever practicable, and particularly that delicate children, or those prone to pulmonary disease, should not be exposed. The fumigation of apartments after attacks should be regularly practised, preferably with formalin gas; this with isolation will do much to control house epidemics.

The disease usually runs its course, when uncomplicated, in from three to seven days. As there is no specific for influenza, the indications are to sustain the patient, to make him comfortable during the attack, and to prevent so far as possible the occurrence of complications. Every child with influenza should be put to bed and kept there during acute symptoms. At the outset the bowels should be opened by castor-oil or calomel, and free perspiration induced by the use of hot drinks, the hot pack, or small doses of Dover's powder in combination with phenacetine. A very high temperature should be relieved by cold sponging or the cold pack, precisely as in pneumonia, but large doses of antipyretic drugs are to be avoided. The nervous symptoms—restlessness, pain, headache, and other disturbances—are best controlled by phenacetine in combination with codeine—e. g., to a child of one year, phenacetine gr. j, codeine gr. $\frac{1}{40}$, every three or four hours. Double the dose may be given to a child of four years. Alcoholic stimulants are required whenever the pulse shows signs of weakness, as it does in most of the severe cases, and in most young infants. They should be given according to the same rules as in pneumonia. Next to alcohol, strychnine is the most valuable heart stimulant.

In older children there is a decided advantage in the use of moderately large doses of quinine—e. g., gr. ij, four or five times a day, to a child five years old; but in infants this should be omitted, on account of its tendency to upset the stomach. The cough which so often persists after influenza is best controlled by cod-liver oil and creosote, used as after acute bronchitis. With persistent bronchitis which resists ordinary remedies, a patient should be sent to a warm, dry climate. The complications of influenza are to be treated as they arise, in the same manner as when they occur under other conditions. In all cases careful feeding in accordance with the general rules laid down for feeding in acute diseases, good nursing, and care to avoid exposure during convalescence, are essentials in treatment. One should be particularly anxious about patients who have a strong tendency to tuberculosis, and such cases should be watched with the greatest solicitude.

In prolonged or constantly recurring attacks nothing is of much avail except a change of air. If this is impossible, a child should be frequently removed from one apartment to another, as re-infection often appears to take place from the sick-room.

CHAPTER XIII.

MALARIA.

MALARIA is a general infectious disease due to the presence in the blood of a specific organism often called the *plasmodium*, but more exactly the *hæmatocytozoön malariæ*. It manifests itself in children by the ordinary acute febrile attacks which are seen in adults and by chronic malarial poisoning. Both of these forms may present certain peculiar symptoms dependent upon the age of the patient.

Etiology.—The malarial organism was discovered by Laveran in 1881; it is a parasite of the blood and belongs to the group of protozoa. It is now well established that the parasite enters the blood through the bite of certain forms of mosquito, those belonging to the genus *Anopheles*, and probably in no other way. For this knowledge we are indebted chiefly to the work of Ronald Ross, in India, in 1897. For a general discussion of the malarial parasite, its methods of staining, etc., the reader is referred to works on clinical medicine.

Malaria affects all ages, even the newly-born infant. We must accept with some allowance the statements made by the older writers upon the subject of intra-uterine infection, but in the following case occurring in the practice of my former associate, Dr. Crandall, there seems little doubt that the disease was contracted *in utero*: For ten days before delivery the mother had suffered from a tertian intermittent of moderate severity. Eighteen hours after birth the child was noticed to have cold hands and feet, blue lips and nails, and a pinched face. These symptoms lasted about half an hour and were followed by a distinct fever. Upon the following day the paroxysm was repeated. Examination of the blood of both mother and child was made by Dr. Walter James, who found the malarial organisms in both cases.

Malaria is more frequently overlooked in young children than in later life, from the fact that its forms are more irregular, and this has led to the belief that young children are less liable than adults to the disease. I believe, however, the opposite to be the case. In a large number of instances where families have been exposed to malarial poisoning I have noted that the young children were frequently the first to show the symptoms of the disease.

Malaria is an endemic disease prevailing in certain localities. Exact knowledge regarding the mode of infection has cleared up many obscure points in the etiology of this disease. The *rôle* of the mosquito explains the greater liability to contract malaria after sunset and during the night, the danger from stagnant ponds and pools of water, the peculiar susceptibility of infants and young children, and the greater frequency of the disease in the spring and summer. Malarial attacks may, however, occur at any season, since the poison may be latent in the body for an indefinite time; how long it is impossible to say, but there seems to be conclusive proof that it may be for many months. Attacks of malaria very often occur when the general health has been reduced by some other cause, particularly by disturbances of digestion.

Lesions.—Opportunities for a study of the peculiarities of the lesions of malaria in children are infrequent, especially in New York, as fatal cases are extremely rare. I have myself seen but two. As observed by others, the lesions do not differ in any marked way from the adult form of the disease. The most important changes are the destruction of the red corpuscles of the blood, enlargement, and in chronic cases hyperplasia with pigmentation of the spleen; less frequently pigmentation of the liver, kidneys, and brain. Pneumonia and gastro-enteritis are occasional complications.

Symptoms.—The clinical forms of malarial fever in children from six to ten years old, do not differ essentially from the same disease in adults. Both intermittent and remittent forms occur, the former being the type usually seen. Of the different varieties of intermittent fever, the quotidian (Fig. 219) is the most common, although the tertian (Fig. 220) is fairly frequent, but in this locality the quartan is extremely rare. The stages of the paroxysm are generally well marked. The cold stage begins with a chill or vomiting, with headache, lassitude, and general pains. The hot stage is usually characterized by a higher temperature than in adults, and this is followed by the sweating stage, which is generally marked. The paroxysm may be repeated every day or every other day until controlled by quinine, or the stages may become less and less distinct as the disease progresses until a more or less remittent type of fever develops. Less frequently the fever is remittent from the beginning and the constitutional symptoms are of greater severity. In this form there is marked prostration, the tongue is thickly coated, there are often tenderness and pain in the region of the liver, and occasionally there is slight jaundice.

In infants and very young children peculiar types of malaria are seen. A well-marked intermittent fever with distinct stages is often absent, many cases assuming more of a remittent type or an irregular form of intermittent (Fig. 221). The onset is usually abrupt with vomiting, a well-marked chill being rare. Malarial chills are not often witnessed in children under five years old. They are replaced in infants by cold hands and feet, blue lips and nails, sometimes slight general cyanosis, pallor, drowsiness, and prostration. Vomiting has been present in two thirds of my own cases. Several times have I seen a malarial attack ushered in by convulsions.

The fever is relatively higher than in adults, rising rapidly to 104° or 105° F., occasionally to 106° or 106·5° F. This continues from four to twelve hours and gradually falls, usually to normal. The other constitutional symptoms of the febrile stage are much less severe than in most diseases with the same elevation of temperature. The sweating stage is only slightly marked and is often absent altogether. With the fall in the temperature there is a gradual subsidence of all the other symptoms of the febrile stage.

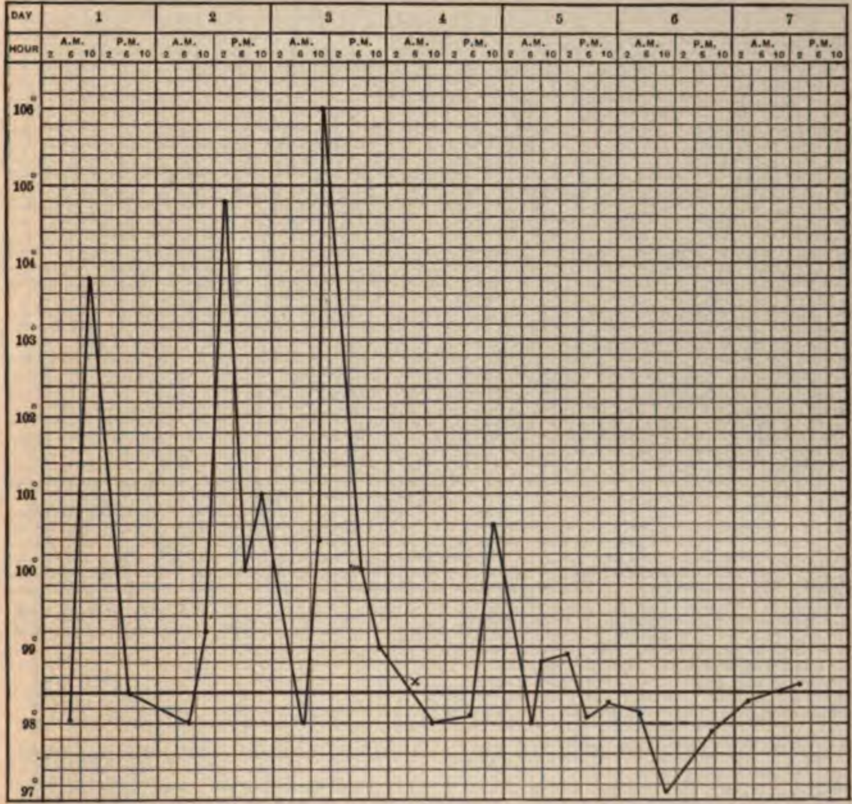


Fig. 219.—Typical malarial temperature, quotidian type, in a boy six years old. Each paroxysm preceded by a chill. It will be noticed that the temperature rose higher with each succeeding paroxysm; x marks the time when quinine was begun.

After the first paroxysm the patient may be quite well for several hours or even for a day, when the second paroxysm occurs. This is generally not so well marked as the first one, the third may be even less so, and the case may resemble more and more one of continuous fever with wide oscillations in the temperature. In some cases it is remittent at first and later becomes intermittent, but it is very rare under any circumstances that the temperature does not touch the normal point at some time in the twenty-four hours. In infants the quotidian has been in my experience very much more frequent than any other type, the tertian being rare and the quartan almost unknown.

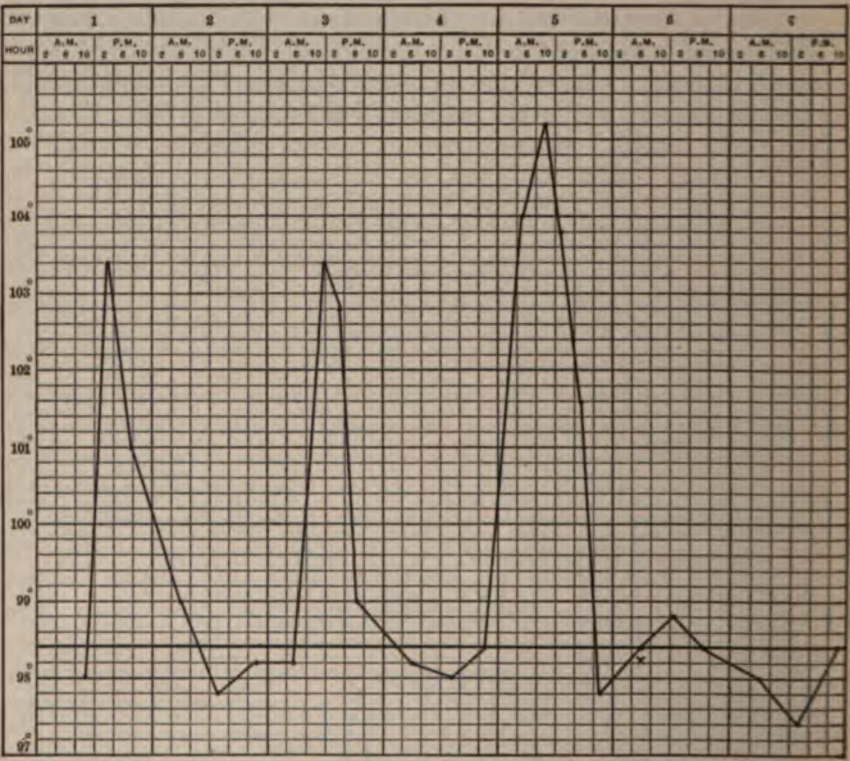


FIG. 220.—Typical malarial temperature, tertian type, in a boy five years old. Onset with vomiting and drowsiness, but no chill. This was an anticipating intermittent, the first paroxysm occurring at 3 P. M., the second at 12 M., the third at 10 A. M.; x marks the time when quinine was begun.

Enlargement of the spleen is present in the great majority of cases, and usually to a sufficient degree to be readily appreciated by examination. The most satisfactory method of examination is by palpation. A spleen which can be easily felt below the ribs (except in the rare cases in which the organ is displaced downward by some condition in the thorax) is enlarged. When it is not sufficiently enlarged to be nessed in children under five years old. They are replaced in infants by cold hands and feet, blue lips and nails, sometimes slight general cyanosis, pallor, drowsiness, and prostration. Vomiting has been present in two thirds of my own cases. Several times have I seen a malarial attack ushered in by convulsions.

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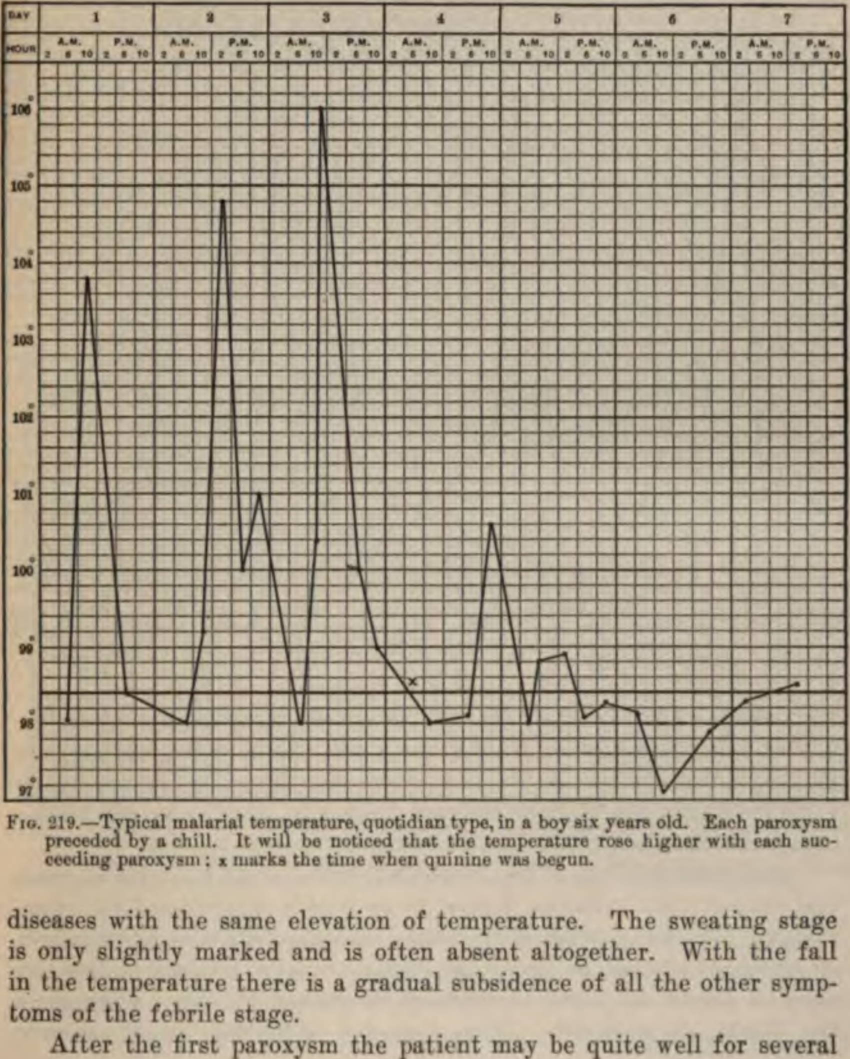

Fig. 219.—Typical malarial temperature, quotidian type, in a boy six years old. Each paroxysm preceded by a chill. It will be noticed that the temperature rose higher with each succeeding paroxysm; x marks the time when quinine was begun.

After the first paroxysm the patient may be quite well for several or liver. Trifacial neuralgia of malarial origin is rare in childhood. Aching or dragging pains in the muscles of the lower extremities are frequent symptoms during acute attacks, but they are of short duration, disappearing with the fever. They are to be distinguished from the acute lancinating pains of multiple neuritis, which is occasionally seen as a result of malarial poisoning. I have seen the latter in young children in three cases, and it has been observed by others. The pain is accompanied by tenderness of the muscles and nerve trunks, and by loss of power, which is usually partial.

Spasmodic torticollis (page 731) I have seen in eight cases, in which the condition seemed very clearly to depend upon malaria. This was shown by the fact that the spasm was intermittent, coming on every afternoon, but being absent in the morning; that it was accompanied by a slight rise in temperature, and usually by enlargement of the spleen; and that it was immediately controlled by quinine. This combination of symptoms seemed to be conclusive evidence of the malarial origin of the affection, although these cases were observed before the time when blood examinations were made.

Accompanying the paroxysm of malaria there is occasionally seen, more often in infants than in older children, acute pulmonary congestion (Fig. 221), which may give rise to obscure and often very alarming symptoms. There is an acute onset with vomiting and prostration, high temperature, cough, rapid respiration, and often slight cyanosis. On examination of the chest there is found feeble or rude respiration over one lung, or over both lungs behind, and sometimes coarse moist râles; these signs and symptoms may disappear in the course of a few hours with the fall in temperature, to return with the next paroxysm, or if quinine is given they may disappear entirely.[*] This group of symptoms has often led to the mistaken opinion that the disease was pneumonia, which had been aborted by the administration of quinine.

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[*] The following case is a good example of this condition in its more severe form, and illustrates the difficulties in the diagnosis of malaria in infancy: A fairly nourished child, nine months old, who had been under observation in an institution for two weeks, was suddenly taken with vomiting and fever (Fig. 221). A cathartic was followed by a large undigested stool, and as the temperature then fell to normal, the attack was regarded as one of indigestion. On the third day the temperature was again high and accompanied by cough; coarse râles were found throughout the chest, and fine râles at the right base; it was then thought that pneumonia was developing. On the fourth day all the symptoms were so much improved that the infant was regarded as convalescent. At 6 P. M. the temperature was normal, and the infant went to sleep quietly. At 9.30 P. M. he awoke with a temperature of 104°, extreme restlessness, and marked dyspnœa. In half an hour his symptoms had increased to a point where he seemed likely to die. He became cyanotic, the respirations were of a panting character and rose nearly to one hundred a minute, and he coughed with almost every breath; the pulse was scarcely perceptible. The severe symptoms continued for about

readily felt by a practised observer under favourable conditions for examination, it is not large enough to be of any diagnostic importance. None of the other symptoms occurring in malarial fever are characteristic; they are quite similar to those which are seen in almost all febrile attacks. They are anorexia, coated tongue, constipation, and restlessness.

Masked or Irregular Forms of Malaria.—These are quite frequent in young children, and are due to the presence of certain special or uncommon symptoms which may readily lead to a mistake in diagnosis. They are more often seen than cases of true malarial cachexia.

Among the most frequent of the irregular forms are those relating to the nervous system. Headache is exceedingly common and is usually frontal. When severe and associated with continuous drowsiness, vomiting, and constipation, it may lead to a strong suspicion of tuberculous meningitis. Vertigo is not a frequent symptom, but it is sometimes very prominent. Pains in various parts of the body are very common. A sharp severe pain at the epigastrium is frequent at the beginning of a paroxysm. It is often associated with tenderness, but has no relation to meals. Less frequently, pain is localized in the region of the spleen

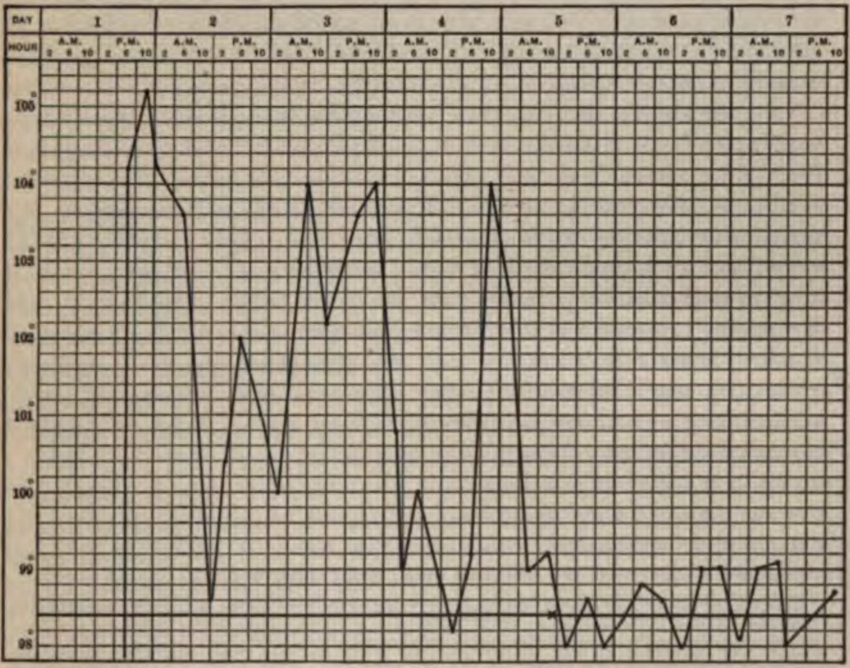

FIG. 221.—An irregular malarial temperature in a child nine months old. The paroxysm on the fourth day was accompanied by an attack of acute pulmonary congestion which came near being fatal; x marks the time when quinine was begun. Although the course of the temperature is irregular, it touched the normal line both on the second and fourth days.

in the absence of an examination of the blood, may be regarded as sufficient to establish the diagnosis of malaria.

The cachexia and course of the temperature in septicæmia, pyæmia, broncho-pneumonia, tuberculosis, and empyema, may easily cause them to be mistaken for malaria. The fever and recurring chills of pyelitis are often attributed to malaria; as are also the heaviness, lethargy, headache, coated tongue, and slight fever of chronic intestinal indigestion. Many conditions accompanied by an enlarged spleen may be confounded with malaria, especially simple anæmia, leukæmia, rickets, and syphilis. While malaria may be multiform in its manifestations, the physician can fall into no more serious error than to regard all ailments with obscure or indefinite symptoms as malarial, neglecting careful physical and blood examinations, by which means alone an accurate diagnosis is reached.

Prognosis.—Although it is seldom fatal in itself, an attack of malaria in an infant may so undermine the constitution that the child may succumb to some other acute disease, usually of the lungs or intestines. Cases are often difficult to cure while the patient remains in the malarial districts, and while a constant absorption of the poison continues. Under other circumstances the prognosis of malaria is good.

Treatment.—*Prophylaxis.*—More exact knowledge regarding the etiology of malaria makes it possible for much to be done in the way of prevention. Besides the general measures proposed for the extermination of the mosquitoes concerned, emphasis should be laid upon the necessity, in the case of young children, of protecting them against the bites of mosquitoes in localities which are or which may possibly be malarial. This can be done by a more thorough use of mosquito netting and by using upon exposed parts of the body lotions or ointments containing menthol, pennyroyal, turpentine, or other substances which keep these pests away. The general treatment is symptomatic, and is to be conducted as in all acute febrile diseases. In the cold stage, stimulants or a hot bath may be required; in the hot stage, ice to the head and frequent sponging. The bowels in all cases should be freely opened, preferably by calomel.

Methods of administration of quinine.—For infants my own preference is to give the bisulphate in an aqueous solution, one or two grains to the teaspoonful, according to the age of the patient. Most infants take such a solution with less difficulty and vomit it less frequently than the combinations with the various vehicles supposed to cover its taste. In the event of failure by this method, the same solution may be given *per rectum* through a catheter. It should then be more largely diluted with some bland fluid such as gruel, and in double the dose. This is necessary, not only because absorption is less certain and complete, but also because a rectal dose can seldom be repeated oftener than every five or six hours. There is sometimes an advantage in giving part of the quinine by the mouth and part of it by the rectum; should both fail it may be

SECTION X.

OTHER GENERAL DISEASES.

CHAPTER I.

RHEUMATISM.

THE rheumatic diathesis manifests itself in children by quite a different group of symptoms from those seen in adults; for this reason the disease was formerly supposed to be a rare one in early life. It is only within recent years that its frequency and its peculiarities have come to be appreciated. For our present understanding of the subject we are indebted largely to the work of English physicians, especially Cheadle,* who has brought out more fully than any one else the close connection existing between many conditions formerly not regarded as rheumatic. One who has in mind only the adult types of articular rheumatism, and regards arthritis as a necessary symptom for a diagnosis, will overlook in early life many manifestations which are clearly the result of the rheumatic poison. There is seen at this period a group of clinical phenomena, which often occur in combination or in succession, whose association was not understood until they were all discovered to be related to rheumatism. Sometimes one member of the group and sometimes another is first seen, but when one has appeared others are likely soon to follow.

Rheumatism in childhood, then, is manifested not alone by arthritis with acute or subacute symptoms, but by a large number of other conditions which are not to be regarded in the light of complications, but rather as forms of the disease.

Etiology.—It is not in the province of this work to discuss the various theories regarding the nature of rheumatism and its exciting cause. The drift of medical opinion to-day is strongly toward the view that acute rheumatism is an infectious disease, probably of microbic origin. Although the character of the micro-organism is not yet determined, the latest observations of Poynton and Paine † point to a diplococcus. The excessive formation of acids in the system may be regarded as a result of the infection, or possibly as a condition necessary for the activity of the specific poison. Under five years of age articular rheumatism is not common, and in infancy it is extremely rare. I once saw, however, in a nursing infant, a typical attack of rheumatic fever with multiple joint succeeded in getting the physiological effects of quinine by inunction, though there are good observers who claim this result. It is certainly a very uncertain way of introducing quinine into the system.

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* See the Harveian Lectures, 1889.

† Lancet, May 4, 1901.

Dosage.—Relatively much larger doses of quinine are required for young children than for adults. Except for its tendency to disturb the stomach, quinine is borne remarkably well by little patients. Generally too small doses are given. An infant of a year with a sharp attack of malarial fever will usually require from eight to twelve grains of the sulphate (ten to fourteen grains of the bisulphate) daily. Occasionally I have found it necessary to give double the quantity referred to, and I have seen no unpleasant cerebral symptoms. It is useless to expect to control an acute attack of malaria by such doses as one grain three or four times a day. Children from five to ten years old require almost as large doses as do adults. None of the substitutes for quinine are to be relied upon in acute cases.

In chronic cases, arsenic and iron are usually required in combination with smaller doses of the quinine than those mentioned. For children over seven years old, Warburg's tincture may be employed. In most chronic cases a cure can be effected only by a change of climate.

The marked and irregular manifestations of malaria are to be treated in the same manner as cases of malarial fever.

Subacute or Chronic Forms of Malaria.—The most constant symptoms are anæmia, enlargement of the spleen, and slight fever. The anæmia is usually marked, often being extreme. The enlargement of the spleen is distinct, easily made out by palpation, and sometimes is very great. The fever is often so slight as to be discovered only when the temperature is taken five or six times in the twenty-four hours. The other symptoms are of a very indefinite character; there may be slight œdema of the lower extremities, general muscular weakness, so that the child is easily fatigued, loss of appetite, coated tongue, constipation, headache, muscular pains, and often cough from a slight bronchitis. These symptoms may depend upon many conditions other than malaria, even when they are seen in a malarial district. The only positive evidence of malaria in such cases is the presence of the malarial organisms in the blood. Even the swollen spleen, anæmia, and slight fever, which are often looked upon as diagnostic, may be present in cases of anæmia with which malaria has nothing whatever to do.

Diagnosis.—The positive diagnosis of malaria rests upon the demonstration of the malarial organisms in the blood. They will be found in nearly all the cases provided a careful examination is made during the paroxysm, and also that no quinine has been administered. When their number is small they may be missed at the height of the fever, although they may readily be found just before the temperature begins to rise. Blood from the spleen is more certain to show the organisms than that from the finger; and if possible the examination should be of fresh blood as well as of stained specimens. While a positive result is conclusive, a negative one is not always so because of the impossibility of fulfilling all the above conditions. This fact and lack of experience in blood examinations make it necessary for a large part of the profession to make the diagnosis by the other symptoms. These, in order of their importance, I would place as follows: Prompt curability (especially in cases of fever) by quinine; distinct periodicity in the symptoms; enlargement of the spleen; and a history of an exposure in a district known to be malarial. Particular importance is to be attached to the therapeutic test. Recent experience emphasizes more and more strongly the fact that quinine has very little influence upon fevers which are not malarial, and, conversely, that a fever immediately and permanently controlled by quinine is pretty certain to be malarial. The combination of all the above symptoms, even

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an hour, then passed away gradually, and at the end of two and a half hours they had completely disappeared, and the child was in a quiet sleep which continued until morning. Malaria was now suspected, and the diagnosis established by the discovery of the plasmodium in the blood. The spleen was at this time much enlarged; the signs in the chest were those only of bronchitis of the large tubes. Quinine was now begun in full doses, and immediately controlled the temperature and the pulmonary symptoms.

given hypodermically. For this purpose the bimuriate of quinine and urea, the hydrochlorosulphate, the hydrobromate, or the bisulphate may be used. The salts first mentioned have the advantage of greater solubility. But all are more or less irritating and there usually follows some induration at the site of the injection, which may last a long time. While the hypodermic use of quinine is sometimes invaluable it should not be employed in infants except in serious attacks and when we are tolerably certain of our diagnosis. In a number of instances both in hospitals and private practice I have seen ugly sloughing follow the use of nearly all the preparations generally employed. The occurrence of abscess points to infection at the time of injection; but necrosis I believe may be due simply to the irritation of the quinine upon tissues having a lowered vitality, as in the case of young or delicate infants. I have seen this happen when the strictest precautions against infection were observed. The frequent repetition of the hypodermic injections should be avoided; in most cases, one or two good doses are sufficient, the effect being continued by quinine given by other methods.

For children from two to seven years old the taste of quinine must be concealed. An aqueous solution of the bisulphate may be mixed with the syrup of sarsaparilla, orange, or yerba santa; or the sulphate may be given in suspension in the same vehicle, the mixture being made just before the dose is taken; otherwise the partial solution of the drug will render the whole dose exceedingly bitter. When the dose required is not large, as in the milder cases, the lozenges of the tannate of quinine combined with chocolate answer the purpose admirably, for these are so nearly tasteless that children will take them without difficulty. Each lozenge usually contains one grain of the tannate, which is equivalent to about one third of a grain of the sulphate of quinine. A similar lozenge containing one grain of the sulphate may be made, which is often taken by children without the slightest objection. The bisulphate may be given in solution by the rectum, or, better, at this age, in the form of suppositories; but, as in infancy, with very urgent symptoms, it is better to resort at once to the hypodermic method in case of failure by the stomach.

For children over seven years old, the same methods of administration may usually be employed as in adults. It is always preferable to give quinine in solution, or if not so, in capsule, but never in pill form.

In a case with well-marked paroxysms the quinine should if possible be given in the interval, with the largest dose about four hours before the expected paroxysm. With infants this plan is sometimes impracticable, as frequent small doses are usually better borne by the stomach than a few large ones. In them also vomiting seems less likely to occur when it is given on an empty stomach. For this reason it is advantageous to give the drug at regular two- or three-hour intervals during the night, and omit all medication during the day. I have never

SECTION X.

OTHER GENERAL DISEASES.

CHAPTER I.

RHEUMATISM.

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Rheumatism in childhood, then, is manifested not alone by arthritis with acute or subacute symptoms, but by a large number of other conditions which are not to be regarded in the light of complications, but rather as forms of the disease.

Etiology.—It is not in the province of this work to discuss the various theories regarding the nature of rheumatism and its exciting cause. The drift of medical opinion to-day is strongly toward the view that acute rheumatism is an infectious disease, probably of microbic origin. Although the character of the micro-organism is not yet determined, the latest observations of Poynton and Paine † point to a diplococcus. The excessive formation of acids in the system may be regarded as a result of the infection, or possibly as a condition necessary for the activity of the specific poison. Under five years of age articular rheumatism is not common, and in infancy it is extremely rare. I once saw, however, in a nursing infant, a typical attack of rheumatic fever with multiple joint lesions; and undoubted cases have been reported at as early an age as two months. In 1899 Miller (Philadelphia) could find in medical literature but nineteen cases under one year. The condition is therefore so exceptional that one should be cautious in making the diagnosis of rheumatism in infancy. Most of the cases so regarded are examples of scurvy. After the fifth year both the articular and the other manifestations of rheumatism become very common, and occur with increasing frequency up to the time of puberty.

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* See the Harveian Lectures, 1889.

† Lancet, May 4, 1901.

Heredity is a very important etiological factor, and in fully two thirds of the cases that have come under my care, a rheumatic family history was obtained. Of the other important causes, the most frequent are living in damp dwellings, direct exposure to cold and wet, poor hygienic surroundings, and insufficient food. While seen among all classes, rheumatism is more common among those who are badly housed. Attacks of rheumatism occur at all seasons, but are much more frequent in the spring months. One attack strongly predisposes to a second, and in most cases there is a history of a large number of attacks of greater or less severity. Among my own patients, girls have been affected with greater frequency than boys.

Symptoms.—*The general and articular manifestations.*—The clinical types of rheumatism in children present very notable contrasts to those seen in adults. A typical attack of acute articular rheumatism such as is seen in adult life, with a sudden onset, high temperature, severe inflammation of several joints, profuse acid perspiration, and occasional delirium, is rarely seen in a child under eight or ten years old. In most of the attacks in childhood the onset is not very acute, the temperature is but slightly elevated—only 100° or 101·5° F.—the swelling and pain are moderate, and the redness is often absent. The number of joints involved is generally small, those most frequently affected being the ankles, the knees, the small joints of the foot, the wrists, or the elbows. These symptoms are often not severe enough to keep the patient in bed, and only the pain in the joints of the lower extremities prevents him from walking. The duration of these attacks is from one to three weeks, and in the course of a month most of them recover even without treatment.

Not infrequently the symptoms are limited to a single joint, usually the hip, knee, or ankle. Possibly the joints of the upper extremity are affected oftener than would appear, but disease here is much more likely to be overlooked than when lameness is present. The swelling is moderate and may not be evident except on a close examination; in some cases there is none. There is stiffness of the joint, as shown by lameness, and there may be so much pain and soreness that the child refuses to walk altogether. Muscular spasm about the affected joint is often marked, and may be the most striking objective symptom. The tenderness is sometimes localized, but it may affect the ligaments, tendons, and even the muscles. These symptoms may persist for two or three weeks and lead to the suspicion of incipient tuberculous disease of the joint. Rheumatism is distinguished by its more acute onset and usually by the presence of slight fever; some elevation of temperature being the rule, though it is not often much over 100° F. A family history of rheumatism, or a history of previous similar attacks in the patient affecting the same or other joints, or other manifestations of rheumatism, are also of assistance in the diagnosis. Occasionally all doubt is removed by the disease extending to other joints, or by the development of endocarditis. In some cases the symptoms are less in the articulation than in the muscles, and they are dismissed as simply "growing pains," having nothing characteristic about them except their occurrence in damp weather.

Cardiac manifestations.—These may occur where the articular symptoms are very mild, and in some cases where they are entirely absent. The most frequent is endocarditis. This is much more often seen in the acute rheumatism of children than of adults, and probably occurs in the majority of all severe cases; if it does not come in the first attack, it is likely to be seen in the later ones. It frequently occurs with a mild rheumatic arthritis, often being unnoticed until valvular disease of considerable severity has developed. Sometimes there is only high fever with severe constitutional symptoms of an indefinite character, but no arthritis, and no suspicion that the attack is rheumatic until endocarditis is discovered. Such cases are not infrequent. If the patients are kept under observation, articular symptoms are almost certain to develop later, and often there are other manifestations of rheumatism, especially chorea.

Pericarditis is less frequent than endocarditis, and usually occurs in children over seven years old. It is often associated with endocarditis. The most characteristic form of inflammation in early life is a subacute, dry, fibrous form, often resulting in great thickening with extensive adhesions, and frequently in obliteration of the pericardial sac. When once started it shows a strong tendency to recurrence and persistence.

The heart is so frequently affected in the rheumatism of childhood that it should be closely watched whenever articular symptoms are present, no matter how mild they may be; and not only in these cases, but in all the conditions hereafter enumerated with which rheumatism is likely to be associated.

Inflammations of other serous membranes—the pleura, peritonæum, and pia mater—were much more frequently ascribed to rheumatism in the past than now. There is little doubt that on rare occasions any one of these may be due to rheumatism. The pleura is most often involved, but even this is rare in young children.

Torticollis when it occurs acutely is frequently rheumatic. This form is characterized by its sudden development, continuous spasm, the great amount of muscular soreness, the moderate pain, and the fact that it usually disappears spontaneously after a few days. It is often seen in connection with a rheumatic sore throat. Other manifestations of muscular rheumatism are less characteristic and usually affect the muscles of the extremities.

Anæmia is almost invariably seen in rheumatic patients, both during and between the attacks. The effect of the rheumatic poison upon the blood resembles that of malaria. The presence of anæmia is so evident and its degree often so marked, that one may have great difficulty in distinguishing cardiac murmurs which are hæmic from those due to endocarditis.

Chorea.—In the article upon Chorea I have already discussed the association of that disease with rheumatism and expressed my own belief in a very close relationship existing between them. Not very infrequently chorea is the first manifestation of the rheumatic diathesis, to be followed soon by articular symptoms or by endocarditis without such symptoms. In other cases chorea and acute endocarditis occur together without articular symptoms, or all three may be associated. Whichever of the three conditions is first seen, the physician should always be on the lookout for the others. The frequency of rheumatism in choreic patients has been variously estimated by different observers; in my own cases over fifty-six per cent gave unmistakable evidence of the rheumatic diathesis.

Tonsillitis.—The association of tonsillitis and pharyngitis with rheumatism appears in many cases to be a close one. Children who are the subjects of frequent attacks should be regarded as possibly rheumatic, and closely watched for other signs of that disease. Acute tonsillitis often ushers in an attack of rheumatic arthritis, and occasionally acute endocarditis without articular symptoms. Rheumatism may be associated with any form of tonsillitis, but its connection with quinsy seems closest. The nature of the relationship is not yet fully explained; by many the tonsils are regarded as the structures through which the rheumatic poison is absorbed. Packard (Philadelphia), however, regards the tonsillitis as non-rheumatic, and the endocarditis as of septic origin.

Subcutaneous tendinous nodules.—General attention was first drawn to these as a manifestation of rheumatism by Barlow and Warner, in 1881, who described them as " oval, semi-transparent, fibrous bodies like boiled sago grains." They are most frequently found at the back of the elbow, over the malleoli, at the margin of the patella; occasionally on the extensor tendons of the hands, fingers, or toes, or over the spinous processes of the vertebræ or the scapulæ. They are composed of fibrin, cells, and fibrous tissue, and vary in size from a large pin's head to a small bean, sometimes being as large as an almond. The nodules may come in crops, lasting for a few weeks and then disappearing, or they may last for months. An eruption of nodules is usually coincident with other rheumatic manifestations. These nodules are better felt than seen, although, as Cheadle observes, they are visible if the skin is tightly drawn. They are certainly not common in this country; and although I have made it a rule to examine rheumatic patients for them, I have seen them but seldom, and they have been prominent in only two or three cases. This, I think, has also been the experience of most observers in New York. From published reports, however, they appear to be much more frequent in England. There can be no doubt regarding the connection of these nodules with rheumatism.

Erythema.—The connection between rheumatism and the various forms of erythema—marginatum, papulatum, and nodosum—has been very clearly shown by Cheadle. None of these are frequent conditions in childhood, but when seen they should always suggest rheumatism.

Purpura.—The association of purpura with rheumatism is so often seen that there can be little doubt of the close connection between the two conditions. Rheumatic purpura, however, is quite distinct from the other forms of purpura, and is a much less frequent disease.

Diagnosis.—In order to recognise rheumatism in a child, one must free his mind from preconceived notions of the disease drawn from its manifestations in adults, as very few cases correspond to the adult type of acute rheumatism. In early life the disease is recognised not by any one or two special symptoms, but by the association or combination of a number of conditions which may appear unrelated. In determining whether or not any given set of symptoms is due to rheumatism, one should consider: (1) The family history, since in early life heredity is so important an etiological factor; (2) the previous history of the patient, not only as regards articular pains and swelling, the slight joint-stiffness without swelling, the indefinite wandering pains of damp weather, and the so-called growing pains, but also the previous existence of chorea, frequent attacks of tonsillitis, torticollis, or erythema; (3) the examination of the patient, which should include a careful search for tendinous nodules, as well as a thorough examination of the heart for signs of endocarditis or pericarditis, and, in cases which are at all acute, the temperature. In doubtful cases with mon-articular symptoms much importance is to be attached to the presence of slight fever, the abrupt onset, and tenderness of the neighbouring muscles and tendons,—all occurring without a history of traumatism. Rheumatism is more often overlooked than confounded with other diseases; although in childhood multiple neuritis and tuberculous and syphilitic bone disease are often mistaken for it, and in infancy the same is true of scurvy. The extreme infrequency of rheumatism during the first two years of life should always make one skeptical regarding it. In an infant, when the symptoms are confined to the legs and are not accompanied by fever, they are almost certain to be due to scurvy even though the gums are normal and ecchymoses have not yet appeared. Multiple gonococcus arthritis has often been diagnosticated rheumatism.

Prognosis.—Rheumatism in a child is in itself seldom if ever dangerous to life. In the great majority of cases the articular symptoms soon disappear, even without special treatment. The danger from the disease consists in its cardiac complications. One attack of rheumatism is almost certain to be followed by others, and when once the heart has been affected its lesions are likely to increase with each recurrence of the disease.

Treatment.—Rheumatism in children derives its chief importance from its relation to cardiac disease. Cardiac complications are so frequent and so serious that everything possible should be done to avert rheumatism from those who by inheritance are especially predisposed to it, to prevent its recurrence in a child who has once had the disease, and during an attack to prevent the heart from becoming involved. The relation of diet to rheumatism is very imperfectly understood; but it is certainly a fact that rheumatic children do much better upon a diet composed largely of nitrogenous food, where starches are restricted in amount, than the reverse. Milk should be freely given in all cases. The underclothing should be of flannel during the entire year, in summer the lightest weight being worn. The feet should be carefully protected, and exposure in damp weather avoided. In-door occupations should be chosen for rheumatic boys.

The tendency to recurrence is so strong in this disease that a child of rheumatic antecedents, who has shown in the various ways mentioned a marked predisposition to rheumatism, and who has had an attack, even though a mild one, should, if possible, spend the winter and spring in some warm, dry climate, or even remain there permanently. Otherwise in most such children, it is only a question of time when, with the repeated attacks, the heart will become involved.

To avert the danger of cardiac complications during an attack of rheumatism, or to limit their extent, there are two things which should invariably be insisted on: first, to confine to the house and in a warm room every child with rheumatic pains, no matter how mild; secondly, if fever is also present, to keep the child in bed while it continues, even though it may never be above 100° F. Absolute rest and the equable temperature thus secured are unquestionably of more importance than anything else in protecting the heart during a rheumatic attack. With these precautions must be combined an early diagnosis. In very many, perhaps in most cases, the harm is done before the true nature of the disease is suspected, the symptoms being dismissed as of slight importance because the articular manifestations are not very severe. Children who have once had rheumatism should be closely watched during chorea and other diseases related to rheumatism, the heart should be frequently examined, and the physician should be on the alert for the first articular symptoms.

Aside from the measures just mentioned, the treatment of rheumatism in childhood is to be conducted very much like that of adult life. In the most acute attacks either salicylate of soda, oil of wintergreen, or salicin should be given; as the majority of cases are not very acute, marked improvement is by no means always obtained by these drugs. Alkalies should be given in all cases, but particularly in those in which there is hyperacidity of the urine. Either the acetate or citrate of potassium or the bicarbonate of sodium may be used, a sufficient quantity being administered to render the urine alkaline.

Quite as important as these drugs is the use of general tonics, particularly iron and cod-liver oil. These should be given not only between attacks to fortify patients against their recurrence, but also in subacute cases which are sometimes influenced very little or not at all either by salicylates or alkalies.

CHAPTER II.

DIABETES MELLITUS.

In this chapter will be attempted only a description of the peculiar features which diabetes presents when affecting young patients. It is a very infrequent disease in children. Of 1,360 cases of diabetes collected by Pavy, only eight were under ten years of age. In a series of 700 cases collected by Prout, only one case was under ten years. In a series of 380 cases collected by Meyer, only one case was under ten years of age.

Etiology.—Stern, in a series of 117 collected cases of diabetes in children, states that 47 were females and 31 males, the sex in the other cases not being given. Although extremely rare, cases have been observed during the first two years, and even during the first year of life. Statistics on this point are not altogether trustworthy, since some cases of temporary glycosuria have certainly been included.

Among the etiological factors, heredity is one of the most important. Pavy reports the case of a child dying of diabetes at two years in whose family the disease had existed for three generations. Inherited gout, insanity, and nervous diseases generally, may be looked upon as factors in the production of diabetes. Several of the cases reported in children have been preceded by injuries received upon the head. In a number of my own cases the disease has followed the consumption of large quantities of sugar for a long time. Often no adequate cause can be found.

Symptoms.—The most important early symptoms are thirst, polyuria, and wasting; their development is often quite rapid. The thirst is intense, often leading children to drink four or five pints of fluid a day. The amount of urine passed varies from one to eight quarts daily. The specific gravity is from 1,026 to 1,040, and the usual amount of sugar is from three to five per cent, rarely more. Albumin is not infrequently present. Incontinence of urine is an important symptom, and often one of the earliest to be noticed. The wasting is usually quite rapid, so that a child may lose as much as six or eight pounds in a month. It is generally accompanied by anæmia. The appetite may be poor; at times, however, it is voracious. Other symptoms of less importance are a dry mouth, scanty perspiration, irregular sleep, occasional epistaxis, furuncles and abscesses, decayed teeth, and genital irritation.

The course of the disease is much more rapid in children than in adults, and, as a rule, the younger the child the more rapid its progress. The majority of cases prove fatal in from two to four months from the time the symptoms are sufficiently marked to make the diagnosis possible. Very few last more than six months; occasionally, however, one of the milder type may be prolonged from one to two years.

The progress of the disease is marked by continuous wasting, which may result in a marked degree of marasmus, and prove fatal. Some are carried off by intercurrent pneumonia or tuberculosis, but the majority die comatose. When coma develops, the case may be considered hopeless, and death is likely to be postponed but a few days. The cause of diabetic coma has not yet been satisfactorily explained, but it is usually believed to be due to acetonæmia.

Diagnosis.—Diabetes is apt to be overlooked, because of the common neglect of urinary examinations in children. The prominent symptoms—thirst, polyuria, and wasting—when associated, should always attract attention. Incontinence of urine, accompanied by marked wasting, is always suspicious. In some cases genital irritation may be the most prominent early symptom. A positive diagnosis is made only by an examination of the urine.

Prognosis.—In few diseases is the prognosis so bad as in diabetes in children. So high an authority as Senator declares that diabetes in children is hopeless and all treatment is useless. From a study of seventy-seven cases, Stern reaches the same conclusion. There are, however, cases on record in which recovery is believed to have taken place. The cases which I have seen have all terminated unfavourably. In a given case the prognosis, as to the duration of the disease, is rendered much worse by the presence in the urine of diacetic and oxybutyric acids. This condition is even more serious than is a high percentage of sugar; that the patient will then live more than three months is highly improbable.

Treatment.—The indications for treatment are the same in children as in adults: first, diet; secondly, general hygienic measures; and, finally, the use of drugs, of which at the present time the favourites are codeine, salicylate of soda, and the bromide of arsenic.

INDEX.

Abdomen, examination of, 39; growth of, 24; in rickets, 265.

Abscess, alveolar, 279; cerebral, 780; symptoms, 781; treatment, 782; cerebral, in acute otitis, 948; ischio-rectal, 457; mammary, 116; hepatic, 460; peritoneal, 467; peritonsillar, 310; perityphlitic (see appendicitis), 439; psoas, in spinal caries, 906; retro-œsophageal, 316; retro-pharyngeal, in Pott's disease, 298, 908; retro-pharyngeal, of infancy, 295; subphrenic, 477.

Abscess, multiple, in malignant endocarditis, 626; multiple, in newly born, 85.

Acetonæmia in diabetes mellitus, 1148.

Acetonuria, 651.

Achondroplasia (see CHONDRO-DYSTROPHY), 810.

Acid, hydrochloric, increased by lavage, 346; hydrochloric, in gastro-enteric intoxication, 379; hydrochloric, in stomach digestion, 320; lactic, in stomach digestion, 320.

Adenie (see HODGKIN'S DISEASE), 895.

Adenitis, acute, 831; acute axillary, 885; acute cervical, 885; acute inguinal, 885; cervical, in diphtheria, 1027; in influenza, 1125; in measles, 989; retro-œsophageal, 316; retro-pharyngeal, 295; simple acute, 883; simple chronic, 886; syphilitic, 887; tuberculous, 888; treatment, 893.

Adenoid vegetations of pharynx, 299, 481; symptoms, 301; treatment, 303; asthma from, 524; causing acute nasal catarrh, 481; chronic laryngitis with, 506; in rickets, 268; removal advised in tuberculous adenitis, 894; with adenitis, 887.

Adenoma of umbilicus, 114.

Agenesis, cortical, 796.

Airing, when allowed out of doors, 8.

Air-space required by infants, 10.

Alalia, 740.

Albinism, stigma of degeneration, 818.

Albumin water, preparation of, 337.

Albuminuria, functional or cyclic, 644; in chronic cardiac disease, 630; in chronic nephritis, 669; in influenza, 1128; in measles, 988; in pertussis, 1010; in scarlet fever, 967; in typhoid fever, 1066.

Alcohol, as stimulant, 51; as tonic, 52; effect on breast milk, 174; use of, in diet of nurse, 138.

Amaurotic family idiocy, 807.

Amyloid degeneration, in chronic bone disease, 902; of the intestines, 410; of the liver, 410; of the spleen, 410.

Anæmia, cardiac murmurs in, 637; following diphtheria, 1029; pernicious, 865; pseudo-leukæmic, of infancy, 863; treatment, 867; simple, 860; treatment, 867; with adenoids, 303; in malaria, 1137; in malnutrition, 232; in marasmus, 241; in rheumatism, 1144; in rickets, 267; in scurvy, 248; in tuberculosis, 1096; preceding tuberculosis, 1089.

Anæsthesia, partial, in multiple neuritis, 849.

Anæsthetics, those best for children, 68.

Anasarca, general, in acute diffuse nephritis, 664; in chronic cardiac disease, 630.

Aneurism, 639.

Angina, catarrhal, in measles, 986; in scarlet fever, 964.

Anglo-Swiss food, 166.

Ankle, enlarged epiphyses in rickets, 264.

Anodynes, 53.

Antipyretic drugs, 50.

Antipyretics, 48; in acute broncho-pneumonia, 559.

Antipyrine, in chorea, 727; in catarrhal croup, 491; in pertussis, 1014; scarlatiniform rash from, 971.

Antitoxin, in the treatment of tetanus, 92; eliminated by human milk, 139; results without, in membranous laryngitis, 497; with, 1053 (see DIPHTHERIA ANTITOXIN); streptococcus, 1062.

Anuria, 652

Anus, fissure of the, 454; imperforate, 117.

Aorta, abnormal origin of, 612; aneurism of, 639; atheroma of, 639; congenital narrowing of, in chlorosis, 862; hypoplasia of, 639; thrombosis of, 640.

Aortic insufficiency, 633; stenosis, 632.

Aphasia, functional, 740; in acquired cerebral paralysis, 802; after typhoid fever, 1077; motor, in cerebral tumour, 785, 786.

Aphonia, hysterical, 735; in diphtheritic paralysis, 852.

Appendicitis, 438; lesions, 438; symptoms, 440; diagnosis, 443; leucocyte count an aid in, 444; treatment, 444.

Arm, paralysis of, at birth, 111.

Arnold sterilizer, 156.

Arsenic, as a tonic, 52; dosage in chorea, 727.

Arteries, hypogastric, in fœtal circulation, 606; hypoplasia of, 639; umbilical, in fœtal circulation, 606.

Arthritis, acute, of infants, 899; acute suppurative, syphilitic, 916; gonococcus, 686, 691, 899; rheumatic, 664.

Arthrogryposis (see TETANY), 716.

Artificial feeding, 182; *versus* wet-nursing, 170.

Ascaris lumbricoides (see WORMS, INTESTINAL), 448.

Ascites, 476; detection of, 476; chylous, 476; in acute diffuse nephritis, 664; in cirrhosis of liver, 462; rare with amyloid liver, 463; with chronic peritonitis, 469; with tuberculosis of the peritonæum, 471.

Asphyxia, death from, in young children, 46; from overlying, 44; from aspiration of food, 45; from enlarged thymus, 45; in convulsions, 706; in retro-pharyngeal abscess, 297; in the newly born, 69; from tuberculous bronchial lymph nodes, 1102; methods of resuscitation, 72; sudden, from tongue-swallowing, 278; sudden, in retro-œsophageal abscess, 317.

Aspiration of chest in empyema, 602.

Asthma, 523; etiology, 524; symptoms, 524; diagnosis, 526; prognosis, 526; treatment, 526; catarrhal, 525; with adenoids, 302; long uvula, cause of, 295; simulated by tuberculous bronchial glands, 1101.

Astigmatism, stigma of degeneration, 819.

Ataxia, Friedreich's, 841; in multiple neuritis, 849.

Atelectasis, acquired, 588; from compression, 588; from obstruction, 588; in delicate infants, 589; causing sudden death, 45; congenital, 74; in marasmus, 239.

Atheroma, 639.

Athetoid movements, 728; in acquired cerebral paralysis, 802; in birth paralysis, 799.

Athetosis, 728.

Athrepsia (see MARASMUS), 238.

Atomizer, 57, 61.

Atresia ani, 352.

Atrophy, infantil
muscular, faci
neuritis, 849;
gressive musc
peroneal type,

Atropine, hypod
fantum, 384.

Aura of epilepsy

Autopsies, princi

Babcock's centrif

Bacillus of diph
tribution in t
142; in health
geal diphther
1042; of dys
colitis, acute,
intoxication, a
typhoid fever,
acute broncho
Loeffler (see
lactis aerogene
fluenza, 1123;
of tuberculosis
pneumonia, 5
paths of infect

Backwardness, 8

Bacteria, etiolog
human milk,
146, 153, 157;
cow's milk, 14

Bacterium coli
dicitis, 438;
tion, 368; in

Bacterium lactis

Balanitis, 686.

Band, abdominal

Barley water, di
use during firs

Barlow's disease

Bath, at birth,
broncho-pneum
newly born, 7
56; hot air,
56; bran, 57
cold sponge,
newly born, 7

Bed-wetting, 692

Beef, broth, 164
pressed, 163;
163; preparati
164.

Belladonna, 53;
139; scarlatin

Bile, physiologic

Bile-ducts, cong
77.

Birth paralyses
spinal, 107; p

Bladder, control
of, 685; hæm

born, 105; stone in, 698; training to control, 4.
Bleeders, 870.
Blindness, hysterical, 734; stigma of degeneration, 819; transient, in pertussis, 1010.
Blisters, 54.
Blood, circulation of, in early life, 606; corpuscles, red, 856; corpuscles, white, 857; diseases of, 856; hæmoglobin, 856; in chlorosis, 863; in diphtheria, 1029; in empyema, 600, 859; in leukæmia, 869; in measles, 988; in pernicious anæmia, 866; in pertussis, 1011; in pneumonia, 859; in pseudo-leukæmic anæmia, 864; in scarlatina, 969; in simple anæmia, 861; leucocytes of, varieties of, 857; leucocytosis, 858; plasmodium malariæ in, 1137; specific gravity, 856; blood letting, local, 55.
Blood-vessels, diseases of, 639; aneurism, 639; arterial hypoplasia, 639; atheroma, 639; embolism, 640; thrombosis, 640.
Boil (see FURUNCULOSIS), 935.
Bone-marrow in leukæmia, 868.
Bones, diseases of, 899; in hereditary syphilis, 1109; in late syphilis, 1117; lesions of, in rickets, 254; microscopical changes of, in rickets, 255; syphilitic diseases of, 915; tuberculous diseases of, 900; etiology, 901; lesions, 901.
Bothriocephalus latus, 447.
Bottles, nursing, choice and care of, 204.
Bowels, hæmorrhages from (see HÆMORRHAGE, INTESTINAL); movements of, irregularity in times for, 424; training to control movements, 4.
Bow-legs in rickets, 263.
Bradycardia, 638.
Brain, diseases of, 747; abscess of, 780; atrophy and sclerosis of, 797; atrophy and sclerosis of, in acquired cerebral paralysis, 800; cysts of, in infantile cerebral paralysis, 797; malformations of, 747; tuberculosis of, 1085; tumour of, 783; water on the, 770; weight of, 699.
Bran bath, 57.
Breast, abscess of, in newly born, 116.
Breast-feeding, 171; schedule for, 172.
Breast milk (see MILK, WOMAN'S).
Breath, offensive, in ulcerative stomatitis, 284.
Breathing, noisy, with adenoids, 301; stridulous, in diseases of the larynx, 490, 493, 496; in retro-œsophageal abscess, 317.
Bright's disease (see NEPHRITIS), 660.
Bromides, elimination of, in milk, 139.
Bronchi, catarrhal spasm of, 525; diphtheria of, 1025; foreign bodies in, 508; lesions of, in acute broncho-pneumonia, 533; lymph nodes of, in tuberculosis, 1074, 1082; tube casts of, 521.
Bronchial glands (see also LYMPH NODES, BRONCHIAL), enlarged, cause of asthma, 524; in acute broncho-pneumonia, 540; reflex cough from, 523.
Bronchitis, acute catarrhal, 512; symptoms, 513; diagnosis, 515; treatment, 516; prophylaxis, 516; asthma following, 525; capillary (see BRONCHO-PNEUMONIA, ACUTE), 531, 542; attacks of asthma resembling, 524; chronic, 521; etiology, 521; symptoms, 521; diagnosis, 522; treatment, 522; chronic, bronchiectasis in, 522; chronic, in rickets, 258; diphtheritic, broncho-pneumonia in, 552; fibrinous, 520; treatment, 520; in pertussis, 1009; in typhoid fever, 1066; spasmodic (see ASTHMA), 523.
Bronchiectasis in chronic bronchitis, 522; in broncho-pneumonia, chronic, 582.
Broncho-pneumonia, acute, 531; bacteriology, 532; complications, 553; complicating influenza, 1126; diphtheria, 1028; measles, 985; pertussis, 1009; pseudo-diphtheria, 1060; rickets, 258; diagnosis, 554; etiology, 531; lesions, 533; associated, in the lung, 540; physical signs, chart of, 548; protracted or persistent form, 550; secondary pneumonia with measles, 552; ileo-colitis, 553; influenza, 553; pertussis, 551; diphtheria, 552; prognosis, 555; protracted cases, 550; symptoms, 541; temperature charts of, 545; terminations, 539; treatment, 557; prophylaxis, 557; summary of, 561.
Broncho-pneumonia, chronic, 582; lesions, 583; symptoms, 583; physical signs, 584; treatment, 585.
Broncho-pneumonia, tuberculous, 1077, 1090; rapid cases, 1091; protracted cases, 1102 (see also TUBERCULOUS PNEUMONIA).
Broths, directions for making, 164.
Bubo, with gonorrhœal urethritis, 686.
Buhl's disease, 93.
Buttermilk, 162.

Calamine lotion, 933.
Calculi, biliary, 464; renal, 677; pyelitis with, 678; vesical, 698.
Calomel fumigations, 497.
Calomel, how best given, 48.
Calories, required daily by healthy infants, 182; method of calculating, 182; value of different food stuffs in, 129.
Cancrum oris (see STOMATITIS, GANGRENOUS), 290.
Carbohydrates, function of, in diet, 127.
Carcinoma of brain, 783; of kidney, 671; of stomach, 350.

Cardiac cough, 523.

Carnrick's soluble food, 166.

Casein, 149, 185; in the fæces, 323; stools in difficult digestion of, 415.

Caseinogen, 149.

Casts in urine of chronic nephritis, 668.

Catarrh, Eustachian, in hypertrophy of tonsils, 312; fœtid (see RHINITIS, ATROPHIC), 485; gastric, 340; nasal acute, 478; prophylaxis, 480; chronic, 481; with adenoid growths, 301; foreign bodies in nose, 481; nasal polypi, 482; rhinitis, simple chronic, 482; hypertrophic, 484; atrophic, 485; syphilitic, 485; rhino-pharyngeal, with adenoids, 301.

Catheters, sizes required for infants, 642.

Cellulitis of abdominal wall with peritonitis, 466; of neck, in scarlet fever, 965.

Centrifugal machine, 135, 147.

Cephalhæmatoma, external, 97; internal, 97; symptoms, 98; diagnosis, 98; treatment, 99.

Cereals, 165; allowed from third to sixth year, 222.

Cerebellum, abscess of, 780; tumours, 783.

Cerebral paralysis, 795; from hæmorrhage, 107; etiology, 107; lesions, 108; symptoms, 109; prognosis, 110; treatment, 110.

Cerebro-spinal meningitis (see MENINGITIS, ACUTE CEREBRO-SPINAL), 754.

Cerebrum, abscess of, 780; tumour, 783.

Chest, circumference of, 20; development of, 24; "funnel" chest, 24; in rickets, 261; lateral depressions of, in adenoids, 301; lateral furrowing of, in rickets, 258.

Cheyne-Stokes respiration in cerebro-spinal meningitis, 762; in tuberculous meningitis, 773.

Chicken-pox (see VARICELLA), 996.

Chloral, dosage and administration, 53.

Chlorosis, 862; etiology, 862; lesions, 862; symptoms, 863; blood in, 863; prognosis, 863; diagnosis, 863; treatment, 867.

Cholera infantum, 364 (see also INTOXICATION, ACUTE GASTRO-ENTERIC), 381.

Chondro-dystrophy, 810.

Chorea, 721; acute endocarditis in, 624; diagnosis, 725; endocarditis in, 725; etiology, 721; following birth paralysis, 799; typhoid fever, 1067; habit, 727; heart murmurs in, 725; prognosis of, 726; hysterical, 735; with adenoids, 302; in rheumatism, 1144; pathology, 723; post-hemiplegic, 729; in cerebral palsy, 799; prognosis, 726; relation to rheumatism, 722; speech in, 725, 740; symptoms, 724; treatment, 726; urine in, 725.

Circulation, chan
fœtal, 606; in
Circulatory systen
Citrate of Soda, u
ing cases, 211.
Claw-hand, 843.
Cleft palate, 274.
Clothing at birth
night, 3; in sur
Club-foot with spi
Codeine, doses of,
Cod-liver oil as tc
Cold, as an antip
sponging, 49; p
gation of the
with adenoids,
Cold sores, 275.
Colic, habitual, f
203; intestinal,
Colitis, acute (se
385; amœbic,
membranous ga
Collapse, in ac
treatment of, 5
467; in append
gastritis, 341;
Collapse, pulmon
ACQUIRED), 588
Colles's law, 1108
Colon, abnormal
genital atresia
405; dilatation
follicular ulcer
of, 428; irrigati
enteric intoxica
indigestion, 41
mation of, 391
of, in chronic il
Colostrum, 130;
position of, 13
Coma, in tubercul
diabetes mellitu
Compression-myeli
Condensed milk,
composition of,
fants, 150; fres
Congenital, ichth
730; rickets, 25
culosis, 1072.
Conjunctiva, cat
measles, 982;
newly born, 10
Constipation, a c
causes of, in ric
dilatation of co
from, 454; ear
259; from defi
in appendicitis,
gestion, chroni
susception, 434.
Contractures, hyst
Convulsions, 701;
nosis, 704; pro

706; attributed to dentition, 280; causing death without other symptoms, 46; chloral in, 707; epileptic, 710; hysterical, 735; in acquired cerebral paralysis, 801; in cerebral hæmorrhages, 109; in congenital atelectasis, 75; in pertussis, 1010; in rickets, 267; morphine in, 707; in status lymphaticus, 881.

Cooley creamer, 152.

Cord, spinal, diseases of, 820; malformations of, 820; position of, 826; meningitis, 826; myelitis, 827; pressure-paralysis of, 829; tumours of, 839; weight of, 699.

Cord, umbilical, care of, 1; separation of, 2.

Cornea, ulcers of, in chronic ileo-colitis, 407.

Corpuscles of blood, 856.

Coryza, 478; early symptom of measles, 980; syphilitic, 485, 1113.

Cough, hysterical, 735; reflex, 522; from pharyngeal irritation, 522; elongated uvula, 522; from pharyngeal mucus, 522; from aural irritation, 522; from cardiac disease, 523; of puberty, 523; periodical, at night, 523; from Pott's disease, 523; symptoms, 523; diagnosis, 523; treatment, 523; spasmodic, in retro-œsophageal abscess, 317; in tuberculous bronchial glands, 1111; whooping (see PERTUSSIS), 1004.

Counter-irritants, 54.

Cow's milk (see MILK).

Cranio-tabes, early symptom in rickets, 259.

Cranium, syphilitic nodes on, 920.

Cream, 150; to secure different percentages of, 151, 152.

Cream-gauge, 135, 148.

Credé's method of preventing ophthalmia neonatorum, 1; treatment of ophthalmia, 88.

Cretinism, sporadic, 813.

Croup, bronchial, 520; catarrhal, 489; kettle, 60; membranous, 495; membranous, in scarlet fever, 965; spasmodic, 489; true, 495.

Cry, causes and varieties of, 34; in diseases, 34; in colic, 421; in retropharyngeal abscess, 296.

Cryptorchidism, 685.

Cups, dry, indications for, 55; wet, condemned, 55.

Curds and whey, 162.

Cyanosis, in acute broncho-pneumonia, 541, 543; in acute inanition, 228; in chronic cardiac disease, 630; in congenital atelectasis, 75; in congenital disease of heart, 613; in diphtheritic paralysis, 852; in malaria, 1133, 1136; of face, from pressure at root of lung, 1102.

Cyclic vomiting, 331.

Cyst, of brain, 783; of brain in infantile cerebral paralysis, 797; of intestinal mucosa, 405.

Cysticercus, 446.

Dactylitis, scrofulous, 913; syphilitic, 922; tuberculous, 913.

Deaf-mutism, 819; stigma of degeneration, 819.

Deafness following mumps, 1018; with adenoids, 301; with hypertrophy of tonsils, 312; sudden, in late syphilis, 1117.

Death, most frequent causes of, at different ages, 43; sudden, causes of, 44.

Deformities, hysterical, 734; in rickets, 259.

Degeneration, stigmata of, 818.

Deltoid, paralysis of, at birth, 111.

Dentition, 27; eruption of first teeth, 28; eruption of permanent teeth, 29; delayed, 28; before birth, 28; difficult, 279; in rickets, 266; in the etiology of diarrhœa, 356; often delayed in malnutrition, 232.

Dermatitis, exfoliative, of newly born, 922; gangrenous, 936.

Development, conditions interfering with, 30; muscular, 25; of body, 15.

Dew's method of inducing artificial respiration, 72.

Dextro-cardia, 613.

Diabetes insipidus, 652.

Diabetes mellitus, 1147.

Diacetonuria, 651.

Diagnosis, general considerations in, 31.

Diapers, 3.

Diaphragm, hernia through, 118.

Diarrhœa, general consideration of, 354; deaths from in New York in five years, 354; prevalence during summer, 355; impure milk as a cause, 356; observations of Rockefeller Institute on association of feeding impure milk and diarrhœal disease, 356 *et seq.*; different varieties of, 359; inflammatory (see ILEO-COLITIS, ACUTE), 385; in chronic intestinal indigestion, 414; in intestinal tuberculosis, 412; summer, 364; mycotic, 364.

Diastatic ferment of pancreas, 321; of bile, 321.

Diathesis, lymphatic, with adenoids, 300.

Diet (see also FEEDING), as cause of chronic constipation, 423; cause of rickets, 251; in acute gastro-enteric infection, 375; in acute gastric indigestion, 337; in chronic constipation, 424; in chronic gastric indigestion, 346; in eczema, 927; in intestinal indigestion, 418; in malnutrition, 235; in rickets, 271; in scurvy, 251; of nurse, effect on milk, 138.

Dietary of the infant, 129.

Digestion, gastric, 319; duration of, 320; in infancy, 318; intestinal, 321.

Digestive system, diseases of the, 274.

Digitalis, dosage for infant, 683.

Dilatation of the stomach, 347.

Diphtheria, 1019; bacillus (see BACILLUS OF DIPHTHERIA), 1020; broncho-pneumonia in, 552, 1028, 1036; blood in, 1029; cardiac failure in, 1035; cardiac thrombi in, 1028; catarrhal, 1023, 1030; complications and sequelæ, 1036; croupous bronchitis in, 520; diagnosis, 1038; bacteriological, 1041; clinical, 1038; from pseudo-diphtheria, 1040; disinfection after, 1047; distribution and mode of communication, 1020; entero-colitis in, 1038; etiology, 1020; immunization, 1045; incubation, 1022; lesions, 1022; membrane, 1023; membranous gastritis in, 339; proctitis in, 455; myocarditis in, 636, 1037; nasal syringing in, 1048; nephritis in, 1028, 1037; of œsophagus, 315; otitis in, 1036; paralysis after, 1034; paralysis in, 851; prognosis, 1043; prophylaxis, 1044; quarantine, 1044; septicæmia in, 1035; simulated after tonsillotomy, 314; symptoms, 1029; thrombosis in, 1037; toxins of, 1023; treatment, 1047; local, 1048; serum, 1049; of children exposed, 1045; of suspected cases, 1045; supplementary to antitoxin, 1056; false (see PSEUDO-DIPHTHERIA), 1056; laryngeal, 495, 1033; nasal, 1030, 1032; pseudo (see PSEUDO-DIPHTHERIA), 1020, 1056; scarlatinal (see PSEUDO-DIPHTHERIA), 1056; scarlatinal, 965; scarlatiniform erythema in, 971; streptococcus (see PSEUDO-DIPHTHERIA), 1056; tonsillar, 1030.

Diphtheria antitoxin, dosage of, 1050; immunizing dose of, 1046; influence on mortality of cities, 1054; local and general effects of, 1051; other treatment with, 1047, 1055; real and alleged dangers from, 1052; strength of, 1050; time of administration, 1050.

Diplegia, in birth paralysis, 798; in meningeal hæmorrhage, 109; spastic, 795.

Disease, peculiarities of, in children, 30; etiology, 30; symptomatology and diagnosis, 31; pathology, 40; prognosis, 42; prophylaxis, 46; therapeutics, 47.

Diverticulum, Meckel's, 114.

Dover's powder, dosage of, 53.

Dropsy (see also ŒDEMA); in acute diffuse nephritis, 662, 663; in chronic cardiac disease, 630; in chronic nephritis, 668; in newly born, 120; in tuberculosis, 1095; without renal disease, 682.

Drugs, administration of, 48; antipyretics, 50; elimination of, in breast milk, 139; well borne, 54; not well borne, 54.

Duct, omphalo-mesenteric, 114, 118.

Ductus arteriosus, closure of, 606; in fœtal circulation, 606; patent, 612; venosus, closure of, 606; in fœtal circulation, 606.

Duodenum, catarrhal inflammation of, 341; congenital atresia of, 117.

Dura mater, hæmatoma of, 751; thrombosis of the sinuses of, 778.

Dysentery (see ILEO-COLITIS, ACUTE), 385.

Dysphagia, hysterical, 735; in retropharyngeal abscess, 296.

Dyspnœa, evidence of, 36; from tuberculous bronchial lymph nodes, 1102; in acute catarrhal laryngitis, 493; in catarrhal spasm of larynx, 490; in membranous laryngitis, 496; in chronic cardiac disease, 629; in retro-pharyngeal abscess, 296; inspiratory, in retro-œsophageal abscess, 317; pressure of abscess on pneumogastric, 317; spasmodic, in asthma, 524.

Ear, anomalies of, as stigmata of degeneration, 818; hæmorrhage from, in newly born, 106; middle, inflammation of (see OTITIS), 943; in measles 987; in scarlet fever, 966.

Ears, development of hearing, 26.

Eberth's bacillus of typhoid fever, 1062.

Ecchymoses in purpura, 874; in scurvy, 245; in leukæmia, 869.

Echinococcus of liver, 464.

Eclampsia (see CONVULSIONS), 701.

Ecthyma gangrenosa, 936.

Ectocardia, 613.

Eczema, 926; etiology, 926; diagnosis, 930; treatment, 931; exacerbations during dentition, 280; intertrigo, 929; pustular, of scalp, 929; rubrum, 928; seborrhœic, 926, 929; simple chronic, 928.

Electrotherm, 12.

Emboli, infectious, in malignant endocarditis, 626.

Embolism, 640; in diphtheria, 1037.

Emphysema, 589; symptoms, 591; acute, in bronchitis of infants, 513; in acute broncho-pneumonia, 541; in pertussis, 1009.

Empyema, 596; lesions, 597; symptoms, 599; diagnosis, 600; treatment, 602; tuberculous, 1077; in acute broncho-pneumonia, 540.

Encephalocele, 747; symptoms, 748; treatment, 749.

Endarteritis, syphilitic, of brain, 1111; tuberculous, 770.

Endocarditis, acute simple, 622; lesions, 623; symptoms, 624; treatment, 625; acute simple, in chorea, 624; chronic (see also HEART, VALVULAR DISEASE), 627; fœtal, 610; in chorea, 725; in rheumatism, 1143; malignant, 626.

Enemata, 67; nutrient, 67; drugs by, 67; astringent, in chronic ileo-colitis, 408; in chronic constipation, 426; in colic, 422; ice-water in cholera infantum, 384; injuries to rectum from, 454.

Enteritis follicularis (see ILEO-COLITIS, ACUTE), 385.

Entero-colitis, in diphtheria, 1038 (see ILEO-COLITIS, ACUTE), 385.

Enuresis, 692; symptoms, 694; treatment, 694; stigma of degeneration, 819.

Ependymitis, acute, in hydrocephalus, 791; following spina bifida, 825.

Epidemic, hæmoglobinuria, 92; meningitis (see MENINGITIS, ACUTE).

Epidermis, exfoliation of, in congenital ichthyosis, 924; exfoliation of, in newly born, 922.

Epilepsy, 708; diagnosis, 713; hysterical, 735; idiopathic, 708; in acquired cerebral paralysis, 802; in birth paralysis, 798; insanity following, 806; intestinal putrefaction in, 709; Jacksonian, in cerebral tumour, 784; mental condition in, 712; pathology, 709; prognosis, 713; status epilepticus, 713; stigma of degeneration, 819; symptomatic, 712; symptoms, 710; treatment, 714.

Epiphyseal separation in acute arthritis, 899; in scurvy, 248; in syphilis, 915.

Epiphyses, enlargement of, in rickets, 263; in syphilis, 917, 921.

Epiphysitis, acute (see ARTHRITIS, ACUTE), 899; syphilitic, 905, 1115.

Epispadias, 684.

Epistaxis, 487; in anæmia, 861; in pertussis, 1008; in purpura, 874; in scurvy, 248.

Epitrochlear lymph nodes in syphilis, 1117.

Erb's paralysis, 112.

Erysipelas in newly born, 85.

Erythema, following diphtheria antitoxin, 1052; in influenza, 1128; intertrigo, 929; in intestinal indigestion, 417; in rheumatism, 1145; of the buttocks in marasmus, 241; scarlatiniform, causes, 971.

Erythroblasts, 863.

Estlander's operation, 605.

Eustachian tube in acute otitis, 943; inflammation of, in influenza, 1126; obstruction of, in hypertrophy of tonsils, 312.

Examination of sick child, 33; inspection, 34; measurements, 35; vital signs, 35; respiration, 36; temperature, 36; local examinations, 37–40.

Exercise, importance of, 7; caution regarding, in heart disease, 635; in anæmia, 868.

Expectorants in bronchitis, 518.

Exstrophy of bladder, 685.

Extubation, 503.

Eye, anomalies of, as stigmata of degeneration, 818; keratitis, interstitial, in syphilis, 1117; care of, at birth, 1, 3; diphtheritic paralysis of, 852; early use, 25; ectropion of, in congenital ichthyosis, 923; inflammation of, in newly born, 87; in measles, 988; nystagmus, 729.

Face, asymmetry of, as stigma of degeneration, 818; expression of, in disease, 34; cyanosis and œdema of, from pressure at root of lung, 1102.

Facial paralysis, at birth, 110; acquired, peripheral, 853; in otitis, 948.

Fæces, 323; of milk diet, 323; of mixed diet, 324; incontinence of, 457.

Fat, determination of, in milk, 135; in the fæces, 324; test for, 362; lack of, a cause of rickets, 251; lack of, causing constipation, 423; in woman's milk, 134; percentages of, in modification of cow's milk, 185, 190; symptoms from deficiency of, in food, 201, 202; symptoms from excess in food, 201, 203; function of, in diet, 126.

Fatty degeneration of the newly born, 93.

Fauces, syphilitic, ulceration of, 1111.

Feeding, artificial, fundamental principles of, 182; rules for, 196, 205; schedule for first year, 205; *versus* wet-nursing, 170; breast, schedule for, 172; other than milk, first year, 205; difficult cases, first year, 206; summary of infant feeding, 216; daily dietary from fourteen to eighteen months, 220; for healthy infants, second year, 219; difficult cases, second year, 221; from third to sixth year, 222; articles allowed, 222; articles forbidden, 223; dietary, from third to sixth years, 222; during acute illness, 224; in infants, 224; older children, 225; during very hot days, 373; by gavage, in acute illness, 225; nasal, 64; in acute gastro-enteric intoxication, 375; in acute intestinal indigestion, 363; methods of, in etiology of diarrhœa, 356; mixed, indications for, 181; simple rules in, 224.

Feet, anomalies of, as stigmata of degeneration, 818.

Feser's lactoscope, 147.

Fever, puerperal, of the child, 81; from insufficient nourishment, 174; inanition,

120; toxic, in intestinal indigestion, 417 (see also TEMPERATURE).
Finger (see DACTYLITIS).
Fingers, clubbing of, in chronic cardiac disease, 630; in congenital heart disease, 614.
Fissure of the anus, 454.
Fistula, congenital, of the neck, 314.
Flatulence, cause of colic, 420; in intestinal indigestion, 417.
Fœtal circulation, 606; endocarditis, 610.
Fœtus, evidences of syphilis in, 1112.
Follicles, solitary (see LYMPH NODULES); solitary, of intestine, often enlarged in marasmus, 239.
Follicular ulceration of intestine, 389.
Fomentations, hot, 55.
Fontanel, bulging of, in cerebro-spinal meningitis, 762; bulging of, in meningeal hæmorrhage, 109; bulging of, in tuberculous meningitis, 773; in hydrocephalus, 792; closure of, 22; in cretinism, 815; in rickets, 258.
Food, constituents, 125; proteids, 125; fats, 126; carbohydrates, 127; mineral salts, 128; water, 128; farinaceous, a cause of eczema, 927; in chronic indigestion, 346; second year, 219; improper in etiology of diarrhœa, 356; regurgitation of, causes and treatment, 201.
Food-fistula between œsophagus and larynx, 316.
Food-diseases, 244.
Foods, infant, 166; milk, 166; malted, 166; farinaceous, 166; proprietary, dangers of, 124; cause of rickets, 251; cause of scurvy, 245; uses of, in chronic constipation, 425.
Foramen ovale, closure of, 607; function of, in fœtal life, 606; patent, 612.
Fractures, green-stick, in rickets, 255, 263.
Franco-Swiss food, 166.
Freeman's pasteurizer, 154.
Friedländer's bacillus in acute broncho-pneumonia, 532.
Friedreich's ataxia, 841.
Fruit, best time for giving, 220; during second year, 220; allowed during third to sixth year, 223; forbidden during third to sixth year, 223.
Furunculosis, 935; in diabetes mellitus, 1148.

Gangrene, of the face, 290; of intestine, in intussusception, 431; of lung, 586; in acute broncho-pneumonia, 541; in lobar pneumonia, 564; in scarlet fever, 970; in measles, 987.
Gastralgia, 334; in malaria, 1135; in spinal caries, 904.
Gastritis, acute, 337; etiology, 337; lesions, 338; symptoms, 340; treatment, 341; chronic, 343; ulcers in, 349; toxic (see GASTRITIS, CORROSIVE), 339.
Gastro-duodenitis, 341.
Gastro-enteric infection or intoxication (see INTOXICATION, ACUTE GASTRO-ENTERIC), 364.
Gastro-enteritis (see INTOXICATION, ACUTE GASTRO-ENTERIC), 364; in newly born, 84.
Gavage, 64; in acute illness, 225; in acute inanition, 230; in diphtheria, 1047; in premature infants, 14; in thrush, 289.
Genital irritation, 697.
Genital organs, diseases of, 683; anomalies of, as stigmata of degeneration, 818; care of, in newly born, 4; malformations of, 683; female, gangrene of, 290; female, diseases of, 688; hæmorrhage from, in newly born, 106; males, diseases of, 686.
Gingivitis, hæmorrhagic, in scurvy, 246, 247.
Glands, bronchial (see LYMPH NODES, BRONCHIAL).
Glands, lymphatic (see LYMPH NODES), 877.
Glioma of brain, 783; of spinal cord, 840.
Glio-sarcoma of brain, 783.
Glossitis, 277.
Glottis, œdema of the, 505; spasm of, idiopathic, 719.
Glycosuria, 647.
Gonococcus, differentiation of, 690; in gonorrhœal stomatitis, 289; in specific urethritis, 686; in vulvo-vaginitis, 690.
Gout, eczema in children, 927; uric-acid deposits in urine, 650.
Granuloma of umbilicus, 113.
Grippe (see INFLUENZA), 1123.
Growing pains, rheumatic, 1143.
Growth, conditions interfering with, 30; of body, 15; extremities, 21; trunk, 21.
Gumma, syphilitic (see SYPHILIS LESIONS), 1109; in syphilitic bone disease, 919; of brain, 783.
Gums, abscess of, 279; bleeding in ulcerative stomatitis, 285; inspection of, 38; lancing, 281; spongy and bleeding, in scurvy, 246, 248; in ulcerative stomatitis, 285.

Habit-chorea, 727.
Habit-spasm, 727.
Habits, injurious, 743.
Hæmatemesis, 350.
Hæmatoma of the sterno-mastoid, 96.
Hæmatocytozoön malariæ, 1131.
Hæmaturia, 646; in newly born, 107; in purpura, 873; in pyelitis, 676; in scurvy, 248; in tumours of kidney, 672.
Hæmoglobin, 856.

Hæmoglobinuria, 647; epidemic, 92; paroxysmal, 647.
Hæmophilia, 870.
Hæmoptysis in tuberculosis, 1095.
Hæmorrhage, from stomach, 350; in hæmophilia, 871; intra-alveolar, in acute broncho-pneumonia, 536; internal, causing sudden death, 44; intestinal, from tuberculous ulcer, 412; in typhoid fever, 1066; meningeal, causing birth paralysis, 796; in acquired cerebral paralysis, 800; in acute broncho-pneumonia, 553; in convulsions, 704; meningeal, in pertussis, 1009; meningeal, in purpura, 873; nasal, in diphtheria, 1037; pulmonary, in cardiac cases, 630; rectal, from ulcer, 456; in leukæmia, 869; in measles, 988; in pertussis, 1008; in pernicious anæmia, 866; in purpura, 874; in the newly born, 95; hæmatoma of the sterno-mastoid, 96; cephalhæmatoma, 97; visceral, 99; in scurvy, 245, 249; subperiosteal, in scurvy, 245; in syphilis, 1115.
Hæmorrhagic disease of the newly born, 100.
Hæmorrhoids, 457; in chronic constipation, 423.
Hair, anomalies, stigmata of degeneration, 818.
Hand, progressive muscular atrophy of, 843.
Hands, anomalies, stigmata of degeneration, 818.
Harelip, 274.
Hawley's food, 166.
Hay fever, 525.
Head, circumference of, 20; closure of sutures, 22; closure of fontanels, 22; shape of, 23; in rickets, 259; examination of, 37; hydrocephalic, characteristics of, 792; rotary and nodding spasm of, 729; sweating of, in rickets, 259.
Headache, frequent, with adenoids, 302; varieties, 737; diagnosis, 738; treatment, 738.
Hearing, when developed, 26.
Heart, diseases of, 606; aneurism of, 637; aortic disease, congenital, 612; area of absolute cardiac dulness, 609; of relative dulness, 608; auscultation of, 39; diphtheritic paralysis of, 852; examination of, 608; hypertrophy of, in congenital diseases, 615; hypertrophy of, in valvular diseases, 628; in measles, 988; in scarlet fever, 969; malformations of, 610; peculiarities of, in early life, 606; persistent fœtal conditions, 610; position of apex beat, 608; in infancy, 608; size and growth of, 607; sounds of reduplication, 610; sudden failure of, in diphtheria, 1035; thrombus of, ante-mortem, 640; transposition of, 613; congenital anomalies of, 610; functional disorders of, 638; murmurs of, 631; anæmic, 637; in congenital diseases, 614; in chorea, 725; in marasmus, 241; valves, aortic insufficiency, 633; aortic stenosis, 632; mitral insufficiency, 631; mitral stenosis, 632; congenital absence of valves, 613; tricuspid insufficiency, 633; valvular disease of (see also ENDOCARDITIS), 622; chronic valvular disease of, 627; ventricle, left, signs of dilatation, 632; signs of hypertrophy, 631; right, signs of hypertrophy, 615.
Hectic fever in tuberculosis, 1094.
Height, 21; from birth to sixteenth year, 20.
Hemianopsia in cerebral tumour, 786.
Hemichorea, 724.
Hemiplegia in acquired cerebral paralysis, 801; in birth paralysis, 797; in meningeal hæmorrhage, 109; in cerebral tumour, 786; spastic, 795.
Hermaphroditism, false, 684.
Hernia, cerebri, 748; diaphragmatic, 118; umbilical, 115.
Herpes, labialis, 275.
Herpetic stomatitis, 282.
Hiccough, 730; in acute peritonitis, 467; in appendicitis, 442; in hysteria, 735.
Hip, articular ostitis of, 907.
Hip-joint disease (see HIP, ARTICULAR OSTITIS OF), 907.
History-taking, 32.
Hives (see URTICARIA), 938.
Hoarseness with adenoids, 302; in catarrhal spasm of larynx, 490; in syphilis, 1114.
Hodgkin's disease, 895.
Home modification of milk (see MILK, MODIFICATION AT HOME), 191.
Horlick's food, 166.
Hubbell's prepared wheat, 166.
Hutchinson's teeth in late hereditary syphilis, 1116.
Hydatids of liver, 464.
Hydrencephalocele, 747.
Hydrocele, 687.
Hydrocephalus, 789; in chronic basilar meningitis, 776; with spina bifida, 791, 822; acute (see MENINGITIS, TUBERCULOUS), 770–789; chronic external, 789; internal, 789; shape of head, 792; congenital, 750; intra-uterine, 748; syphilitic, 1111.
Hydronephrosis, 655; traumatic, 679; with malformations of kidney, 658; with renal calculi, 678.
Hydromyelus, 840.
Hygiene of infancy, 1.
Hyperæsthesia, general, in cerebro-spinal meningitis, 760; in infantile spinal

paralysis, 833; hysterical, 734; in multiple neuritis, 849; in scurvy, 247; in spinal meningitis, 827.
Hypermetropia, stigma of degeneration, 819.
Hypertrophy, of the tonsils, 312; muscular pseudo-, 844.
Hypodermic medication, 67.
Hypospadias, 684.
Hysteria, 733; etiology, 733; symptoms, 734; diagnosis, 736; prognosis, 736; treatment, 736.
Hystero-epilepsy, 735.

Ice, bag, 56; cap, 49, 56; coil, 56.
Ichthyosis, congenital, 923.
Icterus, 459; in epidemic hæmoglobinuria, 92; in gastro-duodenitis, 342; varieties in newly born, 77; in malformation of the bile ducts, 78; physiological or idiopathic, 78.
Idiocy, 804; Mongolian, 806; amaurotic family, 807; cretinoid, 807.
Ileo-colitis, acute, 385; etiology, 385; lesions, 386; in catarrhal, 387; in follicular, 389; in membranous, 391; associated lesions, 393; symptoms, catarrhal form, 394; with follicular ulceration, 396; membranous form, 398; diagnosis, 400; prognosis, 401; treatment, 401; broncho-pneumonia complicating, 553; following pertussis, 1010; in influenza, 1127; in measles, 987.
Ileo-colitis, chronic, 404; lesions, 404; symptoms, 406; diagnosis, 407; prognosis, 408; treatment, 408.
Ileum, congenital atresia of, 117.
Imbecility, 804.
Imperial granum, 166.
Impetigo, bullous, in newly born, 94; simple, 929; contagiosa, 937.
Inanition, acute, 227.
Inanition fever, 120.
Incubator, 12; in marasmus, 243.
Indican, in urine of chronic constipation, 424; of intestinal indigestion, 418; test for, in urine, 650.
Indicanuria, 650.
Indigestion, acute gastric, 335; etiology, 335; symptoms, 336; diagnosis from gastritis, 336; treatment, 336; vomiting in, 329; chronic gastric, 343; etiology, 343; lesions, 343; symptoms, in infants, 344; in older children, 345; prognosis, 345; treatment in infants, 345; with dilatation, 348; acute intestinal, 361; etiology, 361; symptoms, 361; diagnosis, 362; prognosis, 363; treatment, 363.
Indigestion, chronic intestinal, 413; in young infants, 413; lesions, 414; symptoms, 414; diagnosis, 415; prognosis, 416; treatment, 416; in older children, 416; symptoms, 416; prognosis, 418; treatment, 418.
Infant, care of newly born, 1; when premature or delicate, 10.
Infant feeding, 168.
Infant foods, 166.
Infarctions, uric acid, in kidney, 658.
Infectious diseases, the specific, 952.
Influenza, 1123; etiology, 1123; lesions, 1124; symptoms, 1124; with broncho-pulmonary complications, 1126; with gastro-enteric complications, 1127; in very young infants, 1127; protracted cases, 1127; complications and sequelæ, 1128; diagnosis, 1128; prognosis, 1129; treatment, 1130; broncho-pneumonia, 553, 1126; epidemic, acute otitis in, 943; scarlatiniform erythema in, 971.
Inhalations, 60; in bronchitis, 518.
Inheritance a factor in disease, 30.
Injections, rectal, in ileo-colitis, 403; in intussusception, 437; subcutaneous, of saline solution in cholera infantum, 384.
Insanity, 816; etiology, 817; symptoms, 817; prognosis, 818; following typhoid fever, 1067.
Inspection of sick child, 34.
Intermittent fever, malarial, 1132.
Intertrigo, 929; treatment, 934.
Intestinal obstruction in newly born, 117; acute, from intussusception, 428.
Intestines, diseases of, 352; amyloid degeneration of, 410; bacteria of, 322; digestion in, 321; hæmorrhage from, in newly born, 105; in typhoid, 1066; in tuberculosis, 412; length, 321; malformations of, 352; obstruction, congenital of, 117; obstruction by omphalo-mesenteric duct, 118; perforation of, in tuberculous peritonitis, 473; in tuberculous ulcers, 412; in typhoid fever, 1066; tuberculosis of, 410, 1086; etiology, 411; lesions, 411; symptoms, 412; treatment, 413.
Intoxication, acute gastro-enteric, 364; etiology, 364; lesions, 366; symptoms, simple form, 368; relapses, 370; cases without diarrhœa, 371; diagnosis, 372; prognosis, 372; prophylaxis, 373; treatment, hygienic, 374; dietetic, 375; medicinal and mechanical, 377; cholera infantum, 381; etiology, 381; symptoms, 381; prognosis, 383; treatment, 383.
Intubation, 498; advantages over tracheotomy, 503; retained intubation tubes—prolonged intubation, 504; in acute catarrhal laryngitis, 495; in syphilitic laryngitis, 508; in pertussis, 1014.
Intubation set, O'Dwyer's, 498.
Intussusception, 428; etiology, 429; lesions and mechanism, 430; symptoms, 431; diagnosis, 435; prognosis, 435;

treatment, 436; laparotomy, 437; in the dying, 429.
Invagination of intestine in intussusception, 431.
Iodides, elimination of, in milk, 139.
Iritis, syphilitic, 1111.
Iron, tonic preparations of, 52.
Irrigation, intestinal, in chronic indigestion, 419; as antipyretic, 50; of the colon, method of, 65.
Ischio-rectal abscess, 457.
Italians, rickets in, 252.

Jacket, oil-silk, 61.
Jaffé's test for indican, 650.
Jaundice (see also ICTERUS), 459; catarrhal, 341.
Jaw, necrosis of, from alveolar abscess, 279; in gangrenous stomatitis, 291; in ulcerative stomatitis, 284.
Jejunum, congenital atresia of, 117.
Joints, diseases of, 899; hysterical affections of, 734; in scarlet fever, 968; rheumatism of, 1142; suppuration of, in newly born, 84; swelling of, in scurvy, 248; ecchymoses about, in scurvy, 247; tuberculous diseases of, 900.
Junket, 162.

Keller's malt soup (see MALT SOUP).
Kemp's tube, 65.
Kernig's sign, 760.
Keratitis, interstitial, in late syphilis, 1111, 1117.
Keratoma, diffuse, 923.
Kidney, diseases of, 654; acute congestion of, 659; acute degeneration of, 660; benign tumours of, 674; calculi in, 675; chronic congestion of, 659; contracted (see NEPHRITIS, CHRONIC), 668; cystic degeneration of, 655; floating, 658; granular (see NEPHRITIS, CHRONIC), 668; hæmorrhage from, in newly born, 106; in scurvy, 246, 248; horseshoe, 655; hydronephrosis, 655; traumatic, 679; malformations and malpositions of, 654; malignant tumours of, 671; nephritis, acute diffuse, 660; acute exudative, 660; chronic, 667; perinephritis, 679; pyelitis, 674; pyelo-nephritis, 656; pyonephrosis, 675; single, 655; tuberculosis of, 670, 1086; uric acid infarction, 658; waxy, 667; in diphtheria, 1028; in scarlet fever, 967.
Klebs-Loeffler bacillus (see BACILLUS OF DIPHTHERIA), 1020, 1041.
Knee, articular ostitis of, 911; symptoms, 912; treatment, 913; subluxation of, in infantile spinal paralysis, 835; swelling of, in scurvy, 247; white swelling of (see KNEE, ARTICULAR OSTITIS).
Knee-jerk, in acquired cerebral paralysis, 802; in birth paralysis, 799; lost, in diphtheritic paralysis, 852; in infantile spinal paralysis, 835; in multiple neuritis, 849.
Knee-joint disease (see KNEE, ARTICULAR OSTITIS).
Knock-knee in rickets, 263.
Koplik's sign, in measles, 989.
Kumyss, 160.
Kyphosis in rickets, 261; treatment, 271; in spinal caries, 902.

Lactalbumin, 133, 149, 153.
Lactated food, 166.
Lactation, care of breasts during, 171.
Lactoglobulin, 133.
Lactometer, author's, 135.
Lactoscope, Feser's, 147.
Landry's paralysis, 842.
Laparotomy, in chronic peritonitis, with ascites, 470; in intussusception, 437; in tuberculous peritonitis, 475.
Laryngismus stridulus, 719; in rickets, 267; with tetany, 717.
Laryngitis, acute catarrhal, 492; catarrhal, in measles, 986; chronic, 506; with adenoid vegetations of pharynx, 506; tuberculous, 506; syphilitic, 507; with new growths of larynx, 508; membranous, 495; antitoxin, 497; intubation, 498; spasmodic, 489; submucous (œdema of glottis), 505.
Laryngotomy for foreign body in larynx, 509.
Larynx, diseases of, 489; catarrhal spasm of, 489; from long uvula, 295; with adenoids, 303; diphtheria of, 495, 1033; foreign bodies in, 508; intubation of, 498; in measles, 986; in pseudo-diphtheria, 1057, 1059; new growths of, 508; stenosis of, simulated by tuberculous glands, 1103; syphilis of, 507, 508, 1110; tuberculosis of, 506.
Lassar's paste, 933.
Lavage (see STOMACH WASHING).
Leptomeningitis, acute (see MENINGITIS), 754.
Leukæmia, 868.
Leucocytosis, definition, 858; diagnostic value, 859; prognostic value, 860; in diphtheria, 1029; in acute meningitis, 762.
Lewi's method for examination of woman's milk, 135.
Lichen urticatus (see URTICARIA), 938; tropicus, 925.
Liebig's food, 166.
Limewater, in modification of cow's milk, 187.
Lip, eczema of, 276; perlèche, 276; diseases of, 275; herpes of, 275; malformations of, 274.

Lisping, 739.
Lithuria, 649.
Liver, diseases of, 458; abscess of, 460; acute yellow atrophy of, 460; amyloid degeneration of, 462; biliary calculi, 464; cirrhosis of, 461; congestion of, 460; enlarged, in congestion, 460; in abscess, 461; in cirrhosis (early), 462; in chronic cardiac disease, 630; fatty, 463; fatty, in eczematous children, 927; in marasmus, 239; functional disorders of, 459; hydatids of, 464; in rickets, 267; in syphilis, 1109, 1118; in tuberculosis, 1085; lardaceous, 462; malformations and malpositions of, 459; size and position of, 39, 458; tuberculosis of, 1095; waxy, 462; weight of, in infancy, 458.
Loeffler's bacillus (see Bacillus of Diphtheria), 1020.
Lumbar puncture, 764; tubercle bacilli in fluid, 775.
Lung, diseases of, 509; abscess of, 585; abscesses of, in acute broncho-pneumonia, 541; acute congestion of, in malaria, 1136; calcareous nodules in, 1081; caseous degeneration of, 1078; collapse of, from compression, 588; from obstruction, 588; in acute broncho-pneumonia, 534; congenital atelectasis of, 74; emphysema of, 589; acute, in bronchitis of infants, 513; gangrene of, 586; gangrene of, in lobar pneumonia, 564; hæmorrhages into, in newly born, 99; inflation of, 73; miliary tuberculosis of, 1077; peculiarities in disease, 512; in infancy and early childhood, 509; physical examination of, 510; structure of, 510.
Lymph nodes, diseases of, 877; calcareous cervical, 890; bronchial, 1082; early infection in tuberculosis, 1074; enlarged in eczema, 929; in Hodgkin's disease, 895; in malnutrition, 232; frequency of disease of, 41; inflammation of (see Adenitis), 883; in late hereditary syphilis, 1117; in measles, 988; in pseudo-diphtheria, 1058; in scarlet fever, 965; simple hyperplasia of, 886; situation and drainage areas of the groups of head and neck, 883; syphilitic disease of, 887; tuberculous bronchial, 1001; lesions, 1074, 1082; symptoms, 1001; cervical, tuberculosis of, 888; mesenteric, 410, 1075; in diphtheria, 1027; in rickets, 266; in tonsillitis, 310; epitrochlear, in syphilis, 1117; mesenteric, often enlarged, in marasmus, 239; in typhoid fever, 1063; tuberculosis of, 888; retro-pharyngeal, abscess of, 295.
Lymph nodules of intestines, ulceration of, 389.
Lymphadenoma of stomach, 350.
Lymphangioma of tongue, 275.
Lymphatism (see Status Lymphaticus), 879.
Lymphocytes, 857.

Magendie, foramen of, in hydrocephalus, 789.
Malaria, 1131; etiology, 1131; lesions, 1132; symptoms, 1132; diagnosis, 1137; prognosis, 1138; prophylaxis, 1138; treatment, 1138; quinine, methods of administration, 1138; acute pulmonary congestion in, 1136; contracted *in utero*, 1131; spleen in, 898.
Malformations as cause of sudden death, 44.
Malnutrition, 230; etiology, 231; symptoms in infants, 231; symptoms in older children, 233; diagnosis, 234; prognosis, 234; treatment in infancy, 235; treatment in older children, 237.
Malnutrition, marasmus, 238.
Malted milk, 166.
Malt extracts, use of, in diet of nurse, 138.
Maltose, substitute for milk sugar, 127.
Malt soup of Keller, use of, with difficult feeding cases, 214, 218.
Mania, 817; acute, following typhoid fever, 1067.
Marasmus, 238; etiology, 238; lesions, 239; symptoms, 241; complications, 241; diagnosis, 242; from tuberculosis, 242, 1088; prognosis, 242; treatment, 243; fatty liver in, 464; general œdema in, 682; tuberculosis resembling, 1087.
Massage, 68; in chronic constipation, 425; in malnutrition, 236; of breasts to increase milk, 176.
Mastitis in the newly born, 116.
Mastoid disease, cerebral abscess following, 780; in acute otitis, 947.
Masturbation, 744; a cause of epilepsy, 709; of insanity, 817; of functional disorder of heart, 638.
Matzoon, 161.
Measles, 977; broncho-pneumonia complicating, 552; complications and sequelæ, 985; desquamation, 982; diagnosis, 989; digestive system, 987; diphtheria in, 987; duration of infective period, 978; ears, 944, 987; eruption, 981; etiology, 977; eyes, 988; gangrenous dermatitis in, 936; German (see Rubella), 993; hæmorrhage in, 988; hæmorrhagic, 981; heart in, 988; ileo-colitis, 984; incubation, 978; invasion, 980; kidneys in, 988; larynx in, 986; lesions, 979; lungs, 985; lymph nodes, 988; mode of infection, 979; mortality, 989; nervous system in, 984; other infectious diseases in, 988; otitis, 984; predisposition,

977; prognosis, 989; prophylaxis, 990: pseudo-diphtheria in, 1059; quarantine in, 991; skin in, 988; symptoms, 980; throat, 986; treatment, 991: tuberculosis following, 988.
Meats, allowed from third to sixth years, 222; forbidden from third to sixth years, 223.
Meckel's diverticulum, 114, 353.
Meconium, composition of, 323.
Mediastinum, anterior, abscess of, 1103; tumour of, due to tuberculous lymph nodes, 1103.
Mediastinitis, 618.
Melancholia, 817.
Melæna, 105.
Mellin's food, 166, 167.
Membrane, in diphtheria, 1023; in pseudo-diphtheria, 1057.
Meningeal hæmorrhage, 1, 751, 796.
Meninges, diseases of, 747.
Meningitis, acute, 754; cerebro-spinal, 754; course, duration and termination, 763; diagnosis, 764; etiology, 754; from acute otitis, 948; in newly born, 84; in typhoid fever, 1067; purulent, in acute broncho-pneumonia, 553; lesions, 755; leucocytosis in, 762; lumbar puncture in, 764; symptoms, 759; diagnosis, 764; prognosis, 763; treatment, 766.
Meningitis, simple acute, 767.
Meningitis, chronic basilar, 775; spinal, 826; syphilitic, 1111.
Meningitis, tuberculous, 770; lesions, 770; etiology, 771; symptoms, 771; duration, 774; diagnosis, 774; prognosis, 775; treatment, 775; lumbar puncture in, 775.
Meningocele of brain, 747; of cord, 821.
Meningo-encephalitis, 796.
Meningo-myelocele, 821.
Menstruation, effect on nursing, 137.
Mental defects, 804; classification, 804; diagnosis, 808; treatment, 809.
Mercury, elimination of, in milk, 139; ulcerative stomatitis from, 284; in syphilis, 1122.
Microcephalus, 750.
Micro-organisms in cow's milk, 141; see also BACTERIA.
Micturition, difficult or painful, 697; frequency of, 643.
Miliaria, 924; papulosa, 925; treatment, 925; rubra, 924.
Milk, cow's, 140; average percentages of, 185; bacteriological standard for, 144; handling and transportation of, 145, 146; certified, 146, 147; composition of, 146; average percentages in, from different breeds, 146, 147; examination of, 147; coagulation of, in stomach, 320; cream, 150; contaminated, cause of diarrhœa, 356; differences from human milk, 149; diphtheria bacilli in, 142, 154; essentials of, for infant feeding, 141; formulæ from diluting, 192 *et seq.*; micro-organisms in, 141; modification of, percentage or American method of, 184, 216; at home, 191; top-milk, 151; formulæ from top-milk, 192 *et seq.*; schedule of percentages for first year, 190; schedule showing quantities and intervals of feeding, 205; rules for varying percentages, 197; modifications required by particular symptoms, 200; in difficult cases, 209; in summer diarrhœa, 375; in acute indigestion, 363; in chronic constipation, 424; pasteurization of, 154; proteids of, 125; sterilization of, at 167° F., 154; sterilization of, at 212° F., 153; sterilized, scurvy ascribed to, 245; tubercle bacilli in, 1073; typhoid contamination of, 142; condensed (see CONDENSED MILK), 159; peptonized, 158; peptonized, use of, 211; dangers from long use of, 211.
Milk-laboratories, 188.
Milk-sugar, uses of, as food, 127; solution, how to prepare, 186.
Milk, woman's, 129; physical characters of, 129; colostrum of, 130; daily quantity of, 131; average quantity at one nursing, 132; composition of, 133; proteids, 125, 133, 149; fat, 134; sugar, 134; salts, 134; reaction, 134; specific gravity, 135, 136; average percentages of, 185; conditions affecting composition of, 137; menstruation, 137; diet, 138; drugs, 139; pregnancy, 139; elimination of antitoxin and other protective substances, 139; nervous impressions, 140; examination of, 134; variations in quality, 137; apparatus for examining, 136; flow established, 129; how to modify quantity and quality, 176, 177; indications of scanty supply, 174.
Modified milk, from milk laboratory, 188; schedule for feeding from birth, 190; made at home (see MILK, MODIFICATION OF, AT HOME).
Mongolian idiocy, 806.
Monoplegia, in birth paralysis, 797; in cerebral hæmorrhage, 109; in cerebral tumour, 786.
Morbilli (see MEASLES), 977.
Morbus coxarius (see HIP, ARTICULAR OSTITIS OF), 907.
Morbus maculosus Werlhofii (see PURPURA), 871.
Morphine, dosage of, 58, 468; dosage in convulsions, 707; hypodermically in cholera infantum, 384; in gastro-intestinal intoxication, 379.
Mortality at different ages, 42, 43.

Morton's fluid, 826.
Mouth, diseases of (see also STOMATITIS), 274, 281; applications to, 289; care of, at birth, 1, 3; hæmorrhage from, in newly born, 105; hæmorrhages from, in scurvy, 248; malformations of, 274; mucous patches, in syphilis, 1114; syphilis of, 289; "tapir," 846; syringing of, 59.
Mouth-breathing, with hypertrophy of tonsils, 312; with adenoids, 301; with retro-pharyngeal abscess, 296.
Mucous membranes, frequency of involvement in childhood, 40; in rickets, 266.
Mucous patches, syphilitic, 1114.
Mumps, 1016; complications and sequelæ, 1018; diagnosis, 1019; etiology, 1016; incubation, 1017; pathology and lesions, 1016; prognosis, 1019; quarantine in, 1019; symptoms, 1017; treatment, 1019.
Murmurs, cardiac (see HEART MURMURS).
Muscles, atrophy of, 842; in infantile spinal paralysis, 834; in multiple neuritis, 849; in myelitis, 828; contractures of, hysterical, 735; in acquired cerebral paralysis, 802; in birth paralysis, 799; development of, 25; flabbiness of, in rickets, 264; rigidity of, in birth paralysis, 798; spasm of, about rheumatic joint, 1142.
Muscular atony, as cause of chronic constipation, 423.
Muscular atrophies, different types of, 842.
Muscular pseudo-hypertrophy, 844.
Mustard bath, 56; paste, 54; pack, 54.
Myelitis, 827; symptoms, 828; treatment, 828; compression, from Pott's disease, 829; diffuse, 828; transverse, 828.
Myelocytes in leukæmia, 869; in diphtheria, 1029.
Myocarditis, 636; aneurism in, 637; toxic, in diphtheria, 852, 1028; in scarlet fever, 969; in syphilis, 1111.
Myopia, stigma of degeneration, 819.
Myotonia, congenital, 730.

Nail-biting, 747.
Nails in syphilis, 1115.
Neck, cellulitis of, in scarlatina, 965; congenital fistula of, 314; wry (see TORTICOLLIS).
Necrosis of bone in syphilis, 916, 918.
Negroes, rickets in, 252.
Nematodes (see WORMS, INTESTINAL), 448.
Nephritis, acute diffuse, 660; etiology, 660; lesions, 661; symptoms, 662; prognosis, 664; treatment, 665; in broncho-pneumonia, 554; acute parenchymatous type, 662.
Nephritis, chronic, 667; etiology, 667; lesions, 667; symptoms, 668; of the parenchymatous type, 668; of the interstitial type, 669; prognosis, 669; diagnosis, 669; treatment, 670; chronic diffuse, with hydronephrosis, 656; chronic interstitial, syphilitic, 1111; in diphtheria, 1028; interstitial (see NEPHRITIS, CHRONIC), 669; post-scarlatinal, 967.
Nerves, peripheral, diseases of, 846.
Nervous impressions, effect of, on nursing, 140.
Nervous system, diseases of, 699; diseases of, functional, 701; general hygiene of, 5; peculiarities of, in childhood, 700.
Nestlé's food, 166, 167.
Neuritis, multiple, 846; after diphtheria, 851; in malaria, 1136; optic, in acute meningitis, 761; in cerebral tumour, 785; with cerebral abscess, 782.
Newly born, diseases of, 69; acute infectious diseases of, 81; acute pyogenic diseases of, 81; atelectasis, congenital, 74; asphyxia of, 69; blood in, peculiarities of, 856; care of, 1; diseases or accidents at birth, 30; dermatitis exfoliativa in, 922; facial paralysis in, 110; fatty degeneration of, 93; hæmorrhages in, 95; hæmorrhagic disease of, 100; hyperpyrexia in, 120; inanition fever in, 120; icterus in, 77; infection of, 31; malformations of, 30; mastitis in, 116; ophthalmia of, 87; pemphigus in, 94; peritonitis in, 465; sclerema in, 118; skin of, 922; ulcer of stomach in, 349.
Nightmare, 742.
Night-terrors, 742.
Nipples, care of, during lactation, 171; fissure of, hæmatemesis from, 351; rubber, choice of, 204; care of, 204.
Nodding spasm of head, 729.
Nodes, lymph (see LYMPH NODES), 877.
Nodules, subcutaneous tendinous, in rheumatism, 1144.
Noma of face (see STOMATITIS, GANGRENOUS), 290; of vulva, 692.
Nose, diseases of, 478; deformities of, in hereditary syphilis, 485; difficulty in blowing, with adenoids, 301; diphtheria of, 1025; discharge from, with adenoids, 301; foreign bodies in, 481; hæmorrhage in, 487; in newly born, 105; in scurvy, 248; in hereditary syphilis, 485, 1010; in late syphilis, 1118; polypi in, 482; pseudo-diphtheria of, 1057; sprays for, 57; syringing, 58.
Nurse, effect of diet on milk of, 138; requisite qualities in, 10; wet (see WET-NURSE).
Nursery, temperature, ventilation, 9.
Nursing, at night, 172; when discontinued, 172; during acute illness, 224;

during first days of life, 171; hours for, in newly born, 172; during illness, 181; importance of good habits, 172; unsuccessful, symptoms of, 172; maternal, contra-indications for, 169.
Nursing-bottles, choice of, 204; care of, 204.
Nutrient, enemata, 67.
Nutrition, derangements of, 226; acute inanition, 227; malnutrition, 230; marasmus, 238; faulty, diseases due to, 244; importance in pædiatrics, 124.
Nystagmus, 729; in cerebral hæmorrhage, 110; in hydrocephalus, 794; in tuberculous meningitis, 773; stigma of degeneration, 819; with tumour of crura cerebri, 786.

Oatmeal water, 165.
O'Dwyer's intubation set, 498.
Œdema, in acute diffuse nephritis, 662, 663; in anæmia, 861; in chronic nephritis, 668; in cardiac disease, 630; in delicate infants, 120; in leukæmia, 870; of face from pressure at root of lung, 1102; general, in marasmus, 241; not from renal disease, 682.
Œdema glottidis, 505; rare in acute catarrhal laryngitis, 492; in corrosive œsophagitis, 316; in quinsy, 311.
Œsophagitis, acute, 315; catarrhal, 315; corrosive, 315.
Œsophagus, diseases of, 314; abscess behind, 316; congenital narrowing of, 314; congenital obstruction in, 314; diphtheria of, 1026; malformations of, 314; pseudo-diphtheria in, 315; stricture of, 314; thrush in, 315; in scarlatina, 956.
Oil enemata, 67.
Oiled-silk jacket, 61.
Omphalitis in newly born, 82.
Omphalo-mesenteric duct, 118, 353.
Onychia, syphilitic, 1115.
Ophthalmia, gonorrhœal, 87; in newly born, 87; treatment, 88.
Opisthotoaus, cervical, 730; hysterical, 736; in cerebro-spinal meningitis, 758; in birth paralysis, 798; in meningeal hæmorrhage, 109, 110; in chronic basilar meningitis, 776; in marasmus, 242; in tuberculous meningitis, 773.
Opium, elimination of, in milk, 139; in gastro-enteric intoxication, 379; in bronchitis, 518; preparations and dosage, 53.
Optic nerve, atrophy of, in cerebral tumour, 785.
Orange juice in scurvy, 260.
Orchitis, in mumps, 1018; in specific urethritis, 686; syphilitic, 1111; tuberculous, 1086.
Orthopnœa, in chronic valvular disease, 629; in functional disorders of the heart, 638.
Osteo-myelitis, acute (see ARTHRITIS, ACUTE), 899; acute, syphilitic, 916; in newly born, 84; tuberculous, 913; symptoms, 914; diagnosis, 915; treatment, 915.
Osteo-periostitis, chronic, syphilitic, 917.
Osteotomy in rickets, 273.
Ostitis, primary, followed by joint disease, 902; simulated by scurvy, 249.
Otitis, acute, 943; etiology, 943; lesions, 943; symptoms, 944; complications and sequelæ, 947; treatment, 948; cerebral abscess in, 780, 948; thrombosis of lateral sinus in, 948; facial paralysis in, 948; labyrinth in, 948; mastoid disease in, 947; meningitis in, 948; chronic, in late syphilis, 1117; reflex cough from, 523; frequent attacks of, with adenoids, 302; in influenza, 1126; in scarlet fever, 966; in syphilis, 1111; in typhoid fever, 1067; adenitis complicating, 885.
Overlying, causing death by asphyxia, 44.
Oxyuris vermicularis (see WORMS, INTESTINAL), 450.
Ozæna in late syphilis, 1118 (see RHINITIS, ATROPHIC), 485; syphilitic, 485.

Pachymeningitis, acute, 751; chronic (internal), 751; syphilitic, 1111; meningeal hæmorrhage from, 800; hæmorrhagic, 751; pseudo-membranous, 751.
Pack, cold, 49; hot, 56; mustard, 54.
Palate, cleft, 274; deformities of, stigmata of degeneration, 818; diphtheritic paralysis of, 851; hard, ulceration of, 286; in late syphilis, 1118; soft, lesions of, in hereditary syphilis, 486.
Pancreas, ferments of, 321; syphilis of, 1112; tuberculosis of, 1086.
Paracasein, formed from casein in stomach digestion, 149.
Paralysis, ascending, 842; atrophic (see PARALYSIS, INFANTILE SPINAL), 831; birth, 107, 796; atrophy and sclerosis following, 797; meningo-encephalitis, 796; secondary degenerations following, 797; symptoms, 797; Erb's, 111; facial, 110, 853; in acute otitis, 948; hysterical, 736; in compression-myelitis, 830; in multiple neuritis, 848; in myelitis, 828; Landry's, 842; of face in newly born, 110; of the upper extremity in newly born, 111; peripheral, 107 (see also NEURITIS, MULTIPLE), 846; post-diphtheritic, 1028; pseudo-hypertrophic, 844; simulated by scurvy, 247.
Paralysis, infantile cerebral, 107, 795; acute acquired, 799; birth, 796; of intra-uterine origin, 795; varieties and

symptoms, 796, 797, 801; prognosis, 802; diagnosis, 803; treatment, 804.

Paralysis, infantile spinal, 831; etiology, 832; symptoms, 833; course, 834; diagnosis, 837; distribution of primary paralysis, 834; electrical reactions, 835, 838; residual paralysis and deformity, 835; prognosis, 838; treatment, 838; mechanical, 839.

Paraplegia, Pott's (see MYELITIS, COMPRESSION), 829; spastic, 795.

Paregoric, dosage of, 53.

Parotitis, epidemic (see MUMPS), 916.

Paste, mustard, 54.

Pasteurized milk, 154.

Pathology, general considerations of, 40.

Pavor nocturnus, 742.

Peliosis rheumatica, 876.

Pelvis, deformities of, in rickets, 263.

Pemphigus, gangrenosa, 936; syphilitic, 1112; in newly born, 94.

Pepsin in stomach secretion, 319.

Peptonized milk, preparation of, 158; partially, 158; completely, 158.

Percentages of ingredients in milk formulas, how to calculate them, 196.

Pericarditis, 617; acute, in broncho-pneumonia, 554; chronic, with adhesions, 621; diagnosis, 620; dry, 618; external, 618; in newly born, 84; in rheumatism, 1143; mediastinal, 618; prognosis, 620; purulent, 618; sero-fibrinous, 618; tuberculous, 618; with effusion, 618; with effusion of blood, 618; with lobar pneumonia, 565; with pleuro-pneumonia, 580; with transudation of serum, 617.

Pericardium, congenital absence of, 613; tuberculosis of, 1085.

Perinephritis, 679; acute peritonitis complicating, 466.

Peritonæum, diseases of, 465; hæmorrhage into, in newly born, 99; in tuberculosis, 1086.

Peritonitis, acute, 465; etiology, 465; lesions, 466; symptoms, 467; treatment, 468; chronic, non-tuberculous, 469; with ascites, 469; fœtal, cause of malformations, 353; in intussusception, 435; in newly born, 83; in suppurative appendicitis, 439; pelvic, from gonorrhœa, 690; tuberculous, 470; miliary, with general tuberculosis, 471; miliary, with ascites, 471; fibrous form, 472; ulcerative form, 473; with tuberculous mesenteric glands, 474; with intestinal ulcers, 412; with lobar pneumonia, 565.

Perityphlitis (see APPENDICITIS), 438.

Perlêche, 276.

Perspiration (see SWEATING), 922.

Pertussis, 1004; broncho-pneumonia in, 551, 1009; complications, 1008; convulsions, 1010; diagnosis, 1011; etiology, 1005; hæmorrhages in, 1008; incubation, 1006; infective period, 1006; lesions, 1006; paralysis in, 1010; predisposition to, 1005; prognosis, 1011; prophylaxis, 1012; symptoms, 1006; treatment, 1012.

Peyer's patches, in typhoid fever, 1063; swollen, in acute ileo-colitis, 388; tuberculosis of, 411; ulceration of, in ileo-colitis, 390.

Pharyngitis, acute, 293; uvulitis in, 294; chronic catarrhal, syphilitic, 1011.

Pharynx, diseases of, 293; adenoid vegetations of vault, 299, 481; with adenitis, 885; diphtheria of, 1025; diphtheritic paralysis of, 852; lesions of, in hereditary syphilis, 485; pseudo-diphtheria of, 1058; reflex cough from, 522; retro-pharyngeal abscess, 295; syphilitic ulceration of, 1111; syringing of, 59.

Phimosis, 683; reflex phenomena from 684.

Phlebitis, of dural sinuses, 779.

Phosphorus in rickets, 271.

Photophobia, in influenza, 1125; in measles, 980; in tuberculous meningitis, 772.

Phthisis, chronic, 1081, 1101.

Physical examination of the child, 33.

Pick's paste, 934.

Pigeon-breast in adenoids, 301.

Pinworms (see WORMS, INTESTINAL), 450; proctitis from, 454.

Plasmodium malariæ, 1131.

Pleura, effusion into, in acute nephritis, 664; tuberculosis of, 1077, 1084.

Pleurisy, 591; dry, 592; in acute broncho-pneumonia, 540; purulent (see EMPYEMA), 596; tuberculous, dry form, 592; with lobar pneumonia, 574; with serous effusion, 593.

Pleuro-pneumonia, 579; pericarditis in, 617, 619.

Pneumococcus, in broncho-pneumonia, 530, 532; lobar pneumonia, 563; peritonitis, 566; diphtheria, 1027, 1029; empyema, 596; acute meningitis, 767; malignant endocarditis, 623.

Pneumonia, 527; anatomical varieties and classifications of, 527; broncho- (see BRONCHO-PNEUMONIA, ACUTE), 531; catarrhal (see BRONCHO-PNEUMONIA, ACUTE), 531; chronic interstitial (see BRONCHO-PNEUMONIA, CHRONIC), 582; in newly born, 83; in typhoid fever, 1066; sources of infection, 530; varieties, classification, 530; hypostatic, 582; in marasmus, 239; lobular (see BRONCHO-PNEUMONIA, ACUTE), 531; pleuro- (see PLEURO-PNEUMONIA), 579; syphilitic, 1110; tuberculous, 1079 (see also TUBERCULOSIS, PNEUMONIA);

course, duration, termination, 1098; diagnosis, 1098; physical signs, 1097; chronic, 1096.
Pneumonia, lobar, 562; etiology, 562; frequency of, 569; complicating influenza, 1126; complications, 574; course, 565; abortive, 566; cerebral, 566; diagnosis, 575; lesions, 563; lysis, frequency of, 569; pathological differentiation from broncho-pneumonia, 528; physical signs, 571; prognosis, 573; symptoms, 565; cerebral, 570; termination, 574; treatment, 578.
Pneumothorax in pulmonary tuberculosis, 1078.
Pock, in vaccinia, 1002; in varicella, 996.
Poisons, gastritis from, 338, 339.
Poisoning, stomach-washing in, 64.
Poliencephalitis, acute, causing cerebral paralysis, 800.
Poliomyelitis, acute (see PARALYSIS, INFANTILE SPINAL), 831.
Polydactyly, stigma of degeneration, 818.
Polydipsia in diabetes insipidus, 653; mellitus, 1147.
Polypi, nasal, 482; rectal, 452.
Polyuria, 652; hysterical, 736; in diabetes insipidus, 653; mellitus, 1147.
Porencephalus, 751.
Pott's disease (see SPINE, CARIES OF), 902; cervical, causing torticollis, 732; reflex cough in, 523.
Poultices, use and preparation of, 55.
Powders for skin, 4.
Præcordia, bulging of, 608, 632.
Pregnancy, effect on woman's milk, 137, 139; effect on nursing child, 179.
Premature infants, management of, 12; results with, 14.
Prematurity, cause of marasmus, 238.
Prepuce, adherent, 683.
Prickly heat, 925.
Proctitis, 454.
Prognosis, general consideration of, 42.
Progressive muscular atrophy, hand type, 843; peroneal type, 844.
Prolapsus ani (see also RECTUM, PROLAPSE OF), 452; from proctitis, 455; in ileo-colitis, 395; in membranous ileo-colitis, 399.
Prophylaxis, general consideration of, 46.
Proteids, determination of, in milk, 135; function in diet, 125; in the fæces, 323; of woman's milk, 133; of cow's milk, 149; percentages of, in modification of cow's milk, 194, 195, 196; in feeding difficult cases, 209 *et seq.*; vegetable, 126.
Pseudo-diphtheria, 1020, 1056; bacillus, 1042; broncho-pneumonia in, 1059; communicability of, 1057; diagnosis, 1060; etiology, 1057; in measles, 1059; in scarlet fever, 1059; lesions, 1057; mortality, 1060; prognosis, 1060; quarantine in, 1061; streptococcus in, 1057; symptoms, 1058; treatment, 1061.
Pseudo-hypertrophic paralysis, 844.
Pseudo-paralysis in rickets, 265; in scurvy, 247; in syphilis, 917, 1115.
Psoas abscess in spinal caries, 906.
Psoriasis of tongue, 276.
Puberty, delayed, stigma of degeneration, 819; in cretins, 815; in syphilis, 1119; effect of, on heart in valvular disease, 634, 639; reflex cough of, 523.
Pulse, examination of, 35; in early life, 607.
Purpura, 871; arthritic, 876; blood in, 873; fulminans, 875; gangrenous, 876; hæmatemesis in, 875; hæmorrhagica, 874; Henoch's, 875; primary, 872; rheumatica, 876; simplex, 871, 874; symptomatic, 871; cachectic, 872; infectious, 872; neurotic, 872; mechanical, 872; toxic, 872.
Pyæmia, in newly born, 81; of bone (see ARTHRITIS, ACUTE), 899.
Pyelitis, 674.
Pyelo-cystitis, 674.
Pyelo-nephritis, 656, 674.
Pylephlebitis, 460; cause of hepatic abscess, 460.
Pylorus, atresia or stenosis of, 325; stenosis, dilated stomach in, 348.
Pyogenic diseases, acute, in newly born, 81; general symptoms, 86; prophylaxis, 86; treatment, 87.
Pyo-nephrosis following pyelitis, 675.
Pyo-pneumothorax in pulmonary tuberculosis, 1078.
Pyo-salpinx from gonococcus vaginitis, 690.
Pyuria, 650; in pyelitis, 676.

Quartan intermittent fever, 1134.
Quincke's lumbar puncture, 757.
Quinine, dosage, 1139; methods of administration, 1138; scarlatiniform rash, 971.
Quinsy, 310.
Quotidian intermittent fever, 1134.

Race, influence of, upon rickets, 252.
Rachitis (see RICKETS), 251.
Reaction of degeneration, in Erb's paralysis, 113; in facial paralysis, 111, 854; in infantile spinal paralysis, 835, 838; in multiple neuritis, 849.
Rectal injections, astringent, 403; in acute ileo-colitis, 403; opium in, 403; saline, 403.
Rectum, diseases of, 452; administration of drugs by, 67; atresia of, 352; congenital obstruction of, 117; enemata,

67; feeding by, 67; hæmorrhage from ulcers of, 456; inflammation of (see PROCTITIS), 454; malformations of, 352; prolapse of, 452; ulcers of, 455.
Red gum (see MILIARIA RUBRA), 924.
Regurgitation of food, causes of, in young infants, 291; nasal, in diphtheria, 852, 1032, 1040.
Remittent fever, malarial, 1132.
Renal calculi, 677; renal colic, 678.
Rennet, ferment in digestion, 320.
Respiration, artificial, methods of, 72; Cheyne-Stokes, in cerebro-spinal meningitis, 759; in meningitis, tuberculous, 773; noisy at night with adenoids, 301; paralysis of, in diphtheria, 862; rapidity and characteristics, 510; in pulmonary tuberculosis, 1095.
Respiratory system, diseases of, 478.
Restlessness at night in rickets, 259.
Rheumatism, 1141; symptoms, 1142; diagnosis, 1145; treatment, 1146; chorea in, 722, 1144; endocarditis in, 622, 1143; erythema in, 1145; purpura in, 876, 1145; scarlatinal, 968; simulated by scurvy, 249; subcutaneous tendinous nodules, 1144; tonsillitis in, 310, 1144; torticollis in, 732, 1143.
Rhinitis, chronic, 482; simple, 482; hypertrophic, 484; atrophic, 485; syphilitic, 485; membranous, 487; hypertrophic, cause of asthma, 524.
Rhino-pharyngitis, acute, 478; in influenza, 1125; with adenoids, 301.
Rhino-pharynx, diphtheria of, 1025; reflex cough from, 522; simple catarrh of, in acute otitis, 943.
Ribemont's laryngeal tube, 73.
Ribs, beading of, early symptoms in rickets, 252; resection of, in empyema, 604.
Rice water, 165.
Rickets, 251; etiology, 251; pathology, 253; lesions, 254; symptoms, 258; course and termination, 267; acute, 268 (see also SCORBUTUS), 244; congenital, 268; convulsions in, 701; diagnosis, 268; from scurvy, 249, 269; prognosis, 269; treatment, 270; of deformities, 271; dilatation of stomach in, 348; late, 268; spleen in, 897.
Ridge's food, 166.
Ringworm of scalp, 941.
Robinson's patent barley, 165.
Rotary spasm of head, 729.
Rötheln (see RUBELLA), 993.
Round worms (see WORMS, INTESTINAL), 448.
Rubella, 993; complications and sequelæ, 995; diagnosis, 995; eruption, 993; incubation, 993; symptoms, 993; treatment, 995.
Rubeola (see MEASLES), 977.
Saccharomyces albicans in thrush, 287.
Saint Vitus's dance (see CHOREA), 721.
Saline solution, as rectal injection, 403; subcutaneous injection of, in cholera infantum, 384; in acute inanition, 230.
Saliva, 319.
Salivation, in mumps, 1017; in ulcerative stomatitis, 285.
Salts, inorganic, in modification of cow's milk, 187; mineral, function of, in diet, 128; of cow's milk, 150; of woman's milk, 134.
Sarcoma, of brain, 783; of kidney, 671; of spinal cord, 839; of stomach, 350.
Scabies, 939.
Scalp, pustular eczema of, 929; ringworm of, 941; seborrhœa of, 926.
Scapula, angel-wing deformity of, 837.
Scarlatina (see SCARLET FEVER), 953; anginosa, 1058.
Scarlatiniform erythema, causes of, 971.
Scarlet fever, 953; albuminuria in, 967; angina in, 964; blood in, 969; complications and sequelæ, 964; desquamation, 958; diagnosis, 970; diphtheria in, 965; disinfection after, 973; duration of infective period, 955; eruption, 957; etiology, 953; heart in, 969; incubation of, 954; invasion, 956; joints in, 968; kidneys in, 967; lesions, 956; lymph nodes in, 965; mode of infection, 954; mortality in, 972; myocarditis in, 636; nervous system in, 970; other infectious diseases with, 970; otitis in, 966; predisposition to, 953; prognosis, 972; prophylaxis, 973; pseudo-diphtheria in, 964, 1059; quarantine in, 973; relapses, recurrences, and second attacks, 963; symptoms, 956; surgical, 962; throat in, 964; treatment, 975.
Schultze's method of inducing artificial respiration, 72.
Sclerema, 118; in cholera infantum, 383.
Scorbutus, 244; symptoms, 246; treatment, 250; stomatitis in, 284.
Scrofula (see ADENITIS, TUBERCULOUS), 888; (see TUBERCULOSIS).
Scurvy (see SCORBUTUS), 244.
Seborrhœa, 926.
Seborrhœic eczema, 929.
Senses, special, development of, 25.
Sepsis in newly born, 81.
Septum nasi, ulcer of, with hæmorrhage, 489.
Serous membranes, frequency of disease of, 40.
Serum diagnosis of typhoid fever, 1077.
Serum-therapy of diphtheria, 1049.
Sewer-gas, influence on sore throat, 1057.
Shiga bacillus (see BACILLUS OF DYSENTERY), 365, 385.
Shock in intussusception, 434.
Shower bath, 57.

Sight, when developed, 25.
Sigmoid flexure, length, 321.
Singultus, 730.
Sinuses of dura mater, thrombosis of, 779; lateral, in otitis, 948.
Skin, diseases of, 922; anomalies of, as stigmata of degeneration, 818; of newly born, 922; care of, in newly born, 4.
Skull, asymmetry of, in birth paralysis, 799; sutures, separation of, in hydrocephalus, 791; syphilitic nodes on, 920.
Sleep, disorders of, 740; disturbed, 7, 740; with hypertrophy of tonsils, 312; in intestinal indigestion, 416; in rickets, 259; with adenoids, 301; excessive, 443; inspection during, 34; proper periods of, 5.
Sleeplessness, 740.
Smallpox, protection against (see VACCINATION), 998.
Smegma, 683, 686.
Smell, sense of, when developed, 27.
Snoring, with adenoids, 301; with hypertrophied tonsils, 318.
Snuffles, syphilitic, 485, 1113.
Spasm, carpo-pedal (see TETANY), 716; of glottis, 719; habit, 727; nodding, of the head, 729; rotary, of the head, 729; vesical, 697.
Speech, disorders of, 738; when acquired, 27.
Spina bifida, 820; with congenital hydrocephalus, 791.
Spina ventosa (see OSTEO-MYELITIS, TUBERCULOUS), 913.
Spinal cord (see CORD, SPINAL), 820.
Spine, angular curvature of, in caries, 905; caries of, 902; symptoms, 903; physical examination, 887; diagnosis, 907; treatment, 907; causing compression of cord, 829; curvature of, in hip disease, 910; hysterical affections of, 735; in rickets, 261; lateral deviation of, 907; Pott's disease of (see SPINE, CARIES OF), 902.
Spirochœta pallida, in syphilis, 1106.
Spleen, diseases of, 896; amyloid degeneration of, 898; enlargement of, 897; in acute disease, 897; in chronic cardiac disease, 630; in chronic disease, 897; in cirrhosis of liver, 462; in leukæmia, 869; in malaria, 1134; in pseudo-leukæmic anæmia, 865; in rickets, 258; in simple anæmia, 861; in typhoid fever, 1064; with amyloid liver, 463; in diphtheria, 1027; in hereditary syphilis, 1110; in late syphilis, 1118; in tuberculosis, 1095; new growths and tumours of, 898; position and methods of examination, 896; weight, 896.
Sponge bath, cold, 57.
Sponging, cold, 49.
Spotted fever (see MENINGITIS, CEREBROSPINAL), 760.
Spray, nasal, 57; steam, 61.
Sprue (see THRUSH), 286.
Sputum, means of obtaining, for examination, 1100.
Stammering, 739.
Staphylococcus, in pseudo-diphtheria, 1057; in furunculosis, 935; in acute broncho-pneumonia, 532; in diphtheria, 1023; in empyema, 597.
Starch, in the fæces, test for, 324; objections to, as food of young infants, 128.
Status lymphaticus, 45, 879.
Stenosis, laryngeal, in acute catarrhal laryngitis, 492; in membranous laryngitis, 496; in syphilitic, 507; of pylorus, 325; dilated stomach in, 348.
Stercoraceous vomiting, in appendicitis, 442; in intussusception, 432.
Sterilization of milk, 153; changes produced by, 153; at 212° F., 153; at low temperature, 154; indications for, 157; limitations of, 157; methods of, 155.
Sterno-mastoid hæmatoma of, 96; spasm of (see TORTICOLLIS).
Stigmata of degeneration, 818.
Stimulants, alcoholic, 51; indications, 51; contra-indications, 51; administration, 51.
Stomach, diseases of, 318; absorption from, 321; bacteria of, 322; capacity of, 319; congestion of, in acute gastro-enteric intoxication, 366; development of, 319; digestion in, 347; dilatation of, 347; in chronic gastric indigestion, 343; in rickets, 265; hæmorrhage from, 350; in newly born, 105; in scurvy, 248; inflammation of (see GASTRITIS), 337; malformations and malpositions of, 324; round ulcer of, in chlorosis, 862; thrush in, 288; tuberculosis of, 1086; tumours of, 350; ulcer of, 349; in newly born, 349; from acute gastritis, 349; tuberculous, 349; round, perforating, 349.
Stomach washing, in acute gastritis, 341; in acute indigestion, 338; in chronic indigestion, 346; in gastro-intestinal intoxication, 377; method, 62; indications for, 63.
Stomatitis, aphthous (see HERPETIC STOMATITIS), 282; catarrhal, 281; in measles, 987; diphtheritic, 289, 1026; follicular (see HERPETIC STOMATITIS), 282; gangrenous, 290; gonococcus, 289; herpetic, 282; parasitic (see THRUSH), 286; syphilitic, 289; ulcerative, 284; vesicular (see HERPETIC STOMATITIS), 282.
Stone, in the kidney, 677; in the bladder, 698.

Stools, blood in, from ulcer of stomach, 349; in catarrhal ileo-colitis, 394, 396; in membranous ileo-colitis, 397; in intussusception, 432; in purpura, 875; fat in, test for, 203, 362; green, explanation of, 362; in acute intestinal indigestion, 362; in cholera infantum, 382; in gastro-duodenitis, 342; in intestinal indigestion, chronic, 414, 416; in simple gastro-enteric intoxication, 369; indication of improper feeding, 203; mucus in, in malnutrition, 233; undigested casein in, in chronic gastric indigestion, 345.

Strabismus, in tuberculous meningitis, 773; stigma of degeneration, 818; with tumour of crura cerebri, 786.

Streptococcus, antitoxin, 1062; pyogenes, in acute broncho-pneumonia, 532; in complications of scarlet fever, 964; in dermatitis gangrenosa, 937; in diphtheria, 1023, 1027, 1036; in empyema, 596; in peritonitis, acute, 466; in pseudo-diphtheria, 1057; in scarlet fever, 953.

Stridor, in catarrhal spasm of larynx, 490; in acute catarrhal laryngitis, 493.

Strophulus (see MILIARIA RUBRA), 924; (see URTICARIA), 938.

Struma (see TUBERCULOSIS).

Strychnine in acute broncho-pneumonia, 558.

Stupe, turpentine, 54.

Stuttering, 739.

Subcutaneous tendinous nodules in rheumatism, 1144.

Sucking, 318; as a bad habit, 743.

Sudamina, 924.

Sudden death, chief causes of, 44.

Sugar, cane, derivatives in digestion, 321; substitute for milk-sugar, 127, 186; milk, determination of, 135; percentage of, in woman's milk, 134; milk, derivatives in digestion, 321; percentages of, in modification of cow's milk, 186; solutions, rules for making, 205; stools in difficult digestion of, 415; symptoms of excess of, in food, 201, 203.

Summer diarrhœa, 364.

Suppositories, in chronic constipation, 426; medicated, 426; proctitis from long use of, 454.

Suprarenal capsules, in syphilis, 1111; in tuberculosis, 1086; hæmorrhage into, 100.

Sutures, closure of, 22; premature ossification of, 23; separation of, in hydrocephalus, 792.

Sweating, in infants, 922; of head in rickets, 259; in tuberculosis, 1094.

Symptomatology, general considerations, 31.

Syndactyly, stigma of degeneration, 818.

Synovitis, acute purulent (see ARTHRITIS, ACUTE), 899; scarlatinal, 968.

Syphilis, 1106; acute epiphysitis in, 915; acute osteo-myelitis in, 916; bone lesions in, 915; chronic osteo-periostitis in, 917; dactylitis in, 921; of larynx, 507; pseudo-paralysis in, 917; spleen in, 897; acquired, 1106.

Syphilis, hereditary, 1107; adenitis in, 887; bones, 1109; Colles's law, 1108; communicability of, 1109; diagnosis, 1119; etiology, 1107; evidences of, in fœtus, 1112; hæmorrhages, 1115; lesions, 1109; prognosis, 1119; prophylaxis, 1120; pseudo-paralysis, 1115; rhinitis of, 485; spleen, 1010; symptoms, 1112; at birth, 1112; treatment, 1121; late hereditary, 1116; bones, 1117; skin, 1118; spleen, 1118; teeth, 1116; tertiary, chronic laryngitis in, 507; intubation for, 508.

Syringe, nasal, 58; for antitoxin, 1050.

Syringing, nasal, 58; of mouth and pharynx, 59.

Syringo-myelia, 840.

Syringo-myelocele, 822.

Tâche cérébrale in tuberculous meningitis, 773.

Tachycardia, 638.

Tænia, cucumerina or elliptica, 446; flava punctata, 447; nana, 447; saginata or medio-canellata, 446; solium, 446.

Tannic acid as rectal injection, 403.

Tapeworms, 445.

Tar ointment in eczema, 934.

Taste, when developed, 27.

Teeth, 27; eruption of first set, 28; permanent set, 29; presence of, at birth, 28; care of, 3; decayed, cause of adenitis, 884; delayed, in rickets, 266; grinding of, in intestinal indigestion, 416; Hutchinson's, in syphilis, 1116.

Teething, reflex symptoms from, 279.

Temperature, at birth, 36; best taken in rectum, 36; in childhood, 36; subnormal, 36; raised by artificial heat, 36; variations of, in health, 36; general consideration of, 48; of nursery, 9.

Tenesmus, from proctitis, 455; in intussusception, 434; in membranous ileo-colitis, 399; treatment of, 403.

Tent for inhalation and vapourization, 60.

Tertian intermittent fever, 1134.

Testicle, retraction of, with renal calculus, 678; syphilis of, 1111; tuberculosis of, 1086; undescended, 685.

Tetanus, in the newly born, 89.

Tetany, 716.

Therapeutics, general consideration of, 47.

Thirst, in diabetes insipidus, 653; mellitus, 1147; in hot weather, 373.

Thomsen's disease, 730.

Thoracoplasty, 605.

Thorax, description of, 509; measurements of, 20, 24; causes of deformity of, 24.

Threadworms (see Worms, Intestinal), 450.

Throat, diseases of (see Pharynx and Tonsils); importance of inspection of, 38.

Thrombosis, 640; cachectic, of dural sinuses, 778; in diphtheria, 1028, 1037; in infectious diseases, 640; inflammatory, of dural sinuses, 779; of internal jugular vein, 640; of lateral sinus in acute otitis, 948; of sinuses of dura mater, 778; of the aorta, 640; of the vena cava, 640; septic, of dural sinuses, 779.

Thrush, 286.

Thymus, abscess of, syphilitic, 1111; dulness due to, 511; enlargement of, causing convulsions, 45; in status lymphaticus, 879; tuberculosis of, 1086.

Thyroid extract in cretinism, 815.

Thyroid gland, congenital, absence of, in cretinism, 813.

Tibia, deformities of, in rickets, 264; enlarged epiphyses in rickets, 254; sabre-blade deformity in syphilis, 918.

Tinea tonsurans, 941; treatment, 941.

Toes, clubbing of, in congenital heart disease, 614.

Tongue, diseases of, 274; bifid, 275; congenital hypertrophy of, 275; epithelial desquamation of, 276; geographical, 277; inflammation of, 277; malformations of, 275; ulcer of frenum, 278.

Tongue-sucking, 747.

Tongue-swallowing, 278.

Tongue-tie, 275.

Tonics, 52.

Tonsils, diseases of, 307; anatomy of, 307; chronic hypertrophy of, 312; diphtheria of, 1024, 1031; hypertrophy of, cause of asthma, 524; hypertrophy of, in rickets, 266; removal advised in tuberculous adenitis, 894; with adenitis, 887; pseudo-diphtheria of, 1058; membrane upon, in scarlet fever, 960.

Tonsillitis, acute catarrhal, 307; croupous (see Pseudo-diphtheria), 307, 1058; ulcero-membranous, 308; follicular, 309; in rheumatism, 1144; phlegmonous, 310; acute otitis in, 943.

Tonsillotomy, 313.

Top-milk, 151.

Torticollis, 731; congenital, 732; from cervical Pott's disease, 732, 904; from hæmatoma of sterno-mastoid, 96; hysterical, 735; in phlegmonous tonsillitis, 311; in retro-pharyngeal abscess, 297; malarial, 732; rheumatic, 732; spasmodic, 731.

Touch, when developed, 26.

Toxæmia, in intestinal indigestion, chronic, 415; vomiting in, 329; in acute gastric indigestion, 336.

Toxins, of diphtheria, 1023, 1052.

Trachea, diphtheria of, 1025.

Tracheotomy, for foreign body in larynx, 509; in membranous laryngitis, 498; in retro-œsophageal abscess, 318.

Trismus, in tetanus, 90.

Trypsin, 321.

Tubercle bacilli (see Bacillus of Tuberculosis), 1074.

Tuberculin test in herds, 144; in diagnosis, 1100.

Tuberculosis, 1070; age, 1071; anæmia, 1096; bacillus of (see Bacillus of Tuberculosis), 1070; in milk, 141; of brain, 1085; bronchial lymph nodes in, 1074; clinical forms of, 1087; broncho-pneumonia, 1077, 1090; chronic phthisis, 1101; chronic pulmonary, 1081; congenital, 1072; cases resembling marasmus, 1087; cases resembling a continued fever, 1088; course, 1098; chronic, 1081, 1096; diagnosis from marasmus, 242, 1088; from typhoid, 1090; from broncho-pneumonia, 1098; etiology, 1070; following measles, 988; following pertussis, 1012; frequency, 1070; hæmoptysis, 1095; incipient, symptoms in, 1089; intestines, 410, 1086; intra-uterine infection, 1072; kidney, 670, 1086; lesions, 1076; diagnosis, 1103; physical signs, 1103; mesenteric, 410, 1075; mode of infection, 1072; of larynx, 506; of lymph nodes, cervical, 888; paths of infection, 1074; pericarditis in, 619; physical signs, 1097; pleura in, 592, 1084; predisposing causes, 1071; prognosis, 1104; prophylaxis, 1104; spleen, 898, 1085, 1095; sputum, means of obtaining, 1100; treatment, 1105; tuberculin in diagnosis, 1100.

Tuberculous, adenitis, 888; meningitis, 770; nephritis, 670; ostitis, 900; pericarditis, 618; peritonitis, 470; pleurisy, 592; pneumonia, 1090.

Tumour, abdominal, in intussusception, 432; cerebral, 783; tuberculous, 1085; fatty, in cretinism, 815; of spinal cord, 839; mediastinal, tuberculous lymph nodes, 1101; of spleen, 897, 1118.

Tunica vaginalis, hydrocele of, 687.

Turpentine stupe, preparation of, 54.

Tympanites in acute peritonitis, 467; in intestinal indigestion, 416; in rickets, 265; in typhoid fever, 1064.

Typhlitis (see Appendicitis), 438.

Typhoid fever, 1062; bacillus of, in milk, 142; complications and sequelæ, 1060; diagnosis, 1067; etiology, 1062; intestinal hæmorrhage in, 1066; intestinal perforation in, 1063, 1066; lesions, 1063; prognosis, 1068; scarlatiniform erythema in, 971; symptoms, 1064; treatment, 1069; urine in, 1066; Widal's test in, 1067.

Ulcers, catarrhal, of intestine, 389; follicular, of intestine, 389; following tuberculous adenitis, 892; of stomach, 349; tuberculous, of skin, 892, 1118; syphilitic, 1118; tuberculous, of intestine, 411, 1086; typhoid, 1063.
Umbilical vessels, arteritis in newly born, 82; phlebitis in newly born, 83; fistula, 114.
Umbilicus, hæmorrhage from, in newly born, 104; hernia of, 115; inflammation of vessels in newly born, 82; treatment of suppuration, 87; tumours of, 113.
Urachus, persistent, enuresis from, 692.
Uræmia, acute, in scarlet fever, 968; in acute ephritis, 664; in chronic nephritis, 669.
Ureter, dilatation of, 655; supernumerary, 655.
Urethra, hæmorrhage from, in newly born, 105.
Urethritis, 686; gonorrhœal, 686.
Uric acid, in anæmia, 861; in chorea, 725; in cyclic vomiting, 333; in malnutrition, 234; in early infancy, 643; infarctions, in kidney, 658; causing hæmaturia, 106.
Urine, acetone in (see ACETONURIA), 651; arrest of secretion (see ANURIA), 652; albumin in, 644; blood in (see HÆMATURIA), 646; "brick dust" in, 649; composition of, 644; daily quantity of, 642; diacetic acid in, 651; examination of, 40; hyperacidity of, in rheumatism, 1147; incontinence of, 692; with adenoids, 300; in diabetes, 1147; in myelitis, 828; in typhoid, 1066; in vesical calculus, 698; indican in, (see INDICANURIA), 650; in infancy and childhood, 642; methods of collecting, 40, 642; microscopical examination of, 643; physical character of, 643; pus in (see PYURIA), 648; reaction of, 643; specific gravity of, 643; sugar in, 644 (see also GLYCOSURIA), 647; urea in, 644; uric acid in, 644 (see also LITHURIA), 649.
Uro-genital organs, tuberculosis of, 1086.
Uro-genital system, diseases of, 642.
Urticaria, 938; following diphtheria antitoxin, 1052; in influenza, 1128; in intestinal indigestion, 417; papulosa, 938; scarlatiniform rash with, 972.
Uvula, bifid, 275; diphtheria of, 1025; elongation of, 295; cause of asthma, 524; causing cough, 522; œdema of, 294; inflammation of, 294.

Vaccination, 998; choice of virus, 998; methods of, 1000; revaccination, 998.
Vaccinia, 998.
Vaginitis, 688; simple, 688; gonococcus vaginitis, 689.
Vapourizer, 60.
Vapour bath, 56.
Varicella, 996; symptoms, 996; diagnosis, 997; gangrenosa, 936, 997; treatment, 998.
Vegetables, allowed from third to sixth years, 222; forbidden from third to sixth years, 223.
Vegetations on valves in endocarditis, 626.
Vein, internal jugular, thrombosis of, 641; umbilical, 606.
Veins, abdominal, dilated in cirrhosis of liver, 462; in thrombosis of vena cava, 641.
Vena cava, thrombosis of, 641.
Ventricles, cardiac, relative thickness of, 608.
Vertigo, in cerebral abscess, 781; in cerebellar tumour, 787; in functional disorders of heart, 638.
Vesical, calculi, 698; spasm, 697.
Viscera, abdominal, transposition of, 353; frequency of inflammations of, 41; hæmorrhages of, in newly born, 99.
Voice, hoarse or husky, with adenoids, 302; nasal, with hypertrophy of tonsils, 312; with adenoids, 301; in diphtheritic paralysis, 852.
Volvulus, fœtal, cause of malformations, 353.
Vomiting, 328; from overfilling the stomach, 328; in acute gastric indigestion, 329; in acute intestinal obstruction, 329; in peritonitis, 329; in nervous diseases, 329; at onset of acute infectious disease, 329; from toxic substances in the blood, 329; reflex, 330; from habit, 330; chronic, 330; of blood, in ulcer of stomach, 349; stercoraceous, in appendicitis, 442; in intussusception, 432; cyclic, 331; symptoms, 331; treatment, 334.
Vulvitis, gangrenous, 688.

Walking, causes which prevent, 25; delayed, in rickets, 264; late, in malnutrition, 232; when attempted, 25.
Wasting, in tuberculosis, 1094; simple (see MARASMUS), 238.
Water, function of, in diet, 128.
Weaning, 179; time for, 180; indications for, 180; sudden, 181; percentages of milk required at, 197.
Weather, hot, prophylaxis against diarrhœa in, 373.
Weight, 15; at birth, 16; curve during first few weeks, 16; curve of first year, 17; from second to fifth year, 19; of

older children, 19; from birth to sixteenth year, 20; loss of, in acute inanition, 228; stationary, indications in, 199; symptoms of unsuccessful nursing, 173.
Werlhof's disease (see PURPURA), 871.
Wet-nurse, in acute gastro-enteric intoxication, 376; in acute inanition, 229; selection of, 178; dangers of syphilis, 1121.
Wet-nursing, 178; *versus* artificial feeding, 170; indications for, 170; disadvantages of, 170.
Wheal, in urticaria, 938.
Whey, 162; whey mixtures, 210.
White-swelling of knee, 911.
Whooping cough (see PERTUSSIS), 1004.
Widal's test in typhoid fever, 1067.
Winckel's disease, 92.
Worms, intestinal, 445; tapeworm, 445; roundworm, 448; threadworms, 450.
Wrist, enlarged epiphyses in rickets, 263.
Wry-neck (see TORTICOLLIS), 731.

Zoolak, 161.

THE END.

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